

# ACNP 52nd Annual Meeting

## **Final Program**

### December 8-12, 2013 The Westin Diplomat Resort & Spa Hollywood, Florida

President: David A. Lewis, M.D. Program Committee Chair: Randy D. Blakely, Ph.D. Program Committee Co-Chair: Pat R. Levitt, 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 distinct pleasure to welcome you to the 2014 meeting of the American College of Neuropsychopharmacology! This 52nd annual meeting will again provide opportunities for the exercise of the College's core values: the spirit of Collegiality, promoting in each other the best in science, training and service; participation in Community, pursuing together the goals of understanding the neurobiology of brain diseases and eliminating their burden on individuals and our society; and engaging in Celebration, taking the time to recognize and enjoy the contributions and accomplishments of our members and guests.



Under the excellent leadership of Randy Blakely and Pat Levitt, the Program Committee has done a superb job in assembling an outstanding slate of scientific presentations. Based on membership feedback, the meeting schedule has been designed with the goals of achieving an optimal mix of topics and types of sessions, increasing the diversity of participating scientists and creating more time for informal interactions. The presentations will highlight both the breadth of the investigative interests of ACNP membership and the unprecedented depth of studies examining disease mechanisms and new strategies for treating these illnesses. Accordingly, the theme of this year's meeting, reflected in the title of the Plenary Session, is "Neural Circuitry Structure and Plasticity: Substrate for Brain Disorders and Novel Therapeutics."

I believe you will experience at this meeting ample evidence of the continued efforts of ACNP Council and staff to ensure the College's successful leadership in shaping the future of our field, in facilitating the engagement of a broader group of investigators and in preserving the distinctive features that define us as a unique community of scientists and clinicians. I would like to extend a special note of thanks to Ronnie Wilkins, Sarah Timm, Laura Bersacola-Hill and the rest of the ACNP staff for their tremendous help and good humor over the past year.

It has been a pleasure and honor to serve as president of the ACNP. I hope you find the meeting enlightening, energizing and enjoyable!

David A. Lewis, M.D. President, 2013

ACNP AMERICAN COLLEGE OF NEUROPSYCHOPHARMACOLOGY

## 52nd ANNUAL MEETING

## GENERAL PROGRAM

#### HOLLYWOOD, FLORIDA WESTIN DIPLOMAT RESORT & SPA

**DECEMBER 8-12, 2013** 

Disclosures for 2013 speakers (mini-panel, panel, study group, and plenary) and poster presenters may be found online at: <u>www.acnp.org</u> (click the Annual Meeting tab). Vanderbilt CME has determined that there is no conflict of interest.



#### **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 52nd Annual Meeting • Final Program Program at a Glance

#### Saturday, December 7, 2013

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

8:00 AM – 8:00 PM Diplomat Ballroom 1 & 2 Schizophrenia Prodome Meeting

 2:00 PM - 4:00 PM
 Room 208

 Neuropsychopharmacology &
 Neuropsychopharmacology Reviews

 EIC & Deputy Editors Meeting
 EIC

4:00 PM – 5:00 PM Room 218 ACNP Website Editors Meeting

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

5:00 PM – 6:30 PM Room 208 ACNP Publications Committee Meeting

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

#### Sunday, December 8, 2013

8:30 AM – 11:30 AM Regency Ballroom 1 & 2 Neuropsychopharmacology Reviews Plenary: "Frontiers in Discovery and Neurotherapeutics"

11:30 AM – 1:00 PM Room 212 & 213 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 Diplomat Ballroom 1 FNIH Biomarkers Consortium

11:30 AM – 1:00 PM Diplomat Ballroom 2 *Neuropsychopharmacology* Editorial Board

1:00 PM – 2:30 PM Regency Ballroom 1 & 2 NIH Institutes Directors' Briefing

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 9, 2013

6:45 AM – 8:00 AM Room 220 CDI Booster Session

8:00 AM – 11:30 AM Grand Ballroom President's Plenary: "Neural Circuitry Structure and Plasticity: Substrate for Brain Disorders and Novel Therapeutics"

| 10:30 AM – 4:30 PM<br>Poster Viewing   | Great Hall 1-4   |
|--|------------------|
| 11:30 AM – 1:00 PM<br>Women's Luncheon | Great Hall 5 & 6 |

#### Monday, December 9, 2013

12:00 PM – 1:00 PM Room 206 CNS Spectrums Field Editor/Deputy Editor Meeting

1:30 PM – 3:00 PM Grand Ballroom Distinguished Lecture: "The Story of Rett Syndrome and the Insight it Provides into Neuropsychiatric Disorders"

#### Mini-Panel Sessions

3:00 PM – 4:15 PM Diplomat Ballroom 1 & 2 Neuronal Immaturity in Schizophrenia

4:15 PM – 5:30 PM Diplomat Ballroom 1 & 2 Social Processes Initiative in Neurobiology of the Schizophrenia(s)

#### Panel Sessions

3:00 PM – 5:30 PM Atlantic Ballroom 1 Kicking Over the Traces – Noncatecholic Biogenic Amines and Their Receptors

3:00 PM – 5:30 PM Regency Ballroom 2 Can Biology Inform Treatment Prediction and Selection in Depression?

3:00 PM – 5:30 PM Atlantic Ballroom 2 Autism Spectrum Disorders: From Rare Chromosomal Abnormalities to Common Molecular Targets

3:00 PM – 5:30 PM Atlantic Ballroom 3 Circuitry Underlying Obsessive-compulsive Disorder: Lessons from Deep Brain Stimulation and Ablative Surgery

3:00 PM – 5:30 PM Regency Ballroom 1 The Role of Inflammation in the Pathophysiology of Mood, Aggressive and Medical Disorders: A Deadly Combination

3:00 PM – 5:30 PM Regency Ballroom 3 Structural and Functional Brain Changes in Young People at Risk for Severe Mental Illness

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

#### Study Groups

7:30 PM – 9:00 PM Regency Ballroom 1 The Challenges of Designing and Interpreting Clinical Trials with Depot Antipsychotics

7:30 PM – 9:00 PM Regency Ballroom 2 Mental Illness, Violence and the Gun Control Debate: Evidence, Policy, Privacy and Stigma – On Behalf of the ACNP Ethics Committee

7:30 PM – 9:00 PM Regency Ballroom 3 New Models of Open Innovation to Rejuvenate the Biopharmaceutical Ecosystem, A Proposal by the ACNP Liaison Committee

7:30 PM – 9:00 PM Atlantic Ballroom 2 Medical and Non-Medical Use of Stimulant Drugs for Cognitive Enhancement

7:30 PM – 9:00 PM Atlantic Ballroom 1 The Assessment of Suicidal Ideation, Behavior & Risk: At Baseline; As a Measure of Clinical Outcome, and/or as a Treatment Emergent SAE

#### Tuesday, December 10, 2013

6:45 AM – 8:00 AM CDI Booster Session Room 220

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

7:00 AM – 8:00 AM Room 218 CME Institute Executive Directors Meeting

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 316 American Journal of Psychiatry Editorial Board Meeting

#### Mini-Panel Sessions

8:30 AM – 9:45 AM Diplomat Ballroom 1 & 2 Biochemical and Behavioral Pharmacology of Synthetic Cathinone Derivatives Found in Psychoactive Bath Salts Products

9:45 AM – 11:00 AM Diplomat Ballroom 1 & 2 After the Trauma: Developmental Trajectories from Childhood to Adult Psychiatric Disorders

#### Panel Sessions

8:30 AM – 11:00 AM Regency Ballroom 3 Augmentation of Antidepressant Response by Autoreceptor-mediated Mechanisms: Clinical Experience and Mechanisms of Action

8:30 AM – 11:00 AM Regency Ballroom 2 Neuroactive Steroids and Oxysterols as Endogenous Modulators of GABA and Glutamate Receptors: Basic Mechanisms and Therapeutic Implications

8:30 AM – 11:00 AM Atlantic Ballroom 1 The Future of Translational Research in Addiction

8:30 AM – 11:00 AM Atlantic Ballroom 2 At the Crossroads of Physics, Physiology, and Psychiatry: Rational Design of Noninvasive Neuromodulation Therapies

8:30 AM – 11:00 AM Atlantic Ballroom 3 Nutrition, Neurodevelopment, and Risk for Schizophrenia and Autism: From Epidemiology to Epigenetics

8:30 AM – 11:00 AM Regency Ballroom 1 Peripheral Immune and Endocrine Pathways as Markers of PTSD Risk and Symptom Development: Evidence from Prospective Studies

10:30 AM – 4:30 PM Great Hall 1 – 4 Poster Viewing

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

11:00 AM – 12:30 PM Room 217 ACNP Public Information Committee Meeting

#### Tuesday, December 10, 2013

11:30 AM - 1:30 PM **Regency Ballroom 2** Data Blitz Session

1:30 PM - 3:00 PM Regency Ballroom 1 Career Development Session

#### Mini-Panel Sessions

3:00 PM – 4:15 PM Diplomat Ballroom 1 & 2 Emerging Role of the Primary Cilium in Neuropsychiatric Disorders

4:15 PM - 5:30 PM Diplomat Ballroom 1 & 2 Adolescent Brain Development and Affective Disorders: The Role of Reward and Threat Circuitry

#### Panel Sessions

3:00 PM - 5:30 PM Regency Ballroom 1 Treating Addiction: Should We Aim High or Low?

3:00 PM - 5:30 PM Atlantic Ballroom 3 Anxiety and the Striatum, an Unusual Suspect

3:00 PM - 5:30 PM Atlantic Ballroom 2 Posttraumatic Stress Disorder: From Markers to Mechanisms

3.00 PM - 5.30 PM Atlantic Ballroom 1 Pathophysiology and Treatment of Obesity and Glucose Dysregulation in Schizophrenia

3:00 PM - 5:30 PM Regency Ballroom 2 **Biotypes of Psychosis** 

3:00 PM - 5:30 PM **Regency Ballroom 3** An Update from the Clinic on mGluR2/3 Approaches for Treating Schizophrenia -Understanding Human Circuit Engagement through to Recent Clinical Trials

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

6:00 PM - 11:00 PM Room 319 & 320 ACNP Council - Committee Chairs

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

#### Wednesday, December 11, 2013

| 6:45 AM - 8:00 AM   | Room 220 |
|---------------------|----------|
| CDI Booster Session |          |

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 Are the Putative Therapeutic Effects of Kappa-opioid Antagonists Explained by Anti-Stress Actions?

9:45 AM - 11:00 AM Diplomat Ballroom 1 & 2 Developing Imaging Biomarkers for Treatment Development: Beyond CNTRICS, CNTRaCs and NEWMEDS

#### Panel Sessions

8:30 AM - 11:00 AM Atlantic Ballroom 2 Manipulating BDNF-TrkB Signaling in Brain Disorders: Complex Regulation and Cellular and Systems Level Interactions as Novel Substrates for Translational Medicine?

#### **Program at a Glance** Wednesday, December 11, 2013

8:30 AM - 11:00 AM Regency Ballroom 1 The Ventromedial Prefrontal Cortex in Conditioning and Extinction in Chronically **Relapsing Disorders** 

8:30 AM - 11:00 AM Atlantic Ballroom 3 a4B2-Nicotinic Acetylcholine Receptors in Schizophrenia: Implications for Smoking Cessation and Therapeutics

8:30 AM - 11:00 AM Regency Ballroom 2 New Directions for Optogenetics: Investigating Plasticity Mechanisms Underlying Psychiatric Disorders

8:30 AM - 11:00 AM **Regency Ballroom 3** Alterations of the Glutamate Cycle in Severe Mental Illness

8:30 AM - 11:00 AM Atlantic Ballroom 1 Epigenetic Mechanisms in Neuropsychiatric Disorders

10:30 AM - 4:30 PM Great Hall 1 – 4 Poster Viewing

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

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

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

2:00 PM - 3:00 PM Room 209 PMG Board Meeting

#### Mini-Panel Sessions

3:00 PM - 4:15 PM Diplomat Ballroom 1 & 2 Human Brain Evolution and Comparative Epigenomics

4:15 PM - 5:30 PM Diplomat Ballroom 1 & 2 Intergenerational Transmission of Trauma -From Animal Models to Humans

#### Panel Sessions

3:00 PM - 5:30 PM **Regency Ballroom 2** Legal Damages: New Insights into Chronic Marijuana Effects on Human Brain Structure and Function

3:00 PM - 5:30 PM Atlantic Ballroom 3 Glutamate-dopamine Interactions in Nicotine and Cocaine Dependence: Biomarkers and Therapy Opportunities

3:00 PM - 5:30 PM Regency Ballroom 1 Public-private Repositioning Partnerships: A New Path to Achieve Target Validation and Proof of Concept for Novel CNS Indications

3.00 PM - 5.30 PM Atlantic Ballroom 2 Multidimensional Data Integration and Causality: A Systems Approach for Unraveling the Molecular Architecture of Mental Disorders

3:00 PM - 5:30 PM **Regency Ballroom 3** Early Stress and Emotion Dysregulation

3:00 PM - 5:30 PM Atlantic Ballroom 1 Neurobiological Regulation of Palatable Food Binging and Seeking

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

7.00 AM - 8.00 AM Room 212 ACNP/AsCNP/CINP/ECNP Meeting

Panel Sessions 8:00 AM – 10:30 AM Diplomat Ballroom 1 & 2 Molecular Regulation and Clinical Applications of Phosphodiesterase 4, the Major Enzyme for Degrading cAMP

8:00 AM - 10:30 AM Regency Ballroom 1 Naltrexone Revisited: New Findings Beyond Mu, Beyond Dopamine and Beyond Addiction

8:00 AM - 10:30 AM Regency Ballroom 3 Understanding Neurodevelopmental Risk Factors Leading to Anxiety and Depression to Inform Novel Early Interventions in Vulnerable Children

8:00 AM - 10:30 AM Atlantic Ballroom 1 Brain on Fire: Inflammation in Neurological and Psychiatric Illness

8:00 AM - 10:30 AM Atlantic Ballroom 2 Melatonin and Its Receptors: Important Players in Major Depressive Disorder

8:00 AM - 10:30 AM Atlantic Ballroom 3 Building a More Predictive Mouse: Humanized Mouse Models for Neuropsychiatric Disorders

8:00 AM - 10:30 AM Regency Ballroom 2 Broadening the Trajectories of Risk: Specific and Non-specific Markers of Risk of Psychopathology

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

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12:00 PM - 2:30 PM Diplomat Ballroom 1 & 2 Novel Molecules and Mechanisms in Vulnerability and Resilience Throughout Life

12:00 PM - 2:30 PM **Regency Ballroom 1** Cognition, Biomarkers, and Longitudinal Outcomes in Geriatric Mood Disorders

12:00 PM - 2:30 PM Regency Ballroom 3 The Insula Salience Network: Alterations in Its Connectivity in Developmental, Anxiety, Mood and Personality Disorders

12:00 PM - 2:30 PM Atlantic Ballroom 1 Strategies for the Development of Novel Therapies for Schizophrenia: From Clinic to Laboratory (And Back Again)

12:00 PM - 2:30 PM Atlantic Ballroom 2 Behavioral, Endocrine, and Neural Plasticity Changes Reflecting Stress Associated with Mouse and Monkey Models of Heavy Alcohol Drinking

#### Tuesday, December 10th

#### **Morning Mini-Panel Sessions**

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| •      | Nutrition, Neurodevelopment, and Risk for Schizophrenia and       |
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| •      | Peripheral Immune and Endocrine Pathways as Markers of            |
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|   | Reflecting Stress Associated with Mouse and Monkey Models   |
|   | of Heavy Alcohol Drinking   |
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#### **Acknowledgments**

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AbbVie, Inc. Alkermes, Inc. Amgen, USA Astellas Pharma Eisai Medical Research, Inc. **Envivo Pharmaceuticals** Forest Laboratories. Inc. Genentech H. Lundbeck A/S, Denmark Hoffmann-LaRoche, Inc. Janssen Pharmaceuticals, Inc. Lilly USA, LLC Merck Novartis Pharmaceuticals Corporation Otsuka America Pharmaceutical, Inc. Otsuka Pharmaceutical Development and Commercialization, Inc. Sunovion Pharmaceuticals, Inc. Takeda Pharmaceuticals Targacept, Inc.

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> Lilly USA, LLC Otsuka America Pharmaceutical, Inc.

#### Council

#### **Officers and Council**

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|---------------------------|------------------|
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#### **Program Committee**

#### 2013 Program and Scientific Communications Committee

| Chair           | Randy Blakely    |
|-----------------|------------------|
| Co-Chair        | Pat Levitt       |
| Council Liaison | Peter W. Kalivas |

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Ad Hoc: Joseph Callicott David Grandy Robert Greene James Sutcliffe Jeremy Veenstra-Vander Weele

#### **General Information**

#### **Dates and Location**

DatesSunday, December 8, 2013 - Thursday, December 12, 2013LocationThe 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, <u>www.acnp.org</u>.

#### **Itinerary Planner**

All scientific registrants will be able to access the itinerary planner for the 52nd ACNP Annual Meeting at <u>http://www.eventscribe.com/2013/ACNP</u>.

#### **ACNP Executive Office**

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#### **Continuing Medical Education**

The 2013 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)*<sup>TM</sup>. 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 and the ACNP 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 <u>acnp@acnp.org</u>.

#### **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 <u>www.acnp.org</u> (click the Annual Meeting tab). All evaluations must be completed by January 23, 2014.

#### **Future ACNP Annual Meetings**

| Dates                 | Hotel                             | Location                 |
|-----------------------|-----------------------------------|--------------------------|
| December 7 - 11, 2014 | JW 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**

Sydney Spector October 26, 2012

Peter B. Dews November 2, 2012

Svein G. Dahl December 8, 2012

Daniel W. Hommer January 2, 2013

Albert Weissman July 11, 2013

Ernest Hartmann August 7, 2013

Candace B. Pert September 12, 2013

|       | ACNP 52nd Annual Meeting • Final Program |
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| Notes |  |
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#### Neuropsychopharmacology Reviews Plenary

#### "Frontiers in Discovery and Neurotherapeutics"

Co-Chairs: Carol Nilsson and Kathryn Cunningham

| 8:30 a.m.  | Integrated Omics Approach to Neuropharmacological Studies<br>Mark Emmett  |
|------------|---|
| 8:55 a.m.  | Approaches for the Small-volume Analysis of Cell–cell<br>Signaling Molecules in the Brain<br>Jonathan Sweedler                              |
| 9:20 a.m.  | Heteromeric Dopamine Receptor Signaling Complexes:<br>Emerging Neurobiology and Relevance to Neuropsychiatric<br>Disease<br>Susan R. George |
| 9:45 a.m.  | Use of CSF Biomarkers for Alzheimer's Disease in Clinical<br>Trials<br><i>Kaj Blennow</i>   |
| 10:10 a.m. | New Technologies Promise Revitalization of Psychiatric<br>Therapeutics<br>Steven E. Hyman   |
| 10:35 a.m. | Discussion<br>Carol Nilsson and Kathryn Cunningham  |

8:30 a.m. – 11:30 a.m. *Neuropsychopharmacology Reviews* Plenary Regency Ballroom 1 & 2

#### **Integrated Omics Approach to Neuropharmacological Studies**

<u>Mark Emmett</u> University of Texas Medical Branch-Galveston (UTMB)

Understanding the response of neurological cell populations (normal vs. diseased, treated vs. untreated, etc.) and evidence that these cell populations are "drivers" of disease underscores the importance of understanding detailed biology at the molecular level of these cells. Utilizing an integrated omics (systems biology) approach to probe the response of these cells will guide the rational design of therapeutic treatment(s). Due to the complexity of the system biology approach, this research is most often accomplished through multi-disciplinary and interinstitutional collaborations because no one laboratory has expertise in all the technologies required.

Systems biology research is built on knowledge derived from global datasets measured in patterns of response of the transcriptome, proteome, glycome, lipidome, and metabolome. To obtain knowledge of the signaling pathways involved in the maintenance of the normal vs. diseased state or treated vs. untreated states, quantitative phosphoproteomic, glycomic, transcriptomic, lipidomic and metabolomic data sets are collected on each sample (with biological and analytical replicates). These large data sets are then processed with innovative mathematical-computational algorithms tailored to define correlations between the data sets and graphical modeling to reconstruct pathways that are not yet defined in the scientific literature. The focus is to identify and characterize pathway reactions that will bridge the final gap and, for the first time, enable a mechanism to a) understand cellular responses, b) identify new targets and diagnostic biomarkers and c) design appropriate therapeutic interventions specifically targeting disease.

This approach has been developed and applied to the study of glioma and data will be presented linking omics technologies in these studies. All of the technologies developed for these glioma studies, can be directly applied to other neurological disease states as well.

#### **Integrated Omics Approach to Neuropharmacological Studies** (continued)

#### Mark Emmett

Dr. Emmett's research experience is diverse, ranging from a Master's in Microbiology/Molecular Biology to a PhD in Biochemistry/Neuropharmacolgy. He has substantial research experience in the pharmaceutical industry in neuroscience drug discovery groups. He has extensive mass spectrometry experience with emphasis on ultra-high sensitivity analysis of biological samples using Nano-scale Liquid Chromatography, Microelectrospray Mass Spectrometry, which he specifically developed for his Ph.D. research. At the NHMFL, he applied this technology to Fourier Transform Ion Cyclotron Resonance (FT-ICR) Mass Spectrometry in the analysis of endogenous biological compounds. Has has spent the last 17 years in the FT-ICR group at the NHMFL developing applications for and modifications of high-resolution FT-ICR MS instrumentation with an emphasis on interfacing chromatography to MS (LC, CE, SFC, etc), thus he is well versed in analytical methodology and instrumentation. While at the NHMFL, he began working in the field of cancer research (1997-present) he has focused mainly on Neuro-Oncology, but has also worked in breast, prostate and gastro-intestinal stromal tumors (GIST). He established a program of lipidomics in cancer research and with collaborators established a multi-disciplinary research group enabling a systems biological approach integrating genomics, transcriptomics, proteomics, glycomics, lipidomics, metabolomics and phenotypic responses to the study of cancer focusing on the identification of novel biomarkers and therapeutic targets.

8:30 a.m. – 11:30 a.m. *Neuropsychopharmacology Reviews* Plenary Regency Ballroom 1 & 2

#### Approaches for the Small-volume Analysis of Cell-cell Signaling Molecules in the Brain

Jonathan Sweedler University of Illinois

The interactions between neurons in our brain are partially controlled by the chemicals that surround the neurons. In the postgenomic era, one expects the suite of chemical players in a brain region to be known and their functions uncovered. However, many cell-to-cell signaling molecules remain poorly characterized and for those that are known, their localization and dynamics are oftentimes unknown. Significant challenges in the characterization of intercellular signaling molecules arise in part from their large chemical diversity and their broad range of concentrations. Neurotransmitters and neuromodulators vary from small gaseous molecules such as nitric oxide to larger peptides that are only bioactive with particular posttranslational modifications. The enormous biochemical complexity of nervous system where even adjacent cells often have very different and dynamic metabolic profiles necessitates development and application of technologies capable characterization of the neurometabolome on the individual cell level.

Here, we present a suite of bioanalytical approaches that allow the investigation of defined brain regions down to individual neurons. These approaches include capillary electrophoresis with laser induced fluorescence and mass spectrometric detection, and direct mass spectrometric-based profiling and imaging. Several applications of single cell microanalysis are highlighted: investigating novel indolamine neurochemistry, determining the role of d-amino acids in the brain, and uncovering novel neuropeptides. Specifically, new serotonin-related compounds and literally hundreds of new neuropeptides have been characterized in welldefined neuronal networks, and in several cases, the functional roles of these molecules described. Discovery of new neurochemical pathways often relies not only on structural information provided by traditional mass spectrometry but also requires knowledge on the spatial and temporal dynamics of these signaling molecules in the brain. Imaging mass spectrometry and dynamic sampling of the extracellular environment are used for elucidating novel cell to cell signaling

#### Approaches for the Small-volume Analysis of Cell-cell Signaling Molecules in the Brain

#### Jonathan Sweedler

molecules in a range of neuronal model systems. Our overarching goal is to uncover the complex chemical mosaic of the brain and pinpoint key cellular players in physiological and pathological processes.

Prof. Jonathan Sweedler's research involves analytical neurochemistry. He develops new measurement tools that enable small scale chemical analysis, and applies these tools to understanding the chemistry occurring in the brain. Sweedler is the Director of the School of Chemical Sciences, holds the James R. Eiszner Family Chair in Chemistry and is affiliated with the Departments of Molecular and Integrative Physiology and Bioengineering, the Neuroscience program, the Beckman Institute of Science and Technology, and the Institute of Genomic Biology. Sweedler has authored or coauthored over 320 peerreviewed publications, has 14 patents issued, and has delivered over 380 invited lectures to universities, companies, and at scientific meetings. His scientific contributions have been recognized by numerous awards including the Ralph N. Adams Award, the Heinrich-Emanuel Merck Prize, and the American Chemical Society's Analytical Chemistry Award. Sweedler is currently the Editor-in-Chief of Analytical Chemistry.

8:30 a.m. – 11:30 a.m. *Neuropsychopharmacology Reviews* Plenary Regency Ballroom 1 & 2

#### Heteromeric Dopamine Receptor Signaling Complexes: Emerging Neurobiology and Relevance to Neuropsychiatric Disease

<u>Susan R. George</u> University of Toronto

The involvement of the dopaminergic system in many mental health disorders has resulted in the pharmacological targeting of dopamine transmission as the mainstay of the treatment of several disorders, such as schizophrenia, addiction, depression and ADHD. However, the currently available therapies mostly target a single dopamine receptor, limiting the scope of treatment. The recognition that dopamine receptors participate in forming heteromeric complexes, often with distinct anatomical localization in brain as well as signaling and functional properties has significantly expanded the range of physiologically relevant signaling complexes present beyond the five known dopamine receptors. Furthermore, as the physiology and disease relevance of these receptor heteromers is further understood, their ability to exhibit pharmacological and functional properties distinct from their constituent receptors, or ability to modulate the function of endogenous homomeric receptor complexes, may allow for the development of alternate therapeutic strategies and provide new avenues for rational drug design. The emerging neurobiology of the best characterized dopamine receptor heteromers such as the D1-D2, D2-D4 and other complexes, their physiological relevance in brain, and the potential role of these receptor complexes in neuropsychiatric disease will be discussed. The value of these heteromers as targets for future drug development is highlighted and how designing drugs to selectively activate or inactivate these dopamine receptor heteromers may have enormous potential to aid in the search for novel and clinically efficacious pharmacotherapies for these often difficult to treat clinical disorders.

Dr. Susan George is a clinician-scientist with a particular interest in G protein coupled receptor signaling in brain mediated by dopamine and opioid receptors, as they relate to neuropsychiatric disease mechanisms, especially related to drug addiction and schizophrenia.

#### Use of CSF Biomarkers for Alzheimer's Disease in Clinical Trials

<u>Kaj Blennow</u> University of Gothenburg

Research advances have given detailed knowledge on AD molecular pathogenesis, which has been translated into new drug candidates with disease-modifying potential. The majority of the drug candidates evaluated in clinical trials are directed against amyloid- $\beta$  (A $\beta$ ). The main principles for anti-A $\beta$  drugs include active and passive AB immunotherapy and AB lowering drugs (BACE1 and  $\gamma$ -secretase inhibitors). However, there is a growing list of anti-A $\beta$  clinical trials in which it has not been possible to identify any clinical benefit. This has caused concern that A $\beta$  deposition may merely be a bystander, and not the cause, of the disease, and that the amyloid hypothesis only is valid for the familial form of AD. A more optimistic view is that the design of anti-A $\beta$  trials will need refinement to give the drug a fair chance to show a positive clinical effect. There is a growing consensus that biomarkers (CSF tests, amyloid PET and MRI measurements) will have a critical role in this new era in AD drug development. Diagnostic biomarkers will be essential to allow initiation of treatment in the pre-dementia stages, before neurodegeneration is too advanced. Primary biomarkers applied in early clinical phases to study the pharmacodynamic effects of a drug will be important to guide the decision to only advance compounds with target engagement proven in man on A $\beta$  metabolism into large and expensive phase II or III clinical trials. Downstream biomarkers will provide evidence that the drug candidate affects the central neuropathology, which, together with a positive effect on cognition, will be necessary to claim a label as a disease-modifying drug.

Kaj Blennow took his medical degree (MD) in 1984, and holds a Specialist Competence in both General Psychiatry and in Cinical Chemistry. He is Head of the Clinical Neurochemistry Lab at Sahlgrenska University Hospital, Gothenburg, Sweden, and Professor in Clinical Neurochemistry at the Sahlgrenska Academy, Mölndal campus at University of Gothenburg, Sweden. The Clinical Neurochemistry research group includes 15 post-docs and 6 PhD students, 8 laboratory technicians. The major research areas are cerebrospinal fluid biochemical markers, clinical proteomics, and the neurochemical pathogenesis of Alzheimer's disease and other brain disorders. Blennow has published more than

## Use of CSF Biomarkers for Alzheimer's Disease in Clinical Trials (continued)

#### Kaj Blennow

550 original research papers and 80 review articles in peer-reviewed journals, and has a H-index above 80. He has received a number of scientific awards, such as The CINP Award (1992), the IPA Research Award (1993), the Alois Alzheimer Research Award (2001), the ECNP Clinical Research Award (2010), the Henry Wisniewski Lifetime Achievement Award in Alzheimer's Disease Research (2011), and the International Federation of Research in Alzheimer's Disease Grand Prix in Research (2013)

## New Technologies Promise Revitalization of Psychiatric Therapeutics

<u>Steven E. Hyman</u> Broad Institute of Harvard and MIT

To the detriment of patients, the pharmaceutical industry has de-emphasized psychiatric illness. Reasons include a dearth of convincing new molecular targets, disillusionment with current animal "models", and lack of treatment biomarkers. Yet there is reason for optimism. Unbiased genomic methods are beginning to identify pathways. New stem cell and genome engineering technologies promise cellular models to complement animals. Recognition that psychiatric disorders are better conceptualized as spectra and dimensionally should help clear away the obstacles to progress posed by current fictive diagnostic categories

Steven E. Hyman, M.D. is Director of the Stanley Center for Psychiatric Research at the Broad Institute of MIT and Harvard and also serves as Harvard University Distinguished Service Professor of Stem Cell and Regenerative Biology. From 2001 to 2011, Hyman served as Provost of Harvard University, the University's chief academic officer. From 1996 to 2001, he served as director of the U.S. National Institute of Mental Health (NIMH), where he emphasized investment in neuroscience and emerging genetic technologies, and initiated a series of large practical clinical trials to inform practice. Hyman is the editor of the Annual Review of Neuroscience, President of the International Neuroethics Society, and a member of the Institute of Medicine of the U.S. National Academies where he serves on the governing Council, the Board of Health Science Policy, and chairs the Forum on Neuroscience and Nervous System Disorders. He is a fellow of the American College of Neuropsychopharmacology, a fellow of the American Academy of Arts and Sciences, a fellow of the American Association for the Advancement of Science, and a Distinguished Life Fellow of the American Psychiatric Association.

1:00 p.m. – 2:30 p.m. Institute Director's Session Regency Ballroom 1 & 2

#### **NIH Institute Director's Session**

Chair: David Lewis

Panelists:

Kenneth Warren NIAAA

Thomas Insel NIMH

Nora Volkow NIA

PL

#### **Hot Topics**

Co-Chairs: Randy Blakely and Pat Levitt

- 2:30 p.m. Dysregulated Neural Response to Social Evaluation In Bullied Adolescents: A Potential Mechanism that Promotes Risk for Social Anxiety Disorder Johanna Jarcho
- 2:41 p.m. Buspirone Blocks Dopamine D3 Receptors in the Non-Human Primate Brain When Administered Orally *Nora Volkow*
- 2:52 p.m. Naloxone-Reversible Modulation of Pain Circuitry by Left Prefrontal Repetitive Transcranial Magnetic Stimulation Joseph Taylor
- 3:03 p.m. DREADDs in Drosophila: Pharmacogenetic Control of Neurons and Behavior in the Fly *Charles Nichols*
- 3:14 p.m. Striatal Activation Induced by mGluR2 Positive Allosteric Modulation Correlates with Negative Symptom Reduction in Schizophrenia Daniel Wolf
- 3:25 p.m. Prenatal Exposure to Maternal Infection Alters Neonatal Brain Structure John Gilmore
- 3:36 p.m. Application of Sequencing, Fatty Acid Profiling, and Metabolomics Investigations to Explore Pathogenesis and Treatment Strategy for Anorexia Nervosa *Pei-an Betty Shih*

#### **Hot Topics**

Co-Chairs: Randy Blakely and Pat Levitt 3:47 p.m. Retinoid-Related Orphan Receptor Alpha: A Novel Candidate Gene for Psychiatric Disease Joseph Friedman 3:58 p.m. Automated Analysis of Disorganized Communication Predicts Transition to Psychosis Among Clinical High Risk Patients Cheryl Corcoran 4:09 p.m. Dissecting Nucleus Accumbens Dynorphin Neurons in Aversion and Reward Michael Bruchas 4:40 p.m. Mismatch Negativity Predicts Psychosis Onset and is Associated with Plasma Markers of Inflammation in Youth at Clinical High **Risk for Psychosis** Daniel Mathalon Age-related Sperm DNA Methylation Changes are Transmitted 4:51 p.m. to Offspring and Associated with Abnormal Behavior and Dysregulated Gene Expression Maria Milekic Prospective Examination Of Prepulse Inhibition In OIF/OEF 5:02 p.m. Marines Suggests Reduced Sensorimotor Gating Is A Preexisting Factor In Those That Develop PTSD After Combat Deployment Dean Acheson Disrupting AMPA Receptor Endocytosis Restores the Ability to 5:13 p.m.

Form New, and Enables the Recovery of Old, Memories in Mice Genetically Designed to Mimic Alzheimer's Disease Sheena Josselyn

#### **Hot Topics**

Co-Chairs: Randy Blakely and Pat Levitt

- 5:24 p.m. Selective Effects of the 5-HT2C Receptor Agonist Metachlorophenylpiperazine (mCPP) on Intake of a Palatable Snack Food in Healthy Female Volunteers: Correlation with Regional Brain Activations Measured by BOPLD fMRI *Colin Dourish*
- 5:35 p.m. Locus Specific Epigenetic Reprogramming: Bidirectional Regulation of the FosB Gene Using Synthetic Transcription Factors In Vivo Elizabeth Heller
- 5:46 p.m. Suicidal Ideation in Depressed New Mothers: Relationship with Childhood Trauma and Sleep Disturbance Dorothy Sit
- 5:57 p.m. The Contribution of Adult Hippocampal Neurogenesis to Fear Memory Generalization *Mazen Kheirbek*
- 6:08 p.m. Intranasal Ketamine in Treatment-Resistant Depression *Kyle Lapidus*
- 6:19 p.m. Specific Elevation of βCaMKII in the Lateral Habenula Lead to Core Symptoms of Depression *Fritz Henn*

#### Dysregulated Neural Response to Social Evaluation In Bullied Adolescents: A Potential Mechanism that Promotes Risk for Social Anxiety Disorder

Monday, Poster #48

<u>Johanna M. Jarcho</u>, Megan Davis, Ellen Leibenluft, Nathan Fox, Tomer Shechner, Daniel S. Pine, Eric Nelson National Institute of Mental Health

**Background:** Peer victimization is a risk factor for social anxiety disorder (SAD) that engenders fear of negative evaluation, the primary symptom of SAD. While available treatments for SAD can reduce symptoms, they rarely result in full remission. Interventions that target neural circuits dysregulated in adolescent SAD may enhance treatment efficacy. An important first step toward developing such interventions is to isolate dysregulated neural circuits shared by early adolescents with SAD, and at risk for SAD due to peer victimization. Treating early adolescents may alleviate acute symptoms before they become chronic, thereby facilitating normative development, and preventing the high cost of adult SAD. Progress toward this goal has been hindered by limitations in neuroimaging paradigms, which bear little resemblance to contexts that precipitate the primary symptoms of adolescent SAD, or to contexts in which peer victimization occurs. An fMRI paradigm that evokes fear of negative evaluation while modeling an ecologically valid context for bullying may address these limitations, and thereby facilitate the development of novel interventions. To this end, we developed the Virtual School paradigm, which explicitly models unpredictable social evaluation in an ecologically valid classroom setting. Here we present data from the first fMRI study to utilize the Virtual School paradigm. In this study, we assess brain function as healthy adolescents with high or low exposure to victimization anticipate social evaluation from predictable and unpredictable peers. We hypothesize that adolescents with high, relative to low, exposure to peer victimization will differentially engage fronto-striatal-amygdala circuits, implicated in self-reflection, reward, and threat processing, when anticipating unpredictable social feedback from peers.

**Methods:** Healthy adolescents (N = 22; M = 10.73 years; SD = .46) with high and low exposure to peer victimization are told that they are the "New Kid" at our

#### Dysregulated Neural Response to Social Evaluation In Bullied Adolescents: A Potential Mechanism that Promotes Risk for Social Anxiety Disorder

Monday, Poster #48 (continued)

#### Johanna M. Jarcho

Virtual School. They generate a cartoon avatar and personal profile they believe will be shown to a purported group of "Other Students." Participants learn the Other Students have a reputation for being 'Nice, 'Unpredictable,'or 'Mean.' Reputation comprehension is assessed prior to completing the Virtual School paradigm in the fMRI scanner. During the task, participants enter classrooms populated by Other Students. For each trial, participants are cued to anticipate social evaluation when "Typing..." appears above one of the Other Students. Because Other Students have an established reputation, participants anticipate different types of social evaluation from each peer. Unpredictable peers then provide 50% positive and negative feedback, while Nice and Mean peers provide 100% positive or 100% negative feedback (respectively). Participants then make a positive, negative, sarcastic, or avoidant response to peer social evaluation.

**Results:** Replicating prior behavioral findings (Jarcho et al., 2013), adolescents learned Other Student reputations, made responses during the task that varied by peer reputation and feedback, and believed they were interacting with real peers (100% deception). As hypothesized, brain activity during anticipated social evaluation varied based on participant exposure to peer victimization and Other Student reputation (p < .005; cluster extent > 70 voxels). Specifically, while anticipating unpredictable, relative to predictable positive or negative social evaluation, victimized adolescents exhibited heightened activity in frontostriatal-amygdala circuits compared with non-victimized adolescents.

**Discussion:** Exposure to peer victimization is associated with differential engagement of a brain network implicated in self-reflection, reward, and threat processing. This engagement varies depending on the type of social evaluation (i.e., uncertain vs. certain) victimized adolescents anticipate. These data suggest one mechanism by which exposure to bullying may lead to SAD is through disruptions in neural circuits engaged by unpredictable social evaluation. Longitudinal studies are needed to more fully test this hypothesis.

#### **Buspirone Blocks Dopamine D3 Receptors in the Non-Human Primate Brain When Administered Orally**

Monday, Poster #139

Sung Won Kim, Joanna Fowler, Phil Skolnick, Yeona Kang, Dohyun Kim, <u>Nora</u> <u>D. Volkow</u> National Institute on Drug Abuse

Tuttohai Institute on Diug Touse

**Background:** Converging lines of evidence indicate that dopamine  $D_3$  receptor  $(D_3R)$  antagonists may be effective as treatments/medications for substance use disorders (SUDs) in animal and human. However, no selective  $D_3R$  antagonists are clinically available for testing this hypothesis. Buspirone (Buspar®) originally characterized as a selective 5-HT<sub>1A</sub> partial agonist, has been used as an anxiolytic for more than 25 years. However, buspirone also binds to  $D_3R$  and  $D_4R$  with high affinity as an antagonist and with lower affinity to  $D_2R$  *in vitro*. Recently, this azapirone has been shown to interfere with cocaine reward in non-human primates. Here we evaluate buspirone's ability to block  $D_3R$  in the non-human primate brain and compared it to  $D_2R$  and  $D_1R$  blockade in pharmacologically-relevant and safe dose ranges.

**Methods:** In six female baboons, we used PET with [<sup>11</sup>C]PHNO (D<sub>3</sub>R-preferring radioligand), [<sup>11</sup>C]raclopride (D<sub>2</sub>R/D<sub>3</sub>R radioligand) and [<sup>11</sup>C]NNC-112 (D<sub>1</sub>R radioligand) to measure occupancy of oral versus parenteral (IM) buspirone at multiple time points after drug administration. One of major metabolites, 6'-OH buspirone (IM, 1 mg/Kg) was also administered at 3 hrs before [<sup>11</sup>C]PHNO scan. **Results:** Intramuscular administration of buspirone (0.19 and 0.5 mg/Kg) showed high occupancy (50-85%) at 15 min and then rapid wash-out by 2 hrs in a dose dependent manner for both [<sup>11</sup>C]PHNO and [<sup>11</sup>C]raclopride PET studies. Interestingly, oral buspirone (3 mg/Kg) significantly blocked [<sup>11</sup>C]PHNO binding in globus pallidus and substantia nigra (55-74% after 3 hours), while blockade of [<sup>11</sup>C]raclopride was minimal (10%) in striatum. One of buspirone's metabolites, 6'-OH buspirone (D<sub>3</sub>R antagonist) significantly also blocked (89%) [<sup>11</sup>C]PHNO binding in substantia nigra. No blockade was observed for [<sup>11</sup>C]NNC-112 by both oral and parental administration of buspirone.
#### **Buspirone Blocks Dopamine D3 Receptors in the Non-Human Primate Brain When Administered Orally**

Monday, Poster #139 (continued)

Nora D. Volkow

**Discussion:** Since [<sup>11</sup>C]PHNO binding has been known to reflect  $D_3R$  binding predominantly and little blockade was observed in [<sup>11</sup>C]raclopride ( $D_2R/D_3R$  radioligand) binding after oral buspirone, we conclude that oral buspirone/its metabolites blocked  $D_3R$  significantly and would merit testing for therapeutic efficacy in SUDs in human.

## Naloxone-Reversible Modulation of Pain Circuitry by Left Prefrontal Repetitive Transcranial Magnetic Stimulation

Monday, Poster #61

<u>Joseph J. Taylor</u>, Jeffrey J. Borckardt, Melanie Canterberry, Xingbao Li, Colleen A. Hanlon, Truman Brown, Mark S. George Medical University of South Carolina

**Background:** A 20-minute session of 10 Hz repetitive transcranial magnetic stimulation (rTMS) of Brodmann Area (BA) 9 of the left dorsolateral prefrontal cortex (DLPFC) can produce analgesic effects on postoperative and laboratory-induced pain. This analgesia is blocked by pretreatment with naloxone, a  $\mu$ -opioid antagonist. The purpose of this sham controlled, double blind, crossover study was to identify the neural circuitry that underlies the analgesic effects of left DLPFC rTMS and to examine how the function of this circuit, including midbrain and medulla, changes during opioid blockade.

**Methods:** Fourteen healthy volunteers were randomized to receive intravenous saline or naloxone immediately prior to sham and real left DLPFC rTMS on the same experimental visit. One week later, each participant received the novel pretreatment but the same stimulation paradigm. Using short sessions of heat on capsaicin-sensitized skin, hot allodynia was assessed during 3T functional magnetic resonance imaging (fMRI) scanning at baseline, post-sham rTMS, and post-real rTMS. Data were analyzed using whole-brain voxel-based analysis as well as time series extractions from anatomically defined regions of interest representing midbrain and medulla.

**Results:** Consistent with previous findings, real rTMS significantly reduced hot allodynia ratings. This analgesia was associated with elevated BOLD signal in DLPFC and diminished BOLD signal in the anterior cingulate, thalamus, midbrain and medulla during pain. Naloxone pretreatment largely abolished rTMS-induced analgesia as well as rTMS-induced attenuation of BOLD signal response to painful stimuli throughout pain processing regions, including midbrain and medulla.

**Discussion:** These preliminary results suggest that left DLPFC rTMS drives topdown opioidergic analgesia.

# **DREADDs in Drosophila: Pharmacogenetic Control of Neurons and Behavior in the Fly**

Monday, Poster #180

<u>Charles D. Nichols</u>, Jaime Becnel, Oralee Johnson, Zana Majeed, Vi Tran, Bangning Yu, Bryan L. Roth, Robin L. Cooper, Edmund K. Kerut Louisiana State University Health Sciences Center

Background: Drosophila melanogaster is an important genetic model system that has provided much information on the molecular basis of behaviors conserved with mammalian systems and psychiatric disorders. These include sleep, aggression, social interaction, learning and memory, and response to drugs of abuse that are all mediated by similar and fundamentally shared mechanisms involving neurotransmitters like serotonin, dopamine, glutamate, and GABA. One of the advantages of the fly is the extensive toolkit of genetic methods to manipulate gene expression in the fly. In combination with the ability to precisely control temporal and spatial expression in the fly, there are several methods used to conditionally control neuronal activity that include temperature sensitive blockade of synaptic vesicle recycling with *shibire*<sup>ts</sup>, constitutive activation/inactivation with NaChBac/Kir channels, temperature regulated activation/inactivation with TrpA/TrpM channels, and light regulated channelrhodopsins. A disadvantage to these methods is that they each are all essentially switches, and either turn the neuron all on or all off, and have little to no dose responsive control. We have now adapted Designer Receptors Exclusively Activated by Designer Drugs (DREADD) technology to the fruit fly to provide true dose responsive control of neuronal function and behavior through manipulation of GPCR receptors and downstream effector pathways. Unlike these other methods, additional equipment like dedicated temperature incubators or blue light sources and fiber optics are not required, activating drug is simply fed to the fly. Furthermore, conditional control of activity is reversible. Due to the ubiquitous nature of GPCRs, this system will also be useful in the examination of the role of signal transduction pathway effectors in almost every tissue of the fly, and is not limited to study of only neurons and behaviors.

**Methods:** UAS-DREADD constructs were created for each of the three primary mammalian muscarinic DREADDs (hM4Di, a silencing receptor coupled to

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# **DREADDs in Drosophila: Pharmacogenetic Control of Neurons and Behavior in the Fly**

Monday, Poster #180 (continued)

#### Charles D. Nichols

Gi; hM1Dq, an activating receptor coupled to Gq; rM3DBs, coupled to Gs and increases in cAMP levels), and transgenic fly strains created for each. DREADDs were expressed in discreet neuronal circuits and tissues using various GAL4 drivers and the ability of activation of the DREADDs to control behaviors was examined in a panel of behaviors that included sensory perception, learning and memory, circadian, and courtship. Effector pathway responses were measured in tissue culture. Neuronal activity was measured by real-time live cell calcium imaging in larval brain ventral ganglia neurons using GcAMP and confocal microscopy. Heart rate control was measured using larval heart preparations and light microscopy. The activating ligand clozapine-N-oxide (CNO) was administered by feeding to flies in the food, or application in media.

**Results:** DREADD activation was found to dose responsively and reversible control behaviors, neuronal activity, and physiological processes by simple feeding or application of activating ligand, CNO.

**Discussion:** We have successfully translated DREADD technology from systems to Drosophila. DREADD activation confers dose mammalian responsive and reversible control over not only signal transduction and effector pathways, but also neuronal activity, behaviors, and physiological processes. DREADDs provide an additional level of more fine control of neurons and circuits than the current switch-based all on or all off approaches. This control to only partially activate or inactivate a neuron acutely or chronically allows us to study more subtle behaviors that may be masked by more aggressive methods. In our previous work, we generated several genetic tools for examination of the Drosophila serotonin system, and defined a role for serotonin in many behaviors relevant to neuropsychiatric disorders like social interaction and learning and memory. We are now incorporating DREADD technology into our study of serotonin neuropharmacology in the fly to enhance our discovery of conserved mechanisms underlying behaviors that will ultimately enhance our understanding of human psychiatric diseases.

# Striatal Activation Induced by mGluR2 Positive Allosteric Modulation Correlates with Negative Symptom Reduction in Schizophrenia

Monday, Poster #52

<u>Daniel Wolf</u>, Kosha Ruparel, Bruce Turetsky, Christian Kohler, Theodore D. Satterthwaite, Mark Elliott, Mary March, Alan Cross, Mark Smith, Stephen R. Zukin, Ruben C. Gur, Raquel E. Gur University of Pennsylvania Department of Psychiatry

**Background:** Cognitive deficits and negative symptoms contribute strongly to disability in schizophrenia, and are resistant to existing medications, creating a critical need for novel therapeutic targets and agents. Inspired by the glutamate hypothesis, recent drug development efforts have focused on ameliorating putative deficits in NMDA signaling. In animal models, mGluR2/3 agonists and mGluR2 positive allosteric modulators (PAMs) have reversed the physiologic and behavioral effects of NMDA receptor antagonists. However the clinical utility of such agents remains uncertain, and their impact on neural circuit function in humans remains unknown. Progress in this area will benefit from studying novel agents targeting cognition and negative symptoms using integrative paradigms that incorporate clinical, neurocognitive performance and neurophysiological measures in order to evaluate early signals of efficacy. We therefore performed this fMRI study as part of a Phase 1 pilot study (NCT00986531) evaluating the mgluR2 PAM AZD8529 as an adjunctive treatment for cognitive deficits and negative symptoms. We hypothesized the drug would improve cognition and symptoms, and that clinical improvements would correlate with changes in fMRI activation.

**Methods:** Subjects with complete fMRI data were 26 patients (10 female) with DSMIV schizophrenia, stably treated with antipsychotics. 3T MRI scanning was performed following three days treatment with AZD8529 (80mg once-daily) or placebo. The study design was double blind, placebo-controlled, counterbalanced within-subject crossover, with a 14-day washout between drug and placebo phases. During fMRI scanning, subjects performed a fractal n-back task (0, 1, 2, and 3-back block design), as well as a continuous performance task and an emotion

## Striatal Activation Induced by mGluR2 Positive Allosteric Modulation Correlates with Negative Symptom Reduction in Schizophrenia

Monday, Poster #52 (continued)

#### Daniel Wolf

identification task. We focus here on the n-back task; the other two tasks did not show significant drug effects. fMRI analysis focused on task-activated regions of interest including anterior cingulate (ACC) and dorsolateral prefrontal cortex (DLPFC). Exploratory whole-brain voxelwise analyses were also conducted to test for drug effects outside of the a priori ROIs.

**Results:** No significant effects of drug were found on average clinical symptoms or on behavioral performance during in-scanner or out-of-scanner tasks. BOLD activation in DLPFC and ACC showed expected increases with working memory load. Relative to placebo, drug increased activation in ACC (p=.031). Although activation trended higher on drug in left and right DLPFC there was no significant main effect of drug in these regions. An exploratory whole brain analysis demonstrated the most robust drug effects in basal ganglia; we therefore also conducted region of interest analyses in in right and left caudate, putamen, and pallidum. The main effect of drug was significant in all these regions due to increased activation by drug compared to placebo (L Caudate, p<0.001; R Caudate p<0.001; L Putamen p=0.0014; R Putamen p<0.001; L Pallidum p=0.017; R Pallidum p<0.001). No regions showed significant interaction effects of drug with working memory load level. Subjects who showed greater caudate activation by the drug also showed greater reductions in PANSS negative symptom scores (correlation of drug-placebo difference scores, r=-0.47, p=0.02). A similar trend was seen in the putamen (r=-0.37, p=0.06), but not in other drug-activated regions, suggesting the symptom-activation correlation was specific to striatum.

**Discussion:** The mGluR2 PAM was generally well-tolerated. In this pilot study the drug did not significantly improve cognitive performance, nor did it reduce clinical symptoms on average. However, the drug did increase fMRI activity in the anterior cingulate and basal ganglia during a working memory task, and the extent of drug-induced striatal activation correlated with reductions in negative

# Striatal Activation Induced by mGluR2 Positive Allosteric Modulation Correlates with Negative Symptom Reduction in Schizophrenia

Monday, Poster #52 (continued)

Daniel Wolf

symptom severity. These results encourage further investigation of this mgluR2 PAM and related agents, including studies focused on the potential role of striatal mechanisms impacting emotion and motivation. Our results also support the use of fMRI for sensitive detection of drug effects. Imaging biomarkers may reveal therapeutic mechanisms, and help tailor drug development and treatment towards specific patient populations and symptom domains.

PL

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## Prenatal Exposure to Maternal Infection Alters Neonatal Brain Structure

Monday, Poster #49

John H. Gilmore, Mark Connelly, Philip Nielsen, Sandra Woolson, Robert Hamer, Rebecca Knickmeyer, Sarah Short, Xiujuan Geng University of North Carolina at Chapel Hill

**Background:** Prenatal exposure to maternal infection is a risk factor for neuropsychiatric disorder. Studies in animal models suggest that prenatal exposure to infection causes significant alterations in structural brain development, though studies in humans are lacking.

**Methods:** Prospective, longitudinal follow-up study of a cohort of 445 infants, both singleton and twins, born to women assessed for infection during pregnancy by prospective interviews and medical records review. At 2 weeks after birth infants underwent 3T MRI scans. Global and cortical tissue volumes were determined

**Results:** Neonates exposed to maternal infection had a significant reduction in cortical gray matter (1.9%; p = 0.04) and non-significant reductions in intracranial volume (ICV; 1.5%; p = 0.11) and total gray matter (1.65%; p = 0.07) compared to infants with no exposure. Infants with first exposure to infection in the third trimester had significant reductions in ICV (3.5%; p = 0.02), total gray matter (4.1%; p = 0.004), unmyelinated white matter (3.6%, p = 0.03), as well as cortical gray (4.9%; p = 0.008) and cortical white matter volumes (3.5%; p = 0.03).

**Discussion:** Prenatal exposure in maternal infection results in cortical gray matter volume reductions, especially for first exposure to infection in the 3<sup>rd</sup> trimester. This study indicates that prenatal exposure to infection can significantly alter prenatal brain development in humans, providing a plausible mechanistic basis for the relationship between prenatal exposure to infection and increased risk for neuropsychiatric disorders.

### Application of Sequencing, Fatty Acid Profiling, and Metabolomics Investigations to Explore Pathogenesis and Treatment Strategy for Anorexia Nervosa

Wednesday, Poster #68

<u>Pei-an Betty Shih</u>, Jun Yang, Christophe Morisseau, Ashley Van Zeeland, Toni-Kim Clarke, Andrew W. Bergen, Pierre Magistretti, Katherine Ann Halmi, Wade Berrettini, Nicholas Schork, Walter H. Kaye, Bruce D. Hammock University of California, San Diego

**Background:** Individuals with Anorexia Nervosa (AN) restrict eating and become emaciated. They tend to have an aversion to foods rich in fat. We have identified a novel AN susceptibility gene, Epoxide Hydrolase 2 (*EPHX2*), through a series of complementary genetic study designs (GWAS, exon-based sequencing, single-locus association and replication studies) in 1205 AN and 1948 controls (p=0.0004 to 0.00000016)(*Molecular Psychiatry, 2013*). To assess the mechanisms by which *EPHX2* influences AN risk, here we applied a multi-disciplinary approach using lipidomics, metabolomics, and *ex vivo* techniques to evaluate the biological functions of *EPHX2*.

**Methods:** *EPHX2* codes for soluble epoxide hydrolase (sEH) which binds to specific epoxides and converts them to the corresponding diols; thereby it plays a major role in the metabolism of endogenous lipid epoxides, such as the epoxyeicosatrienoic acids (EETs), a derivative of arachidonic acid (ARA). We measured polyunsaturated fatty acids (PUFAs) and eicosanoids (bioactive lipid mediators that are derived from the metabolism of PUFAs) in 20 female AN cases and 17 age-, gender- and race-matched controls using the GC/MS and LC/MS/MS systems. EET-to-DHETs ratios were calculated as proxy markers of *in vivo* sEH activity, whereas *ex vivo* sEH activity was directly measured in 36 controls.

**Results:** Omega 6 PUFAs (DGLA, ARA, and Osbond acids) and omega 3 PUFAs (ALA, SDA, EPA, and DHA) were significantly elevated in ANs compared to controls (p=0.0003 to 0.00004). Controlling for effects of age and BMI, the 8.9.EpETrE of ARA and 8.9.EET-to-Diol ratio were significantly higher in ANs compared to controls (p<0.0001) whereas the eicosanoids markers of LA, another PUFA substrate of sEH, were not significantly different. The *ex vivo* sEH activity

# Application of Sequencing, Fatty Acid Profiling, and Metabolomics Investigations to Explore Pathogenesis and Treatment Strategy for Anorexia Nervosa

Wednesday, Poster #68 (continued)

Pei-an Betty Shih

measured in 13 controls showed marginal association with 8.9.EET-to- Diol ratio (p=0.07), suggesting 8.9.EpETrE of ARA may be a sensitive activity target of sEH. Variant allele carriers of an AN-associated *EPHX2* SNP in the 3'-UTR region showed significant association with 11,12- EET-to-Diol ratio (p=0.02) after controlling for the effects of age, BMI, and disease status, further providing evidence for *EPHX2* variation's influence on sEH activity and the subsequent effects on PUFA metabolism and eicosanoid activity.

**Discussion:** This study suggests that *EPHX2* influences AN risk through biological interaction with the PUFA pathway, specifically the ARA. It demonstrates that an application of multiple genetic designs interrogating common and rare variation is an effective approach to identify otherwise unsuspected risk genes in AN; that joint investigations of genetic mechanisms with their biological non-genetic partners (e.g.: diet, stress) may lead to improved understanding of pathophysiology, and new treatment strategies for AN.

#### **Retinoid-Related Orphan Receptor Alpha: A Novel Candidate Gene for Psychiatric Disease**

Monday, Poster #236

<u>Joseph I. Friedman</u>, Sander Markx, Terry Vrijenhoek, Ronald Kim, Joris A. Veltman, Arthur Mikhno, James R. Moeller, Mala Ananth, David K. Leung, Han G. Brunner, Vincent Giguere, Panayotis K. Thanos Icahn School of Medicine At Mount Sinai

**Background:** Several recent association studies have identified the retinoidrelated orphan receptor alpha (ROR $\alpha$ ) gene as a significant risk locus for posttraumatic stress disorder, bipolar disorder, and autism. Indeed, the naturally occurring *staggerer* ROR $\alpha$  mutant and genetically engineered ROR $\alpha$  null mice demonstrate brain changes including: cerebellar atrophy, Purkinje cell atrophy and loss, degeneration of granule cells, and limted data showing structural changes in the olfactory bulb, in association with severe ataxia and other cerebellar dysunction. This restricted pattern of changes to the cerebellum in the RORa mutant leaves major gaps in any model trying to explain RORa gene-related susceptibility to this diversity of psychiatric disease. Therefore, in order to bridge this translation gap we conducted an investigation of the neuropsychological and neuroimaging intermediate phenotypes in a multiply affected family with a nonfunctional duplication of the RORa gene, and in RORa-deficient mice models.

**Methods:** Clinical, neuropsychological and genetic data were collected from six members of a single pedigree with three members harboring a rare duplication of isoform 1 of the ROR $\alpha$  gene, presumably resulting in a frameshift leading to an early stop-codon and thus a non-functional protein. In addition, magnetic resonance imaging (MRI) was conducted on these same subjects, and fluorodeoxyglucose-positron emission tomography (FDG-PET) brain scans to quantify regional glucose metabolism were obtained from all family members and from 14 unrelated, normal age-matched controls (NC). FDG-PET data processing was carried out via voxel-based, canonical variates analysis and Scaled Subprofile Model of Principal Components analysis.

In a parallel experiment, laboratory generated homozygous (KO) (N=13) and heterozygous (HT) (N=12) ROR $\alpha$ -deficient mice were compared to wild type

## **Retinoid-Related Orphan Receptor Alpha: A Novel Candidate Gene for Psychiatric Disease**

Monday, Poster #236 (continued)

#### Joseph I. Friedman

(WT) (N=11) mice on performance on tests modeling cognitive deficits including novel object recognition and T-maze testing, and tests of locomotor activity and coordination including open field and rotarod tests. In addition, brain glucoe metabolism (BGluM) was assessed in these mice using [18F] FDG micro-PET. **Results:** The three family members harboring the ROR $\alpha$  duplication carried diagnoses of schizophrenia, schizoaffective disorder and major depression. Moreover, the three affected family members demonstrated significant neuropsychological dysfunction, whereas none of the three family members without the RORa duplication demonstrated any neuropsychological impairments. T1-weighted MRI images demonstrated all three individuals harboring the duplication to have peri-sylvian fissure and pre-pontine atrophy in addition to ventricular enlargement. None of the three family members without the duplication showed any anatomical abnormalities on MRI. FDG-PET data robustly differentiated the three family members with the RORa duplication from the family members without the RORa duplication and all NC subjects (p < 0.05). A unique pattern of white matter (WM) hyper-metabolism observed in the corpus callosum, internal capsule, in the vicinity of the medial prefrontal cortex, temporal cortex, and sensorimortor cortex and hypo-metabolism observed in the vicinity of the sensorimotor cortex, occipital cortex, and inferior parietal cortex was unique to the three family members affected with the RORa duplication compared to family members without the RORa duplication and all NC subjects. Neuropsychological test performance of the mice demonstrated KO mice had a significantly decreased ability to recognize novel objects compared to both WT and HT mice. Moreover, KO mice had a significantly lower percentage of correct trials on the T-maze compared to both WT and HT mice. Micro-PET data demonstrated significant hypo-activation in KO animals compared to WT animals in the rhinal cortex, cerebellum, paraflocculuc, thalamic nucleus, primary somatosensory cortex, lateral orbital tract, and piriform cortex.

#### **Retinoid-Related Orphan Receptor Alpha: A Novel Candidate Gene for Psychiatric Disease**

Monday, Poster #236 (continued)

#### Joseph I. Friedman

Contrastingly, KO animals showed significant hyper-activation compared to WT animals in the periaqueductal gray, colliculi, olfactory bulb, cerebellar nuclei, and striatum. Moreover, KO mice showed a significant negative correlation between activation at both the cerebellum (r = -0.839, p < .05) and the periaqueductal gray (r = -0.829, p < .05) and the percentage of correct trials during T Maze testing. **Discussion:** These data provide the first evidence that disruption of the ROR $\alpha$  gene has negative structural and functional brain consequences outside the cerebellum. Indeed, the unique patterns of both abnormal hypo- and hyperactivation associated with ROR $\alpha$  disruptions in the human and animal subjects, and its correlation with impaired spatial memory, suggests abnormalities in the synchrony of interconnected neural networks possibly contributing to the susceptibility to a diverse array of psychiatric disease. Further investigation is warranted.

## Automated Analysis of Disorganized Communication Predicts Transition to Psychosis Among Clinical High Risk Patients

Tuesday, Poster #4

Gillinder Bedi, Facundo Carillo, Guillermo Cecchi, Diego Fernandez Slezak, Mariano Sigman, Jordan E. DeVylder, Felix M. Muchomba, <u>Cheryl M.</u> <u>Corcoran</u> Columbia University Medical Center

**Background:** Subthreshold thought disorder has been identified as predictive of psychosis onset among patients at clinical high risk (CHR) for psychosis (Bearden et al., 2011). Assessment of thought disorder is achieved through clinical ratings of speech production. Analyzing speech with automated methods may present a direct, objective measure to complement existing methods, potentially offering a unique 'window into the mind'. We evaluated the trajectory of disorganized communication leading to psychosis using clinical rating scales in a large cohort of clinical high risk (CHR) patients. We also assessed whether a novel, automated method of speech analysis could differentiate those who went on to transition to psychosis from those who did not over a 2.5 year period.

**Methods:** 100 patients at CHR for psychosis were ascertained and followed quarterly for up to 2.5 years, or until time of dropout or transition to psychosis. Disorganized communication was assessed for predictive value for psychosis both at baseline and as a latent trajectory over time. A subcohort of 35 CHR patients had transcribed interviews, which were analyzed for semantic and syntactic coherence using a novel automated speech analysis approach. We employed machine learning with leave-one out cross validation to assess whether the semantic and syntactic indices identified predicted conversion to psychosis over the period of follow up. To further validate the method, we applied the classification algorithms developed to two separate cohorts of schizophrenia patients and healthy controls.

**Results:** Psychosis transition in the overall sample was 26%. Both baseline disorganized communication and a trajectory of high persistent disorganized communication were predictive of psychosis, with similar sensitivity and specificity ~0.6. Automated speech-based analyses of speech from a single time

#### Automated Analysis of Disorganized Communication Predicts Transition to Psychosis Among Clinical High Risk Patients

Tuesday, Poster #4 (continued)

Cheryl M. Corcoran

point predicted transition to psychosis with a sensitivity of 0.8 and specificity of 0.93. The algorithms developed also accurately discriminated between schizophrenia patients and healthy controls in two separate cohorts.

**Discussion:** Persistent disorganized communication predicts psychosis onset; importantly this feature of psychosis risk can be accurately identified using automated speech analyses. These findings are consistent with neural and electrophysiological studies of psychosis risk, and have important implications for treatment strategies and prognosis assessment in CHR individuals.

## **Dissecting Nucleus Accumbens Dynorphin Neurons in Aversion and Reward**

Wednesday, Poster #196

Ream Al-Hasani, Jordan G. McCall, Nicole Capik, Blessan Sebastian, Daniel Hong, Audra Foshage, Michael Krashes, Bradford Lowell, Thomas Kash, <u>Michael R. Bruchas</u> Washington University, St. Louis

**Background:** The adverse effects of stress are well documented, yet many of the underlying mechanisms remain unclear and controversial. The dynorphin/kappa opioid system is implicated in the mediation of stress and resultant vulnerability to drug abuse. It is thought that stress causes dynorphin release activating kappa-opioid receptors (KOR) within both dopaminergic and serotonergic nuclei as well as their striatal targets. Consequently, much attention has focused on these systems in the modulation of KOR-mediated responses. Despite our current knowledge of central dynorphinergic cell body populations, a clear description of the axonal projections of these neurons is unknown.

**Methods:** We crossed the Cre-dependent tdTomato (Ai9) reporter mouse to a mouse expressing Cre recombinase under the same promoter as dynorphin (Dyn-Cre) so only dynorphinergic cells express tdTomato. This allows complete visualization of dynorphinergic circuitry throughout the brain. We also virally targeted channelrhodopsin-2 to striatal dynorphinergic neurons and optogenetically activated neuronal populations in both the dorsal and ventral NAc shell to measure aversion and reward behaviors using place preference, aversion, and operant conditioning.

**Results:** Using our dynorphin-cre-tDtomato cross we show robust dynorphin expression in cell bodies throughout the brainstem and forebrain. Clear visualization of intact projections throughout the brain and dynorphinergic projections can be seen from and within the cortex, striatum, amygdala, and numerous monoaminergic nuclei. Dynorphinergic neurons within the striatum are particularly interesting for the study of stress and drug abuse. Prior studies have shown that KOR agonists inhibit dopamine and serotonin release in the nucleus accumbens (NAc), which regulates aversive behaviors. Therefore, we investigated

#### **Dissecting Nucleus Accumbens Dynorphin Neurons in Aversion** and Reward

Wednesday, Poster #196 (continued)

#### Michael R. Bruchas

whether specific modulation of dynorphinergic neuronal firing in the NAc is sufficient to induce aversive behaviors. This activation significantly increased c-Fos immunoreactivity in dynorphinergic neurons and inhibited electricallyevoked EPSCs which was reversed by norBNI application. Furthermore, activation of ventral NAc shell induced conditioned and real-time aversive behavior, while dorsal NAc shell stimulation resulted in a place preference which was also shown to be positively reinforcing in an operant task paradigm.

**Discussion:** The results presented here for the first time show a discrete subregion of dynorphin-containing cells in the ventral shell of the accumbens are required for aversion mediated by KOR activation. Furthermore, dorsal accumbens dynorphin cell activity is consistent with reward, perhaps via a classical dopamine D1-mechanism, but this requires further study. Understanding the mechanisms by which the dynorphin/kappa opioid system regulates negative affective behaviors will provide valuable insight into potential treatments for stress disorders and drug abuse.

## Mismatch Negativity Predicts Psychosis Onset and is Associated with Plasma Markers of Inflammation in Youth at Clinical High Risk for Psychosis

Wednesday, Poster #47

Daniel H. Mathalon, Diana Perkins, Kristin Cadenhead, Gregory A. Light, Peter Bachman, Jason Johannesen, Aysenil Belger, Margaret Niznikiewicz, Erica Duncan, Ricardo Carrion, Jean Addington, Tyrone Cannon, Barbara A. Cornblatt, Larry J. Seidman, Elaine Walker, Scott Woods University of California, San Francisco

Background: The mismatch negativity (MMN), an event-related brain potential (ERP) component elicited pre-attentively by deviant auditory stimuli imbedded in a stream of standard stimuli, has been repeatedly shown to be reduced in amplitude in schizophrenia. Prior studies from several groups have shown MMN to be reduced in patients at clinical high risk (CHR) for psychosis, particularly in those who subsequently develop a psychotic disorder, principally schizophrenia. MMN is thought to depend on neurotransmission at the N-methyl-d-aspartate (NMDA) sub-class of glutamate receptors based on human and non-human primate studies showing NMDA receptor antagonists to disrupt MMN. Recent interpretations of the MMN have emphasized its reflection of both short-term (seconds) and longer term (minutes to hours) synaptic plasticity in the service of auditory sensory/perceptual learning, since the amplitude of the MMN to a deviant stimulus increases as a function of the number of repetitions of the preceding standard stimulus and also as a function of the emergence of more enduring memory traces for complex sounds through repeated exposures to these sounds. A plethora of basic neuroscience data indicates that neuroinflammation and inflammatory cytokines compromise mechanisms of synaptic plasticity such as long-term potentiation. Moreover, prior studies have shown schizophrenia and other psychiatric disorders to be associated with elevations in peripheral markers of inflammation. Using data collected as part of the North American Prodromal Longitudinal Study (NAPLS), we examined whether MMN was reduced in CHR patients, particularly those who went on to convert to a psychotic disorder, and further, whether blood plasma markers of inflammation were associated with reductions in MMN amplitude in CHR patients.

# Mismatch Negativity Predicts Psychosis Onset and is Associated with Plasma Markers of Inflammation in Youth at Clinical High Risk for Psychosis

Wednesday, Poster #47 (continued)

#### Daniel H. Mathalon

**Methods:** As part of the ongoing multi-site NAPLS study, 212 CHR youth meeting criteria for a psychosis prodrome syndrome (SIPS interview) and 152 age-matched healthy controls (HC) underwent electroencephalographic (EEG) recording during a 3-deviant (pitch, duration, pitch+duration) MMN paradigm administered while subjects performed a visual distractor task. In addition, 27 CHR individuals who subsequently converted to psychosis were compared with 75 CHR individuals who had not converted by the 24-month follow-up assessment. MMN was measured at electrodes Fz and Cz, using linked mastoids as reference. Blood plasma samples were drawn from a sub-group of subjects including 32 CHR-converters, 40 CHR non-converters, and 35 healthy controls, and were subject to analysis on a Luminex® multiplex platform. Analytes of inflammatory markers that distinguished CHR converters from non-converters were identified (n=30) and aggregated (z-transformed) to form an "inflammatory threshold index". For each subject, this index was the calculated as the number of analytes that exceeded a z-threshold set to flag deviant analyte values.

**Results:** Results showed that CHR patients had reduced MMN amplitudes at baseline relative to healthy controls for duration deviant MMN (p<.05), but not for pitch or combination deviant MMNs (Group x Deviant Type interaction p=.011). Moreover, baseline MMN amplitude, irrespective of deviant type, was significantly reduced in the CHR converters relative to the CHR non-converters (Group main effect p=.024). As expected based on the method used to derive it, the inflammatory threshold index was significantly elevated in CHR converters relative to CHR non-converters (p<.00001). Of note, both the CHR converters (p<.00001) and the CHR non-converters (p<.005) showed significant elevations of the inflammatory index relative to healthy controls. Reduced pitch-deviant MMN amplitudes were significantly associated with elevated scores on the inflammatory threshold index in CHR patients (r=.38, p=.002, n=66), but not in healthy controls

# Mismatch Negativity Predicts Psychosis Onset and is Associated with Plasma Markers of Inflammation in Youth at Clinical High Risk for Psychosis

Wednesday, Poster #47 (continued)

Daniel H. Mathalon

(r=.11, p=.57, n=35). Furthermore, while this MMN-inflammation relationship was strongly evident in the CHR converters (r=.57, p=.007, n=21), it was not evident in the CHR non-converters (r=.16, p=.34, n=39).

**Discussion:** Results to date replicate previous findings showing MMN amplitude to be reduced in CHR patients, particularly those who go on to convert to a psychotic disorder. Accordingly, disruption of NMDA-dependent synaptic plasticity, as reflected by the MMN, appears to precede psychosis onset and predicts the likelihood of conversion to a psychotic illness. In addition, the identification of a number of plasma analyte markers of inflammation that are elevated in CHR converters suggests that an active inflammatory process precedes psychosis onset in CHR patients who develop psychosis. Moreover, consistent with literature showing neuroinflammation to compromise mechanisms of synaptic plasticity, baseline elevations in plasma markers of inflammation were significantly associated with MMN amplitude reductions in the subgroup of CHR patients who subsequently converted to a psychotic disorder.

### Age-related Sperm DNA Methylation Changes are Transmitted to Offspring and Associated with Abnormal Behavior and Dysregulated Gene Expression

Tuesday, Poster #43

<u>Maria H. Milekic</u>, Yurong Xin, Anne O'Donnell, Victoria Fatemeh. Haghighi, Jay A. Gingrich, John Edwards, Timothy Bestor Columbia University

**Background:** Accumulating evidence support that advanced paternal age (APA) poses an increased risk in children for psychiatric disorders such as schizophrenia (SZ), bipolar and autism spectrum disorders (ASD). There is clear evidence that *de novo* single nucleotide and copy number variations contribute to SZ and ASD, and that APA is associated with an increased rate of these types of mutations. Likewise, aging is associated with altered DNA methylation in both mammalian somatic and germ cells, and epigenetic abnormalities have been observed in the psychiatric disorders associated with APA. Accordingly, we hypothesized that DNA methylation abnormalities arising in the sperm of older fathers are inherited by the offspring and result in altered gene expression and behavior.

**Methods:** Old (12mo) and young (3mo) male 129SvEv mice were bred with two female (3mo) 129SvEv mice to generate old (OFO) and young (YFO) father offspring. The males were removed after 2 weeks to prevent direct contact with the offspring and the females were separated to control for maternal and litter effects. At 3mo the offspring were tested on a behavioral battery, including tasks such as open field, startle activity and prepulse inhibition. We determined DNA methylation using a whole genome sequencing approach called Methylation Mapping Analysis by Paired-end Sequencing (Methyl-MAPS) (Edwards et al. *Genomic Res.* 2010). Epididymal sperm from old and young male mice (n=4/group) was collected after the breeding. The brains of OFO and YFO were harvested at the end of behavioral testing. Methyl-MAPS libraries were prepared for both the fathers' sperm (n=4/group), as well as from one hemisphere from OFO and YFO (n=4/group). Mate-pair libraries were prepared for sequencing on the ABI SOLiD platform according to methods described by Edwards et al. (*Genomic Res.* 2010). Data was processed and analyzed as described by Xin et

### Age-related Sperm DNA Methylation Changes are Transmitted to Offspring and Associated with Abnormal Behavior and Dysregulated Gene Expression

Tuesday, Poster #43 (continued)

#### Maria H. Milekic

al. (*Epigenetics*, 2011). Transcriptome RNA-seq was performed on the remaining hemisphere from the same OFO and YFO samples used for Methyl-MAPS. Library construction and sequencing were performed by the Columbia Genome Center on an Illumina HiSeq2000. Off-Line Basecaller (OLB-1.9.4) was used for base calling and the pass filter reads were mapped to the mouse genome (NCBI37/ mm9) using Tophat. We estimated the relative abundance (aka expression level) of genes and splice isoforms using cufflinks with default settings.

**Results:** To determine whether aging alters sperm DNA methylation patterns, we performed genome-wide methylation profiling of epididymal sperm DNA pooled from young or old male mice using Methyl-MAPS. Mapping the methylation difference between the two groups across the regions up- and downstream of the transcription start site (TSS) and the first, internal and last exons of 20,496 RefSeq genes, we found that old mice had a significant loss of methylation at the regions flanking the TSS compared to young males. Comparing CpG island (CGI) and non-CGI promoters, the methylation difference was more profound at the regions up- and downstream of CGI promoter, so called CGI shores. Behavioral testing of the offspring of these old and young males revealed that OFO have significantly reduced exploratory, startle amplitude and prepulse inhibition compared to YFO. Performing Methyl-MAPS on brain DNA from OFO and YFO we found that, similar to the old fathers, OFO have significantly reduced DNA methylation at the regions flanking the TSS. This difference was specific to promoter CGI shores. Because DNA methylation patterns were altered in regions known to regulate transcription, we performed transcriptome RNA-seq on the remaining hemisphere of OFO and YFO. Differential gene expression analysis revealed a significant change in the expression of genes previously implicated in ASD and mental retardation (*En2 and CA8*), as well as genes regulating neural development (NeuroD1), synaptogenesis (Cbln1 and Cbln3) and cell signaling (Gabra6).

## Age-related Sperm DNA Methylation Changes are Transmitted to Offspring and Associated with Abnormal Behavior and Dysregulated Gene Expression

Tuesday, Poster #43 (continued)

#### Maria H. Milekic

**Discussion:** Similar to the epidemiological findings in humans, increased paternal age in mice is associated with behavioral alterations in the offspring. This seems to be mediated, in part, by germ line transmission of DNA methylation abnormalities arising in the sperm of older fathers. Our whole genome methylation experiments on sperm DNA from old and young mice, revealed that there is a loss of methylation at promoter CGI shores with aging and that this specific signature is also present in the OFO. These CGI shores have been shown to contain cell-, tissue- and species specific DNA methylation differences (Irizarry, Nat. Genet, 2009), which are associated with gene expression. RNA-seq on brains from OFO and YFO revealed significant changes in genes implicated in ASD, and known to regulated neural development and synaptic functions. These findings indicate novel pathways and mechanisms that may contribute to ASD and SZ and which may eventually lead to the development of new and more effective therapeutic interventions.

# Prospective Examination Of Prepulse Inhibition In OIF/OEF Marines Suggests Reduced Sensorimotor Gating Is A Preexisting Factor In Those That Develop PTSD After Combat Deployment

Wednesday, Poster #13

Victoria Risbrough, <u>Dean Acheson</u>, Dewleen G. Baker, Caroline Nievergelt, Kate Yurgil, Mark A. Geyer University of California San Diego

Background: Development of combat-related posttraumatic stress disorder (PTSD) is one of the major health concerns arising following the wars in Iraq and Afghanistan (Smith et al., 2008; Polusny et al., 2011). To develop more effective treatment and prevention efforts, a greater understanding of theneurobiological processes involved in the etiology and course of PTSD is needed (Baker et al., 2012). To understand PTSD etiology, it is critical to differentiate components of PTSD-related phenotypes that are pre-existing factors from those that emerge specifically after trauma. This distinction can only be addressed by prospective studies. Here we tested the hypothesis that sensorimotor gating is a pre-existing factor in development of PTSD. Prepulse Inhibition (PPI) is a cross-species operational measure of sensorimotor gating and putative measure of pre-attentional information processing (Geyer & Braff, 1987). Presentation of a neutral acoustic "prepulse" 30-300 ms before a more intense, startling stimulus reduces startle magnitude, possibly via direction of attentional resources toward the prepulse creating a "gate" for the subsequent startle stimulus (Swerdlow et al., 1999). PPI has a well defined neural circuit and is modulated by both subcortical and cortical circuits such as the prefrontal cortex. PPI has been found to be deficient in a number of psychiatric disorders (Swerdlow et al., 2006; Castellanos et al., 1996; Perry et al., 2001; Ahmari etal., 2012; Ludewig et al., 2002), however its role in PTSD is currently unclear (Kohl et al., 2013).

**Methods:** These data are collected as part of the Marine Resiliency Study (MRS), a prospective study of psychological and biological markers in a sample of Marines deployed to either Iraq or Afghanistan from 2008-2011. Marines completed the PPI test as well as a clinician administered PTSD symptom scale (CAPS) prior

# Prospective Examination Of Prepulse Inhibition In OIF/OEF Marines Suggests Reduced Sensorimotor Gating Is A Preexisting Factor In Those That Develop PTSD After Combat Deployment

Wednesday, Poster #13 (continued)

Dean Acheson

to deployment, 3 months post-deployment, and 6 months post-deployment. PPI was assessed as previously described (Acheson et al. 2012). In brief the session used 114 dB acoustic startle pulses, amd 86-dB prepulses (16 dB above the 70 dB background noise) that preceded the startle pulse by 30, 60 or 120 msec (i.e. interstimulus intervals).

**Results:** Of the 1229 Marines that did not have PTSD before deployment and had complete EMG and CAPS data at the 6-month time point, 46 (4%) developed PTSD after deployment (CAPS score >65). A linear mixed effects model found that Marines who tested positive for PTSD 6-months after return from combat deployment displayed significantly lower PPI performance across all pre- and post-deployment assessment periods relative to Marines who did not test positive at 6 months. There were no main effects of time point. PPI reductions in the PTSD group were greatest at 30 and 60 ms interstimulus intervals. No significant differences in either general startle magnitude or startle habituation emerged at any assessment period, although there was a trend for the PTSD group to show higher baseline startle at the pre-deployment time point. Effect of group remained when covarying for cohort, traumatic brain injury, and hearing loss. Further, post-deployment PPI performance did not correlate with Marine self-report of combat experience, suggesting that PPI performance is a stable trait unaffected by trauma experience itself.

**Discussion:** This study represents the first longitudinal test of PPI performance in relation to risk for later development of PTSD following combat experience. These results suggest that deficient PPI performance may represent a pre-existing risk factor for development of PTSD in response to traumatic experience. Ongoing studies are now in progress to determine if environmental or genetic perturbations mediate the role of PPI as a risk factor for PTSD.

## Disrupting AMPA Receptor Endocytosis Restores the Ability to Form New, and Enables the Recovery of Old, Memories in Mice Genetically Designed to Mimic Alzheimer's Disease

Tuesday, Poster #92

<u>Sheena Josselyn</u>, Adelaide Yiu, Valentina Mercaldo, Derya Sargin, Paul Frankland Hospital for Sick Children/University of Toronto

**Background:** The clinical hallmark of Alzheimer's disease (AD) is a progressive decline in cognitive function. The sequence of this decline often follows a stereotyped course; patients first show difficulty forming new memories, then deficits in retrieving older memories followed by deficits in other cognitive domains, and a loss of overall bodily functions. The ultimate outcome of AD is death. B-amyloid (A-beta) is widely implicated in the neuropathology underlying AD and chronically high levels of A-beta may induce cell death. This neurodegeneration readily accounts for the memory and cognitive impairment observed in the later stages of AD. In the early stages of AD, however, patients show deficits in forming new memories and high levels of A-beta but no detectable cell death. This suggests that high levels of A-beta may directly interfere with the synaptic plasticity required for normal memory formation. How A-beta impairs memory is unknown. In vitro, high levels of A-beta have been shown to decrease synaptic strength by promoting the internalization of postsynaptic AMPA-type glutamate receptors (AMPARs), suggesting the some of the memory deficits in AD may be due to excessive AMPAR internalization. Here we investigated the role of AMPAR internalization in the memory deficits observed in several types of mice genetically designed to recapitulate important aspects of AD.

**Methods:** We examined the effects of acutely or chronically increasing A-beta levels on the ability of mice to form stable long-term spatial and contextual fear memories. To transiently increase A-beta levels in wild-type (WT) mice we used replication-defective herpes simplex viral (HSV) vectors expressing human amyloid precursor protein (APP) containing both the Swedish and Indiana familial AD mutations. To chronically increase A-beta levels we used TgCRND8 mice, transgenic mice that chronically express the same mutated APP construct.

## Disrupting AMPA Receptor Endocytosis Restores the Ability to Form New, and Enables the Recovery of Old, Memories in Mice Genetically Designed to Mimic Alzheimer's Disease

Tuesday, Poster #92 (continued)

#### Sheena Josselyn

We used several methods to interfere with AMPAR internalization. First we used viral vectors to express a peptide designed to specifically interfere with GluA2-containing AMPAR endocytosis (GluA2-3Y peptide). Second, we use TAT (Trans-Activator of Transcription) proteins to systemically deliver this small interfering GluA2-3Y peptide. Importantly, the use of this non-toxic method of delivering this peptide via systemic administration may facilitate the translation of our results to the clinic. As a third method to disrupt GluA2-containing AMPAR endocytosis, we targeted Arf6 (ADP-ribosylation factor 6) expression, which is thought to be critical for this form of clathrin-mediated endocytosis.

**Results:** We observed that either acute or chronic increases in A-beta levels impaired the ability of mice to form stable long-term spatial or a contextual fear memory without inducing cell death. These memory consolidation deficits were accompanied by a decrease in the surface levels of GluA2-containing AMPAR, suggesting that the loss of GluA2-containing AMPAR may be responsible for the memory deficits. Consistent with this interpretation, we observed a strikingly similar phenotype when we used viral vectors to express a constitutively active from of Arf6 (Arf6-Q67L) to induce endocytosis of GluA2-containing AMPAR only in the hippocampus of WT mice. Moreover, the memory deficits induced by increasing A-beta levels were occluded by directly activating Arf6, suggesting that A-beta was acting through this pathway to produce the memory deficits. These results are consistent with the interpretation that high levels of A-beta produce memory deficits by facilitating GluA2-containing AMPAR endocytosis. To examine this more directly, we tested whether it was possible to reverse the memory deficits produced by high levels of A-beta by disrupting AMPAR trafficking. Importantly, transiently interfering with AMPAR internalization (by either a specific interfering peptide or interfering with Arf6 function) was sufficient to reverse the memory deficits produced by either acute or chronic overexpression

# Disrupting AMPA Receptor Endocytosis Restores the Ability to Form New, and Enables the Recovery of Old, Memories in Mice Genetically Designed to Mimic Alzheimer's Disease

Tuesday, Poster #92 (continued)

#### Sheena Josselyn

of A-beta. Together, these data are consistent with the interpretation that high levels of A-beta impair memory by inducing the loss of surface GluA2-containing AMPAR.

It is now well-appreciated that memories, even after consolidation, are modifiable. The process of remembering is thought to reactivate memory representations in the brain. The reactivated memory is re-stored in a second wave of consolidation referred to as reconsolidation. Strikingly, similarly disrupting AMPAR endocytosis during a memory reminder enabled the recovery of an otherwise inaccessible memory in mice with chronically high A-beta levels. This result suggests that memory reactivation and subsequent reconsolidation may open a "window of plasticity" in which otherwise "lost" memories may be successfully reconsolidated (and consequently "recovered") by disrupting AMPAR internalization at the time of the reminder. Our findings raise the possibility that targeting AMPAR trafficking could restore both the ability to form new memories as well as enable recovery of lost past memories in AD patients.

**Discussion:** The results from these studies may not only lead to a better understanding of how A-beta disrupts memory but may help identify a novel therapeutic strategy to allow AD patients to form new memories as well as recover "lost" memories.

# Selective Effects of the 5-HT2C Receptor Agonist Metachlorophenylpiperazine (mCPP) on Intake of a Palatable Snack Food in Healthy Female Volunteers: Correlation with Regional Brain Activations Measured by BOPLD fMRI

Wednesday, Poster #107

<u>Colin T. Dourish</u>, Jason M. Thomas, Suzanne Higgs P1vital

**Background:** The 5-HT<sub>2C</sub> receptoragonist meta-chlorophenylpiperazine (mCPP) has been reported to decrease food intake in lean and obese volunteers although the behavioural selectivity of these effects and the brain mechanisms involved are unclear. In a recent study using a universal eating monitor to measure meal microstructure we showed that mCPP caused a dose related decrease in appetite ratings and enhanced the satiation quotient (change in hunger ratings divided by caloric intake) in lean females. However, despite these changes in rating derived measures the effects of mCPP on total food intake were not statistically significant. Therefore, in the present study, we investigated for the first time the potential influence of palatability on the response to mCPP by comparing the effects of the drug on the consumption of a pasta meal and a palatable snack in lean females. In addition, to investigate the brain mechanisms involved we used functional magnetic resonance imaging (fMRI) to determine the effects of a dose of mCPP that decreases appetite on blood oxygen level dependent (BOLD) signals.

**Methods:** The study used a within-subject double blind placebo controlled design and 24 healthy female volunteers received placebo and 30mg mCPP in a counterbalanced order one week apart. Participants were scanned using BOLD fMRI pre and post oral dosing and after the second scan were provided with a pasta meal and allowed to eat to satiety. Food intake and meal microstructure were recorded using a Sussex Ingestion Pattern Monitor (SIPM). The SIPM comprises a concealed weighing system and computer software to enable detailed collection and analysis of human eating behaviour and continuously monitors food intake in parallel with measures of appetite and satiety. When participants had finished their pasta meal they were offered a palatable snack (chocolate chip cookies) and

# Selective Effects of the 5-HT2C Receptor Agonist Metachlorophenylpiperazine (mCPP) on Intake of a Palatable Snack Food in Healthy Female Volunteers: Correlation with Regional Brain Activations Measured by BOPLD fMRI

Wednesday, Poster #107 (continued)

Colin T. Dourish

were again allowed to eat to satiety. Cookie intake and snack microstructure were recorded using the SIPM.

**Results:** mCPP significantly reduced hunger and the desire to eat but did not reduce the amount of pasta consumed during the meal. However, the drug significantly reduced pasta eating rate and increased pause length between mouthfuls of pasta. In contrast, mCPP significantly reduced the amount of cookies consumed. In addition, the drug reduced the size of cookie mouthfuls, the total number of cookie portions eaten and the cookie eating rate and increased the pause duration between mouthfuls of cookies. mCPP also decreased pleasantness ratings of both the pasta meal and the cookie snack but the temporal patterns of these responses were significantly different. Thus, the effect of mCPP on pleasantness ratings of pasta had a slow onset and increased throughout the meal whereas the effect of the drug on pleasantness ratings of cookies was immediately apparent and was maintained at the same level throughout the snack intake. Analysis of the BOLD fMRI results showed that mCPP attenuated activity in the hypothalamus, insula, brainstem, anterior cingulate cortex and dorsolateral prefrontal cortex. In addition, correlational analyses revealed that mCPP-induced BOLD changes in the hippocampus, anterior cingulate cortex, midbrain and orbitofrontal cortex were significantly and negatively correlated with cookie eating rate. These correlations were not apparent with pasta eating rate.

**Discussion:** These results show for the first time that mCPP decreases the consumption of a highly palatable snack food in humans. Furthermore, this was a selective effect as the drug had no effect on the consumption of a pasta meal suggesting that the effects of mCPP on eating may be determined by the hedonic properties of food. This interpretation is consistent with the contrasting time courses of the effects of mCPP on pleasantness ratings of the pasta and cookies

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# Selective Effects of the 5-HT2C Receptor Agonist Metachlorophenylpiperazine (mCPP) on Intake of a Palatable Snack Food in Healthy Female Volunteers: Correlation with Regional Brain Activations Measured by BOPLD fMRI

Wednesday, Poster #107 (continued)

Colin T. Dourish

during the meal. The fMRI results show that mCPP attenuated BOLD signals in key areas involved in the processing of appetitive, rewarding and motivational stimuli. Furthermore, changes in hippocampus, anterior cingulate cortex, midbrain and orbitofrontal cortex were negatively correlated with cookie eating rate but not pasta eating rate suggesting that these brain regions which are known to be involved in reward and memory processing could mediate the selective hedonic effects observed. Finally, as a selective 5-HT<sub>2C</sub> receptor agonist has recently been approved by the FDA for the treatment of obesity these findings could have important implications for drug therapy in obese patients where the over consumption of highly palatable foods may be an important contributory factor to the development and maintenance of the disease.

# Locus Specific Epigenetic Reprogramming: Bidirectional Regulation of the FosB Gene Using Synthetic Transcription Factors In Vivo

Tuesday, Poster #81

<u>Elizabeth Heller</u>, Hannah Cates, Haosheng Sun, Catherine Pena, Deveroux Ferguson, Scott Knight, H. Steve Zhang, Eric Nestler Icahn School of Medicine at Mount Sinai, Department of Neuroscience

**Background:** Transcriptional regulation underlies sensitivity to psychostimulant exposure and is associated with altered expression of several chromatin-modifying enzymes in key brain reward regions. Genome-wide assessments of histone posttranslational modifications (HPTMs) have identified drug and stress regulation at numerous target genes implicated in the associated behavioral abnormalities. However, it has not previously been possible to manipulate the epigenome in order to causally link the chromatin state of a single locus with behavioral and molecular responses to psychostimulant exposure. Engineered transcription factors, such as zinc finger proteins (ZFPs) and transcription activator-like effectors (TALEs), can direct enzymatic moieties to specific genomic loci. We are interested in epigenetic regulation of the immediate early gene, FosB, which is both necessary and sufficient for many of the downstream molecular changes mediating stress and reward response. Previous work has demonstrated that chronic cocaine exposure in both humans and rodents leads to a reduction in the levels of the histone methyltransferase, G9a, and the repressive epigenetic mark, histone H3 lysine 9 dimethylation (H3K9me2), at the FosB locus. This may be the mechanism by which FosB and its stable splice variant, DFosB, accumulate in reward regions of the brain in order to mediate drug responsiveness. Furthermore, DFosB expression is reduced in the brain reward regions of chronically stressed rodents and depressed human patients; we have recently found an increase in H3K9me2 at the *FosB* promoter in depressed human Nucleus accumbens (NAc) relative to healthy controls. Despite the robustness of these correlations, it has not yet been possibly to directly link a particular HPTM at the FosB gene to behavioral response, due to the use of experimental paradigms that affect the entire genome (e.g. overexpression of G9a, HPTM ChIP-chip, cocaine or stress

## Locus Specific Epigenetic Reprogramming: Bidirectional Regulation of the FosB Gene Using Synthetic Transcription Factors In Vivo

Tuesday, Poster #81 (continued)

Elizabeth Heller

exposure). Thus, we have pioneered the use of engineered transcription factors to direct a single epigenetic modification to a single gene of interest, within a single brain region.

Methods: A suite of 65 6-finger cys2/his2 ZFPs were designed in silico to recognize 18bp motifs within -200 to +1000 bp of the FosB promoter relative to the transcription start site. These ZFPs were tethered to the transcriptional activation domain, p65, and screened in vitro for activation of FosB mRNA expression by qRT-PCR. From this screen several ZFPs were selected for fusion to alternative enzymatic domains, such as the preSET/SET domains of G9a and the viral activation domain, VP64. In addition, three TALE-VP64 constructs were designed to target similar sequences. To confirm specificity of the binding of the ZFP across the genome, ZFP-NFD (no functional domain) constructs were fused to 3xFLAG affinity tag, expressed in vitro and subject to chromatin immunopurification (ChIP)-Sequencing. For in vivo analysis, mouse NAc neurons were infected with herpes simplex virus (HSV) expressing each of the constructs, and qRT-PCR and immunohistochemistry was used to determine activation or repression of *FosB* expression. We relied on qChIP using a variety of anti-HPTM antibodies to analyze the chromatin modifications induced by the ZFP-G9a and ZFP-p65 constructs. To determine the role of epigenetic remodeling on behavioral responses to psychotimulant exposure, mice were subject to either cocaine locomotor sensitization or acute social stress following HSV NAc infection with the ZFP constructs.

**Results:** We have found that FosB-ZFP-p65 and -TALE-VP64 constructs efficiently and robustly activate FosB/DFosB expression in NAc neurons, while FosB-ZFP-G9a represses expression. In addition, immunohistochemistry with an anti-FosB/DFosB antibody demonstrates that FosB-ZFP-G9a blocks cocaine induction of FosB/DFosB in NAc, while basal levels of protein are unchanged.

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# Locus Specific Epigenetic Reprogramming: Bidirectional Regulation of the FosB Gene Using Synthetic Transcription Factors In Vivo

Tuesday, Poster #81 (continued)

#### Elizabeth Heller

Using qChIP, we have found that the mechanism of this repression is HSV-G9a deposition of H3K9me2 specifically at the FosB gene in vivo, while FosB-ZFP-p65 activates FosB via H3K9/14 acetylation. In addition, this gene-specific epigenetic remodeling is associated with changes in *FosB* promoter binding by additional HPTMs and the transcription factor, pCREB, which is a known regulator of activity dependent activation of FosB. Engineered transcription factors are also able to modulate behavior, as FosB-ZFP-p65 expression in the NAc enhances cocaine locomotor sensitization, while FosB-ZFP-G9a expression blocks the cocaine effect on locomotor as well as sensitizes animals to stress. Discussion: Engineered transcription factors are effective tools to probe the behavioral and molecular consequences of chromatin remodeling at a single locus in vivo. Using this approach, we have identified a direct molecular mechanism for cocaine-mediated activation of the FosB gene, and have efficiently manipulated behavioral responses to drugs of abuse and stress. This work opens the field for a more mechanistic and causal analysis of the role of epigenetics in regulating the neurobiological mechanisms that underlie reward pathology.

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#### Suicidal Ideation in Depressed New Mothers: Relationship with Childhood Trauma and Sleep Disturbance

Wednesday, Poster #223

<u>Dorothy Sit</u>, James Luther, Jesse Dills, Heather Eng, Dan Buysse, Michele Okun, Stephen Wisniewski, Katherine L. Wisner University of Pittsburgh, School of Medicine, Department of Psychiatry

**Background:** Of new mothers with a positive depression screening (Edinburgh Postnatal Depression Scale - EPDS $\geq$ 10) 19.3 percent (n=1396) had thoughts of self-harm (Wisner et al, JAMA Psychiatry 2013). The high rate of suicidal ideation (SI) in new mothers is a major concern. Without proper treatment, mothers with SI are at increased risk for suicide. From the 1997-1999 UK Confidential Enquiries into Maternal Death suicide was the leading cause of maternal death from 42 days to 1 year postpartum; 28 percent of maternal deaths (n=242) resulted from suicide (Oates 2003). In this cross-sectional study, the objective was to characterize potential risk factors for postpartum women with SI who were enrolled in the primary study to screen for postpartum depression (PPD)(Wisner et al, 2013). The aims were to examine the relationship between SI and childhood or adult history of trauma (physical abuse or sexual abuse) and sleep disturbances in new mothers within 4-6 weeks after delivery. The hypothesis was childhood trauma and current sleep disturbance were associated with SI.

**Methods:** Eligible subjects included new mothers who received an EPDS depression screen within 4-6 weeks after delivery and a completed home visit evaluation. The primary psychiatric diagnosis was confirmed by the Structured Clinical Interview for DSM-IV (SCID). Patients with Bipolar Disorders, primary psychotic disorders, alcohol or substance use disorders were excluded. We compared the groups with an EPDS item 10 ("The thought of harming myself has occurred to me") = 0 ("never"), 1 ("hardly ever"), 2 or 3 ("sometimes" or "quite often"). We recorded baseline demographic characteristics including age, race, educational level, marital status, health insurance and parity. We examined comorbid disorders, adult and childhood abuse history, onset of the current depressive episode, total depression scores on the Structured Interview Guide for

## Suicidal Ideation in Depressed New Mothers: Relationship with Childhood Trauma and Sleep Disturbance

Wednesday, Poster #223 (continued)

#### Dorothy Sit

the Hamilton Depression Rating Scale, Atypical Depression Symptoms Version (SIGH-ADS) - a 29-item instrument that incorporates the Hamilton Rating Scale for Depression and a set of questions to assess atypical neuro-vegetative symptoms and the global assessment of function. For sleep disturbance we examined the SIGH-ADS sleep symptoms (insomnia – items H6, H7, H8 and wake time after sleep onset-WASO greater than 20 minutes).

**Results:** Of 648 eligible mothers, 496 (77%) reported that they "never" had thoughts of self-harm; 98 (15%) had SI "hardly ever", and 34 (5%) had SI "sometimes or quite often". Younger mothers, African Americans and mothers with public health insurance were significantly more likely to have SI. Mothers with the onset of depressive episodes before pregnancy or in the post-partum, a history of physical abuse in childhood, and lowered global functioning were significantly more likely to have SI. Cumulative logistic regression models suggested a main effect for history of childhood physical abuse (odds ratio-OR=1.681, 95% confidence interval-CI=1.045, 2.704, p=0.032) and a marginally significant interaction of childhood physical abuse with WASO > 20 minutes (OR=1.576, 95%CI 0.950, 2.614, p=0.078).

**Discussion:** A high percentage of maternal deaths are from suicide (Oates, 2003). Our data suggested that among mothers with a positive depression screening, having a past history of childhood trauma and possibly having recent sleep disturbance contributed to increased risk for thoughts of suicide. Altered stress responses in patients with early life abuse could increase their susceptibility to suicide (Lupien et al 2009). Patients with childhood abuse who completed suicide expressed reduced levels of glucocorticoid receptor-GR whereas non-abused patients who completed suicide did not express altered GR (McGowan,Meaney et al 2009). New research is imperative to extend our knowledge of the relationship between abnormal responses to stress and the cognitive processes involved with making decisions and experience of reward (Dombrovski et al, 2013) which could underlie suicidal thoughts and precede suicide in postpartum mothers.
### The Contribution of Adult Hippocampal Neurogenesis to Fear Memory Generalization

Tuesday, Poster #25

<u>Mazen A. Kheirbek</u>, Liam J. Drew, Elizabeth Balough, Christine A. Denny, Rene Hen New York State Psychiatric Institute/RFMH

**Background:** Maladaptive fearfulness is a hallmark of a number of anxiety disorders. In particular, in disorders such as post-traumatic stress disorder, it is frequently observed that fear becomes expressed in safe situations that are similar to the original trauma. That is to say, fear is overgeneralized to neutral situations. The dentate gyrus (DG) subregion of the hippocampus functions in pattern separation, a process by which representations of similar experiences or events are transformed into non-overlapping representations so as to facilitate their storage as discrete units. We hypothesize that impairments in pattern separation contribute to the overgeneralization of fear seen in certain anxiety disorders. Recently, adult-born granule cells (GCs) in the DG have been implicated in pattern separation. However, it remains unclear how these young neurons facilitate this process, or whether their functional contribution differs from that of mature GCs. Here, we have probed the mechanism by which immature GCs of the DG may act to prevent the generalization of a contextual fear memory. To address the online role of immature GCs in contextual encoding, generalization and anxiety, we used optogenetic techniques to selectively and bidirectionally modulate the activity of immature and mature GCs in a region-specific manner.

**Methods:** To target opsins selectively to immature GCs, a Nestin-CreER<sup>T2</sup> line was crossed to either a conditional archaerhodopsin-3 (Arch) or a channelrhodopsin-2 (ChR2) line. Tamoxifen injection in adult mice induced recombination in neural stem cells and transit-amplifying progenitors to generate opsin-expressing adultborn GCs. For targeting mature GCs, we crossed the conditional opsin lines with an Arc-CreER<sup>T2</sup> line, which directs recombination to cells expressing the immediate early gene Arc. Injection of Tamoxifen paired with exploration of a novel environment induced recombination and expression of opsins in a sparse population of mature GCs (but not immature GCs that don't express significant levels of Arc). Six weeks after TMX, mice were tested for behavioral effects

## The Contribution of Adult Hippocampal Neurogenesis to Fear Memory Generalization

Tuesday, Poster #25 (continued)

#### Mazen A. Kheirbek

of light-induced inhibition or excitation in the dorsal or ventral DG. As this manipulation allows for epoch selective modulation of activity in adult-generated neurons and their mature counterparts, we tested whether these cells play a context-specific role in a pattern separation task that requires mice to discriminate between a shock-paired context and a similar, safe context. Activity in adult-born GCs or a similar number of mature GCs in the dorsal DG was suppressed during exposure to either the conditioning context or the similar, safe context.

**Results:** Histological analysis showed that Nestin-ChR2 and Nestin-Arch lines expressed opsins in nearly all immature adult-born, GCs, that Arc-ChR2 and Arc-Arch lines expressed opsins almost exclusively in mature GCs and that similar numbers of opsin-expressing neurons were generated in the Nestin-opsin and the Arc-opsin lines. Optical modulation *in vivo*, demonstrated that while none of the manipulations impacted baseline anxiety state, excitation of either cohort of cells impaired context acquisition. Optical inhibition of adult-generated GCs, but not of an equal number of mature GCs in the dorsal DG disrupted the rapid encoding of contextual fear memories, indicating a selective contribution of adult-generated neurons to rapid encoding of contexts. In a contextual fear discrimination experiment, we found that inhibition of young GCs during exposure to the similar, safe context, but not the conditioning context, impaired discrimination. Surprisingly, inhibition of a population of mature GCs in the similar context improved the animals' ability to discriminate between contexts.

**Discussion:** This study reveals differential contributions of mature and immature neurons of the DG to contextual fear encoding and discrimination. Specifically, we show that while young GCs are not needed for the maintenance of an already learned contextual memory, they are necessary for the disambiguation of similar information from already learned information. In contrast, inhibiting the activity of mature neurons improves discrimination, indicating opposite functions for these two populations of cells within the DG. Immature neurons in the dorsal

# The Contribution of Adult Hippocampal Neurogenesis to Fear Memory Generalization

Tuesday, Poster #25 (continued)

Mazen A. Kheirbek

DG are required for the rapid encoding of novel contextual information and to disambiguate novel representations from already learned ones, which is consistent with their proposed role in pattern separation. In contrast mature GCs appear to be involved in generalization. We hypothesize therefore that strategies aimed at stimulating neurogenesis or modulating the activity of mature GCs may restrain overgeneralization and may be effective for the treatment of anxiety disorders.

#### **Intranasal Ketamine in Treatment-Resistant Depression**

Tuesday, Poster #139

<u>Kyle A. Lapidus</u>, Cara F. Levitch, Laili Soleimani, Andrew M. Perez, Jess W. Brallier, Michael K. Parides, Dan V. Iosifescu, Dennis S. Charney, James W. Murrough Mount Sinai Medical Center

**Background:** Current treatments for depression are only partially effective and exhibit delays in onset of therapeutic efficacy. Several studies have reported a rapid onset of antidepressant action for intravenous (IV) ketamine – and NMDA receptor antagonist – in patients with treatment resistant depression (TRD). Despite potential efficacy, the requirement for IV administration imposes potential limitations to therapeutic delivery. In contrast, ketamine delivery via an intranasal (IN) route may provide a more feasible treatment approach. Herein we report the first placebo-controlled study of IN ketamine in TRD.

Methods: Twenty subjects with TRD in a current major depressive episode were randomized to receive IN ketamine hydrochloride (50 mg) or 0.9% saline solution, in a crossover design with one of two treatment orders: either ketamine-placebo (KET-PBO) or placebo-ketamine (PBO-KET); KET or PBO were administered 1-2 weeks apart. 18 subjects received both treatments under randomized, double blind conditions. Before administration, subjects were admitted to a clinical research unit and study drug was administed by an anesthesiologist. Continuous vital signs monitoring was employed during and for 4 hours following treatment. The primary efficacy outcome measure was change from baseline in the Montgomery-Asberg Depression Rating Scale (MADRS) score at 24 hours following KET, compared to PBO. Response and remission rates at 24 hours represented secondary outcomes. Clinical response was defined as MADRS decrease  $\geq$  50% from baseline and remission was defined as a MADRS score of  $\leq 9$ . To assess hemodynamic side effects, vital signs were monitored and clinically significant changes were defined as systolic or diastolic blood pressure (BP) >180/100 or >20% increase above baseline level or tachycardia with heart rate >110 beats/minute. Management by medication interventions and treatment discontinuation was to be provided for significant hemodynamic changes. Other secondary outcomes included general adverse events, acute psychotomimetic

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#### **Intranasal Ketamine in Treatment-Resistant Depression**

Tuesday, Poster #139 (continued)

#### Kyle A. Lapidus

effects, and dissociative effects, measured with the Systematic Assessment for Treatment Emergent Effects (SAFTEE), Brief Psychiatric Rating Scale (BPRS), and Clinician-Administered Dissociative States Scale (CADSS) respectively.

**Results:** Subjects evidenced significant improvement in depressive symptoms within 24 hours after ketamine compared to placebo (t=4.4, p<0.001). Following KET, 8 of 19 subjects responded (42%), compared to 2 of 19 (11%) following PBO. Among study completers (those who received both treatment conditions), 8 (44%) met the response criterion. Of these 8, 1 (6%) also responded to placebo, while no completer met response criteria only for placebo (p=0.0325 based on McNemar's test). Intranasal ketamine was well tolerated with few side effects. Specifically, following ketamine, the mean increase in BPRS was 0.3, and in CADSS was 1.37. No patient exhibited clinically significant changes in hemodynamics.

**Discussion:** In this study, we demonstrated a rapid antidepressant effect of IN ketamine in patients with TRD. IN KET was well tolerated with almost no psychotmimetic or dissociative side effects and with no significant hemodynamic effects including blood pressure and heart rate changes. No interventions by anesthesiology were needed. Future studies are indicated to identify strategies for maintaining antidepressant response in patients who respond to intranasal ketamine.

# Specific Elevation of βCaMKII in the Lateral Habenula Lead to Core Symptoms of Depression

Monday, Poster #13

Hailan Hu, <u>Fritz Henn</u>, Kun Li, Tai Zhou, Zhongfei Yang, Lujian Liao, Catherine Wong, Roberto Malinow, John R. Yates III Mount Sinai School of Medicine

**Background:** The congenital lines of leartned helpless rats (cLH) have been useful in identifying the anatomical circuit which appears to mediate depression in humans as well as helplessness in animals, and these studies point to a critical role of the lateral habenula. The cLH lines show increased activation of the l. habenula and this has been seen in patients undergoing tryptophan depletion with return of depressive symprotoms. Responders to both ketamine and scopolamine have been shown to have decreased habenular activity.

**Methods:** The current study was a proteomic investigation of protein differences in disected l. habenula tissue between cLH animals and control animals animals using <sup>15</sup>N enrichment for mass spectometry to look for protein differences. A mouse model was created which could express the increase in  $\beta$ CaMKII found was made and tested for depressive features Down regulation or inhibition of  $\beta$ CaMKII was used to determine if this reversed the depressive phenotype.

**Results:** The proteomic screen revealed an almost doubling in  $\beta$ CaMKII in the helpless animals which was restricted to the l. habenula, the levels in the hippocampus were actually decreased by about 40% reflecting the loss of neuronal tissue in the model. Histological examination of the habenula showed uniform increase in neuronal levels of the enzyme in the lateral habenula and no increase in the medial habenula. This was verified using western blots. Overexpression of the protein in mouse models resulted in a doubling of the immobility time in the forced swim test and resulted in increased synaptic activity in the lateral habenula restricted to neurons showing overexpression of the protein. A knockdown of the  $\beta$ CaMKII resuced the depressive phenotype and reduced synaptic activity. Increasing  $\alpha$ CaMKII in mouse models had no effect on synaptic efficacy or behavioral measures of depression. Blocking the target of  $\beta$ CaMKII, GluR1 reversed the depressive sympotomolgy.

# Specific Elevation of βCaMKII in the Lateral Habenula Lead to Core Symptoms of Depression

Monday, Poster #13 (continued)

Fritz Henn

**Discussion:** These data suggest that the central role of the l. habenula in depression suggested by both animal studies and human imaging studies is due to a specific change in protein levels of  $\beta$ CaMKII, which increases synaptic firing leadingndirectly to the downstream regulation of the 5HT and DA systems. This data provides a toally new and clearly defined target for antidepressant development.

|       | ACNP 52nd Annual Meeting | Final Program |
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| Netes |                          |               |
| NOTES |                          |               |
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8:00 a.m. – 11:30 a.m. President's Plenary Grand Ballroom

#### **President's Plenary**

Welcoming Remarks and Moment of Silence David Lewis President

Presentation of Honorific Awards John Krystal Chair, Honorific Awards Committee

# Neural Circuitry Structure and Plasticity: Substrate for Brain Disorders and Novel Therapeutics

| 8:30 a.m.  | Homeostatic Plasticity: Keeping Your Brain in Balance<br>Gina Turrigiano  |
|------------|---|
| 9:15 a.m.  | Genetic Dissection of Cortical Circuit Organization and<br>Assembly: Chandeliers Light Up the Path<br>Z. Josh Huang                   |
| 10:00 a.m. | Surprise at the Synapse: MHC Class I, Pruning and Plasticity<br>Carla Shatz   |
| 10:45 a.m. | Dynamic Modulation of Dorsolateral Prefrontal Cortex<br>Microcircuits: Focus of Vulnerability in Mental Disease<br><i>Amy Arnsten</i> |

#### Homeostatic Plasticity: Keeping Your Brain in Balance

<u>Gina Turrigiano</u> Brandeis University

The positive-feedback nature of Hebbian synaptic plasticity can destabilize the properties of neuronal networks. Recent work from my lab and others has suggested that this destabilizing influence is counteracted by homeostatic plasticity mechanisms that stabilize neuronal activity. One such mechanism, homeostatic synaptic scaling, is a form of synaptic plasticity that adjusts the strength of all of a neuron's excitatory synapses up or down to stabilize firing. Here I will discuss our recent work showing synaptic scaling is a cell-autonomous process in which neurons detect changes in their own firing rates through a set of calcium-dependent sensors that then regulate receptor trafficking to increase or decrease the accumulation of glutamate receptors at synaptic sites. I will discuss the signaling pathways that underlie this process, the biophysical changes at synapses that allow synaptic strength to be scaled up or down, and the role this plasticity plays in keeping neocortical activity stable.

Gina Turrigiano received her BA from Reed College in 1984 and her PhD from University of California San Diego in 1990. She then trained as a postdoc with Eve Marder at Brandeis University before joining the faculty in 1994. She is now a full professor in the Dept. of Biology, the Volen Center for Complex Systems, and the Center for Behavioral Genomics at Brandeis. She has received numerous awards for her research including an NIH career development award, a Sloan Foundation fellowship, a MacArthur foundation fellowship, McKnight Foundation Technological Innovation and Neurobiology of Disease awards, an NIH director's pioneer award, the HFSP Nakasone Award, election to the American Academy of Arts and Sciences (2012) and election to the National Academy of Sciences (2013). Her scientific interests include mechanisms of synaptic and intrinsic plasticity and the experience-dependent refinement of neocortical microcircuitry.

### Genetic Dissection of Cortical Circuit Organization and Assembly: Chandeliers Light Up the Path

Z. Josh Huang

Cold Spring Harbor Laboratory

Despite their immense complexity and sophisticated operations that underlie mental functioning, the fundamental plan for the cellular organization of cerebral cortex is encoded in the genome, which directs cascades of developmental programs in each fetus. Therefore, genetic approaches that engage intrinsic biological mechanisms have the potential to penetrate cortical complexity and achieve appropriate cellular and molecular specificity, and analyses of circuits assembly will facilitate deciphering their functional organization. I suggest that, similar to "genetic screens" that so powerfully identified genes and principles underlying embryonic patterning, systematic genetic targeting of cell types and their progenitors, basic units of circuit organization and construction, will not only provide experimental entry points but also establish a paradigm that coherently link molecular, developmental, and systems studies of cortical circuits. I will summarize our progress on the genetic targeting of GABAergic interneurons and glutamatergic projection neuron, an effort that has facilitated reliable identification and manipulation of cell types and enabled comprehensive analysis from cell specification, connectivity, to their functional role in networks. The chandelier cell (ChC) is arguably the most distinctive class of interneurons that may shape cortical ensembles by exerting decisive control over pyramidal cell firing. I will present results on the stringent genetic mechanisms that specify ChC identity and their laminar deployment, evidence of a massive postnatal pruning process that likely shapes their circuit integration, and on-going studies on their local and long-range synaptic connectivity. With increasing experimental access to cellular build blocks of the cortex, we are poised to explore how stereotyped circuit motifs are assembled and organized, how they are molded by neural activity, and how their developmental trajectory might be altered in models of mental disorders.

Dr. Z. Josh Huang is currently Charles and Marie Robertson Professor of Neuroscience at the Cold Spring Harbor Laboratory in Long Island, New York. He received his PhD in Cell and Molecular Biology at Brandeis University

#### Genetic Dissection of Cortical Circuit Organization and Assembly: Chandeliers Light Up the Path

Z. Josh Huang (continued)

and his postdoctoral training at Massachusetts Institute of Technology. His long term research goal aims to understand the basic mechanisms underlying the developmental assembly and function organization of neural circuits in the cerebral cortex. His laboratory has pioneered the use of mouse engineering toward a genetic dissection of GABAergic inhibitory circuitry by systematically targeting distinct cell types. This approach establishes concrete entry points for studying cortical circuits, builds a solid middle ground that coherently link systems neuroscience and molecular-development neuroscience, and provides a paradigm to examine the development mental trajectory of circuit pathogenesis in models of neuropsychiatric disorders. He is the recipient of Pew Scholar Award in Biomedical Science, McKnight Scholar Award in Neuroscience, Distinguished Investigator of NARSAD-Brain and Behavior Research Foundation, and Simon's Investigator of Simons Foundation Autism Research Initiative

#### Surprise at the Synapse: MHC Class I, Pruning and Plasticity

<u>Carla Shatz</u> Stanford School of Medicine

Connections in adult brain are highly precise, but they do not start out that way. Precision emerges during development as synaptic connections remodel in a process requiring neural activity (action potentials and synaptic transmission). Activity also regulates neuronal gene expression. In an unbiased screen, Major Histocompatibility Class I (MHCI) genes were unexpectedly discovered to be in neurons, at synapses and regulated by activity and visual experience (Corriveau et al, 1998). To assess requirements for MHCI in CNS, mutant mice lacking stable surface expression of all MHCI, or of specific MHCI genes Kb and Db, were examined. Synapse pruning in developing visual system fails, and ocular dominance (OD) plasticity in visual cortex is greater than in WT (Huh et al, 2000; Datwani et al, 2009). In a search for receptors that could interact with neuronal MHCI, PirB, an innate immune receptor, was found expressed in neurons throughout mouse CNS. In mutant mice lacking PirB, OD plasticity is enhanced(Syken et al., 2006), LTP and LTD are altered, and spine density on L Pyramidal neurons is increased. Thus, PirB, like MHCI, appears to act to "brake" synaptic plasticity. The commonality of phenotypes present in these mice suggests a model (Shatz, 2009) in which PirB may bind and transduce signals from MHCI ligands in neurons. Together, results imply that this family of molecules, thought previously to function only in immunity, may also act at neuronal synapses to limit how much- or how quickly- synapse strength changes in response to new experience. These molecules may be crucial for controlling circuit excitability and stability in developing as well as adult brain. Changes in their function could contribute to developmental disorders such as Autism and Schizophrenia.

Dr. Carla Shatz is Sapp Family Provostial Professor of Biology and Neurobiology and Director of Bio-X, Stanford University's pioneering interdisciplinary biosciences program that brings together faculty from across the entire university-Clinicians, Biologists, Engineers, Physicists, Computer Scientists- to unlock the secrets of the human body. She received her B.A. in Chemistry from Radcliffe College in 1969, an M.Phil. in Physiology in 1971 from University College London as a Marshall Scholar, and a Ph.D. in Neurobiology from Harvard Medical

#### Surprise at the Synapse: MHC Class I, Pruning and Plasticity

#### Carla Shatz (continued)

PL

School in 1976. Dr. Shatz is a neuroscientist who has devoted her research career to understanding the dynamic interplay between genes and environment that shapes brain circuits - the very essence of our being. Her research on cellular and molecular mechanisms of how the early developing brain is transformed into adult circuitry during critical periods of development has relevance not only for treating disorders such as autism and schizophrenia, but also for understanding how the nervous and immune systems interact. Dr. Shatz is past president of the 40,000 member Society for Neuroscience; prior to Stanford, she was Chairwoman of the Department of Neurobiology at Harvard Medical School. She has received many awards and honors including election to the the National Academy of Sciences, the American Philosophical Society, the Institute of Medicine. In 2011 she was elected as a Foreign Member of the Royal Society of London. Most recently (2013), she received the Sackler Prize for Distinguished Achievement in Developmental Psychobiology and she shared the Robert J. and Claire Pasarow Foundation Award in Neuropsychiatry Research with Karl Deisseroth and Helen Mayberg.

#### **Dynamic Modulation of Dorsolateral Prefrontal Cortex Microcircuits: Focus of Vulnerability in Mental Disease**

#### Amy Arnsten

Yale University School of Medicine

Working memory relies on layer III pyramidal cell circuits in the dorsolateral prefrontal cortex (dlPFC), the circuits most afflicted in patients with schizophrenia. These neurons excite each other through NMDA-NR2B receptor synapses on spines to keep information "in mind" in the absence of sensory stimulation. The strength of these synaptic connections is dynamically gated by the arousal systems. Cholinergic stimulation of nicotinic alpha7 receptors permits NMDA actions, while catecholamines sculpt or strengthen inputs through regulation of feedforward cAMP-Ca<sup>2+</sup>- $K^+$  channel signaling. For example, exposure to an uncontrollable stressor causes a rapid loss of dIPFC neuronal firing via cAMP-PKA opening of HCN and KCNQ channels near the synapse, while DISC1 anchors PDE4A to regulate this process. Many of the proteins regulating layer III dlPFC network strength are genetically altered in patients with schizophrenia, suggesting that this is a key site for vulnerabilities in mental illness. Dysregulation of cAMP-Ca<sup>2+</sup>-K<sup>+</sup> channel signaling with advancing age also contributes to age-related cognitive decline, and may increase susceptibility for Alzheimer's Disease. In contrast, agents that inhibit Ca<sup>2+</sup>-cAMP signaling in PFC, such as the alpha2A-AR agonist guanfacine, are an effective new strategy for treating PFC cognitive deficits.

Dr. Amy F.T. Arnsten is Professor of Neurobiology at Yale Medical School. She received her BA from Brown University, and her Ph.D. in Neuroscience from UCSD, followed by post-doctoral fellowships with Dr. Susan Iversen at Cambridge, and Dr. Patricia Goldman-Rakic at Yale. Dr. Arnsten studies the molecular regulation of the primate cortex, with the goal of discovering informed strategies for the treatment of cognitive disorders. 11:30 a.m. – 1:00 p.m. Women's Luncheon Great Hall 5 & 6

# Women's Luncheon

# "ACNP Women Presidents Panel: Past, Present, and Future Challenges"

#### Presented by the Women's Task Force

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

Panelists:

Huda Akil

Raquel Gur

Judith Rapaport

Carol Tamminga

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

#### **Distinguished Lecture**

## The Story of Rett Syndrome and the Insight it Provides into Neuropsychiatric Disorders

Presented by: Huda Zoghbi PL

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

PL

#### The Story of Rett Syndrome and the Insight it Provides into Neuropsychiatric Disorders

<u>Huda Zoghbi</u> Baylor College of Medicine

Rett Syndrome, a postnatal neurological disorder that causes a broad range of severe neurological and behavioral disabilities, is fascinating in that its symptoms appear after a period of normal development and point to disturbances in most brain cells and regions. The quest for the gene revealed that the disease is caused by mutations in the X-linked *MECP2*. Interestingly, some patients with either hypomorphic mutations in *MECP2* or favorable patterns of X chromsome inactivation present with mild intellectual disabilities and psychiatric phenotypes. The path from gene discovery to therapy, however, is not a straightforward one and requires deep understanding of pathogenic mechanisms and key molecular and anatomical determinants of various symptoms and pathologies. We have used genetic, behavioral, physiological and molecular approaches to interrogate the pathogenesis of Rett and *MECP2* disorders. Recent discoveries suggest that MeCP2 is critical for many neuronal functions, especially for the ability of neurons to respond to change. Moreover, the findings reveal functions of the protein that were not suspected previously.

Huda Zoghbi is Professor of Pediatrics, Neurology, Neuroscience, and Molecular and Human Genetics at Baylor College of Medicine and serves as an Investigator with the Howard Hughes Medical Institute. She is also the Director of the Jan and Dan Duncan Neurological Research Institute at Texas Children's Hospital. Zoghbi's interest is in using the tools of modern genetics to understand the proper development of the brain as well as what goes awry in specific neurodevelopmental and neurodegenerative conditions. She has published seminal work regarding the molecular basis of Rett syndrome and of late-onset neurodegenerative diseases. Dr. Zoghbi is a member of several professional organizations including the McKnight Foundation Neuroscience Board and is a senior editor for the newly founded journal eLife. In 2000 she was elected to the Institute of Medicine, and in 2004 she was elected to the National Academy of Sciences. Among Dr. Zoghbi's honors are, the IPSEN prize in neuronal plasticity, the Bristol Myers-Squibb Neuroscience Distinguished Achievement Award, the Vilcek Prize, the Gruber prize in Neuroscience, and the Dickson prize in Medicine.

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

#### Neuronal Immaturity in Schizophrenia

Chair: Mickey Matsumoto

- 3:00 p.m. GABA Signaling in Postmortem Human Brain and Schizophrenia: A Question of Immaturity? *Joel E. Kleinman*
- 3:25 p.m. Immature Neurons in Schizophrenia? Support from Investigations on Proteoglycan Expression Sabina Berretta
- 3:50 p.m. Immature Dentate Gyrus as a Candidate Endophenotype of Neuropsychiatric Disorders *Tsuyoshi Miyakawa*

4:15 p.m. – 5:30 p.m. Mini-Panel Diplomat Ballroom 1 & 2

# Social Processes Initiative in Neurobiology of the Schizophrenia(s)

Chair: Anil Malhotra

- 4:15 p.m. The Neural Circuitry of Social Impairments in Schizophrenia Spectrum Disorders *Robert W. Buchanan*
- 4:40 p.m. Altered Structural and Functional Network Topology in Deficit Schizophrenia *Philip R. Szeszko*
- 5:05 p.m. Network Topology in Deficit Schizophrenia, Nondeficit Schizophrenia, and Bipolar Sisorder: From Circuits to Functional Outcome

Aristotle Voineskos

MP

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

# Autism Spectrum Disorders: From Rare Chromosomal Abnormalities to Common Molecular Targets

Chair: Ted Abel Co-Chair: Noboru Hiroi

| 3:00 p.m. | Role of Copy Number Variants in Autism Spectrum Disorders<br>Santhosh Girirajan  |
|-----------|--|
| 3:30 p.m. | Comprehensive Phenotyping of Mouse Autism Models<br>Ted Abel   |
| 4:00 p.m. | Tbx1 and Sept5 Contribute to Behavioral and Neuronal<br>Phenotypes in MouseModels of 22q11.2-Associated ASD<br><i>Noboru Hiroi</i> |
| 4:30 p.m. | The Translation of Translational Control in Autism Spectrum<br>Disorders<br><i>Eric Klann</i>                                      |
| 5:00 p.m. | Discussant: Alice Luo Clayton  |

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

### Can Biology Inform Treatment Prediction and Selection in Depression?

Chair: Amit Etkin Co-Chair: Madhukar Trivedi

3:00 p.m. Large-scale Pre-treatment Prediction of Remission with Antidepressants for Individual Patients Based on Cognitive and Emotional Test Performance, as Well as Its Neuroimaging Correlates Amit Etkin 3:30 p.m. Initial Results of the NIMH-funded EMBARC Study Madhukar Trivedi 4:00 p.m. Inflammatory Biomarkers Predict Differential Outcome of Depression Treatment with Escitalorpam and Nortriptyline in the **GENDEP** Project Rudolf Uher 4:30 p.m. Brain Serotonin 1A Receptor Binding as a Predictor of Treatment Outcome in Major Depressive Disorder Ramin V. Parsey 5:00 p.m. Discussant: Thomas R. Insel

3:00 p.m. – 5:30 p.m. Panel Atlantic Ballroom 3

# **Circuitry Underlying Obsessive-compulsive Disorder:** Lessons from Deep Brain Stimulation and Ablative Surgery

Chair: Suzanne Haber Co-Chair: Gregory J. Quirk

| 3:00 p.m. | Ablative Limbic System Surgery for the Treatment of OCD<br>Emad N. Eskandar   |
|-----------|---|
| 3:30 p.m. | The Circuitry of Deep Brain Stimulation and Cingulatomy:<br>Monkey Tracing vs. Human Tracking<br>Suzanne Haber                          |
| 4:00 p.m. | DBS of Ventral Striatum in Rodents Modulates Fear Extinction<br>via Prefrontal and Orbitofrontal Projections<br><i>Gregory J. Quirk</i> |
| 4:30 p.m. | Deep Brain Stimulation for Intractable OCD: Population and<br>Outcomes<br>Benjamin Greenberg  |
| 5:00 p.m. | Discussant: Scott Rauch   |

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

# Kicking Over the Traces -Noncatecholic Biogenic Amines and Their Receptors

#### Chair: David K. Grandy Co-Chair: Gregory M. Miller

| 3:00 p.m. | Evidence from Molecular Modeling, Site-Directed Mutagenesis<br>and Behavioral Testing Indicate Trace Amine-Associated<br>Receptor 1 is a Methamphetamine Receptor<br><i>David K. Grandy</i> |
|-----------|---|
| 3:30 p.m. | Selective TAAR1 Ligands and Transgenic Animal Models<br>Reveal a Role of TAAR1 in Cognitive, Neurologic and<br>Psychiatric Disorders<br><i>Raul R. Gainetdinov</i>                          |
| 4:00 p.m. | Trace Amine Associated Receptor 1 Modulation of the<br>Rewarding and Immunological Effects of Drugs of Abuse<br>Supports its Relevance as a Therapeutic Target<br><i>Gregory M. Miller</i>  |
| 4:30 p.m. | The Activation of Intracellular Signaling Systems by<br>Amphetamines: A Potential Role for Trace Amine Receptors<br>Susan G. Amara  |
| 5:00 p.m. | Discussant: David Shurtleff   |

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

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# Structural and Functional Brain Changes in Young People at Risk for Severe Mental Illness

Chair: Martin Alda Co-Chair: Nitin Gogtay

| 3:00 p. | Clinical Stages and Developmental Trajectories of Bipolar<br>Disorder: Family-based Analysis<br><i>Martin Alda</i>                                     |
|---------|--|
| 3:30 p. | Population Neuroscience and Psychiatric Genetics: A Two-way<br>Street<br>Tomas Paus  |
| 4:00 p. | Vulnerability or Resilience? Brain Developmental Studies in No.<br>Psychotic Siblings of Childhood Onset Schizophrenia Patients<br><i>Nitin Gogtay</i> |
| 4:30 p. | Neuroanatomical Changes in Bipolar Disorders – Causes Versus<br>Consequences of the Illness<br><i>Tomas Hajek</i>                                      |
| 5:00 p. | Discussant: Mary L. Phillips   |

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

# The Role of Inflammation in the Pathophysiology of Mood, Aggressive and Medical Disorders: A Deadly Combination

Chair: Emil F. Coccaro

| 3:00 p.m. | Plasma Markers of Inflammation are Elevated in Subjects with<br>Intermittent Explosive Disorder and Correlate Directly with<br>Aggression in Human Subjects<br><i>Emil F. Coccaro</i>  |
|-----------|--|
| 3:30 p.m. | Stress, Trauma, and Inflammation in Non-Psychiatric Subjects<br>Janice Kiecolt-Glaser  |
| 4:00 p.m. | Inflammation and Depression: Sleep Disturbance Moderates<br>Induction of Depressed Mood by an Inflammatory Challenge<br><i>Michael R. Irwin</i>  |
| 4:30 p.m. | Transcriptional Signatures Related to Glucose and Lipid<br>Metabolism Predict Treatment Response to the Tumor Necrosis<br>Factor Antagonist Infliximab in Patients with Treatment-<br>Resistant Depression<br>Jennifer C. Felger |
| 5:00 p.m. | Discussant: Charles B. Nemeroff  |

7:30 p.m. – 9:00 p.m. Study Group Atlantic Ballroom 2

#### Medical and Non-medical Use of Stimulant Drugs for Cognitive Enhancement

Chair: James Swanson Co-Chair: Wilson M. Compton

Participants: William Pelham Trevor W. Robbins Barbara J. Sahakian James T. McCracken Susanna N. Visser Ruben Baler Kathleen Ries. Merikangas Raul Gonzalez James G. Waxmonsky Tim Wigal Marc Lerner

7:30 p.m. – 9:00 p.m. Study Group Regency Ballroom 2

### Mental Illness, Violence and the Gun Control Debate: Evidence, Policy, Privacy and Stigma - on Behalf of the ACNP Ethics Committee

Chair: David Pickar Co-Chair: Jerrold Rosenbaum

> Participants: David Pickar Jerrold Rosenbaum Emil F. Coccaro Kenneth L. Davis Paul S. Appelbaum Brian Frosh J. Dee Higley

> > 7:30 p.m. – 9:00 p.m. Study Group Regency Ballroom 3

# New Models of Open Innovation to Rejuvenate the Biopharmaceutical Ecosystem, A Proposal by the ACNP Liaison Committee

Chair: Dean F. Wong Co-Chair: Lisa Gold

Participants: Dean F. Wong Robert Innis Lawrence M. Sung Steven Paul Phillip Phan Steven Grant Husseini Manji

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

## The Assessment Of Suicidal Ideation, Behavior & Risk: At Baseline; As a Measure of Clinical Outcome, and/or as a Treatment Emergent SAE

Co-Chair: Eric Youngstrom Chair: Roger E. Meyer

Participants: Roger E. Meyer Ahmad Hameed John Greist Phillip Chappell J. John Mann David V. Sheehan Cheryl McCullumsmith Larry Alphs Richard C. Shelton Paula J. Clayton Kelly Posner

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

## The Challenges of Designing and Interpreting Clinical Trials with Depot Antipsychotics

Chair: W. Wolfgang Fleischhacker

Participants: Raymond Sanchez Srihari Gopal Maxine X. Patel Stephan Heres Keith H. Nuechterlein Hiroyuki Uchida

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| Notes | 4     | ACNP 52nd Annual Meeting | Final Program |
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8:30 a.m. – 9:45 a.m. Mini-Panel Diplomat Ballroom 1 & 2

# After the Trauma: Developmental Trajectories from Childhood to Adult Psychiatric Disorders

Chair: Michael D. De Bellis

- 8:30 a.m. The Neurobiology of PTSD Symptoms in Maltreated Children and Adolescents *Michael D. De Bellis*
- 8:55 a.m. Sex-specific Effects of Childhood Emotional Abuse on Affective Processing in Bipolar Disorder Patients *Katherine E. Burdick*
- 9:20 a.m. The Long-Term Consequences of Childhood Maltreatment: Effects on Brain Structure and Subclinical Psychopathology in Healthy Adults Pamela DeRosse

MP

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9:45 a.m. – 11:00 a.m. Mini-Panel Diplomat Ballroom 1 & 2

## Biochemical and Behavioral Pharmacology of Synthetic Cathinone Derivatives Found in Psychoactive Bath Salts Products

Chair: Richard B. Rothman Co-Chair: Michael H. Baumann

- 9:45 a.m. Effects of Newly-emerging Synthetic Cathinone Derivatives on Monoamine Transporter Function in Rats *Michael H. Baumann*
- 10:10 a.m. Intravenous Self-administration of
  3,4-Methylenedioxypyrovalerone (MDPV) and
  4-Methylmethcathinone (4-MMC, Mephedrone) in Rats
  Michael A. Taffe
- 10:35 a.m. Abuse-related and Abuse-limiting Effects of Synthetic Cathinone "Bath Salt" Derivatives on Intracranial Self-Stimulation in Rats *Matthew L. Banks*

MP

8:30 a.m. – 11:00 a.m. Panel Atlantic Ballroom 2

# At the Crossroads of Physics, Physiology, and Psychiatry: Rational Design of Noninvasive Neuromodulation Therapies

Chair: Sarah Lisanby

- 8:30 a.m. Targeting of Transcranial Direct Current Stimulation: Insights from Cellular and Computational Models Marom Bikson
- 9:00 a.m. Optimizing Stimulus Pulse Characteristics for Transcranial Magnetic Stimulation and Electroconvulsive Therapy Via Device Development, Computational Modeling, and Biophysicallymotivated Dosing Paradigms *Angel V. Peterchev*
- 9:30 a.m. Mechanisms of Targeting Cortical State Dynamics with Neuromodulation *Flavio Frohlich*
- 10:00 a.m. Enhancement of Working Memory in Sleep Deprived Young Adults and in Elderly Adults using rTMS Informed by Covariance-modeled fMRI Bruce Luber
- 10:30 a.m. Discussant: Zafiris J. Daskalakis

8:30 a.m. – 11:00 a.m. Panel Regency Ballroom 3

# Augmentation of Antidepressant Response by Autoreceptor-Mediated Mechanisms: Clinical Experience and Mechanisms of Action

Chair: Salomon Z. Langer Co-Chair: Torgny H. Svensson

| 8:30 a.m.  | Autoreceptor-mediated Regulation of Neurotransmission:<br>Pharmacological Targets and Potential for Improved Treatment<br>of Major Psychiatric Disorders<br>Salomon Z. Langer                           |
|------------|---|
| 9:00 a.m.  | Rapid Augmentation of Antidepressant Effect in Treatment-<br>resistant MDD by Add-on Low Dose Aripiprazole<br>Daniel E. Casey   |
| 9:30 a.m.  | Low Doses of Atypical Antipsychotic Drugs Added to Selective<br>Serotonin Inhibitors Produce a Ketamine-like Facilitation of<br>Prefrontal Glutamatergic Neurotransmission<br><i>Torgny H. Svensson</i> |
| 10:00 a.m. | Emerging Role of Atypical Antipsychotics as Add-on Therapy in<br>Major Depression<br>Siegfried Kasper   |
| 10:30 a.m. | Discussant: Dennis S. Charney   |

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8:30 a.m. – 11:00 a.m. Panel Regency Ballroom 2

# Neuroactive Steroids and Oxysterols as Endogenous Modulators of GABA and Glutamate Receptors: Basic Mechanisms and Therapeutic Implications

Chair: Steven Paul Co-Chair: Charles Zorumski

- 8:30 a.m. GABAergic Neurosteroids as Novel Targets for Therapeutic Drug Development in Psychiatry *Charles Zorumski*
- 9:00 a.m. Natural and Synthetic Neuroactive Steroids and Oxysterols as Potent NMDA Receptor Allosteric Modulators: Therapeutic Considerations Steven Paul
- 9:30 a.m. Neuroactive Steroids Ganaxolone and Allopregnanolone in the Treatment of Epilepsy, Traumatic Brain Injury, and Neurobehavioral Disorders *Michael A. Rogawski*
- 10:00 a.m. Neurosteroids as Novel Therapeutics and Biomarker Candidates in Schizophrenia and PTSD *Christine E. Marx*
- 10:30 a.m. Discussant: Bruce S. McEwen

8:30 a.m. – 11:00 a.m. Panel Atlantic Ballroom 3

#### Nutrition, Neurodevelopment, and Risk for Schizophrenia and Autism: From Epidemiology to Epigenetics

Chair: Joshua L. Roffman Co-Chair: Donald Goff

- 8:30 a.m. Periconceptional Folic Acid and Neurodevelopmental Disorders: Historical Context and Current Research *Ezra Susser*
- 9:00 a.m. Effects of Periconceptional Folate on Language Delay and Autism Spectrum Disorders: The Norwegian Mother and Child Cohort Study *Camilla Stoltenberg*
- 9:30 a.m. Longitudinal Effects of In Utero Folate Exposure on Cortical Thickness: Implications for Neurodevelopmental Disorders Joshua L. Roffman
- 10:00 a.m. The Placental and Neuronal Methylomes at the Interface of Genetic and Environmental Risk and Protective Factors in Autism

Janine LaSalle

10:30 a.m. Discussant: Donald Goff
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8:30 a.m. – 11:00 a.m. Panel Regency Ballroom 1

# Peripheral Immune and Endocrine Pathways as Markers of PTSD Risk and Symptom Development: Evidence from Prospective Studies

Chair: Victoria Risbrough

- 8:30 a.m. Longitudinal Plasma Testosterone Trajectory and its Relation to Combat, Temperament and PTSD *Eric Vermetten*
- 9:00 a.m. Evidence for Plasma C-Reactive Protein Concentration as Biomarker of PTSD Risk Dewleen G. Baker
- 9:30 a.m. Blood-Based Gene-expression Predictors of PTSD Risk and Resilience Among Deployed Marines: A Pilot Study Stephen J. Glatt
- 10:00 a.m. Exaggerated Threat Sensitivity and Avoidance as Contributors to Elevated Inflammation in Posttraumatic Stress Disorder: Data from the Mind Your Heart Study *Aoife O'Donovan*
- 10:30 a.m. Discussant: Thomas Neylan

8:30 a.m. – 11:00 a.m. Panel Atlantic Ballroom 1

# The Future of Translational Research in Addiction

Chair: Harriet de Wit

| 8:30 a.m.  | Cross-species Behavioral Tests for Investigations of Addictive<br>and Psychiatric Disorders<br><i>Mark A. Geyer</i> |  |
|------------|---|--|
| 9:00 a.m.  | Neurocircuitries for Social Stress and Drug Abuse: Novel Targets<br>for Intervention<br><i>Klaus A. Miczek</i>      |  |
| 9:30 a.m.  | Towards Consilience in Animal and Human Behavioral Mode<br>in Addiction<br>David Stephens                           |  |
| 10:00 a.m. | Identifying the Molecular Determinants of Inhibitory Control<br>Problems in Addictions<br>J. David Jentsch          |  |
| 10:30 a.m. | Discussant: Harriet de Wit  |  |

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#### **Data Blitz Session**

Chair: William Carlezon

This session is comprised of rigorously timed 5 minute presentations by 12 young investigators.

- 11:30 a.m. Can Serotonin Put Your Mind at Rest? Julia Sacher
- 11:40 a.m. Effects of Pharmacogenetic Manipulation of the Nucleus Ac+H3cumbens on Neuronal Activity and Alcohol-Related Behaviors Angela Ozburn
- 11:50 a.m. Feedforward and Feedback Control Abnormalities During Precision Grasping Implicate Cerebellar Dysfunction in Autism Spectrum Disorder *Matthew Mosconi*
- 12:00 p.m. Frequency and Characteristics of Isolated Psychiatric Episodes in Anti-NMDA Receptor Encephalitis *Matthew Kayser*
- 12:10 p.m. Genetic Background Regulates the Effect of Antidepressant Treatment on Behavioral Despair and Hippocampal Neurogenesis in Mice *Brooke Miller*
- 12:20 p.m. High-throughput Behavior-based Neuroactive Drug Discovery in Zebrafish David Kokel
- 12:30 p.m. Modulation of N-methyl-D-aspartate (NMDAR)-type Glutamate Receptors in Psychiatric Disorders Joshua Kantrowitz

PL

#### **Data Blitz Session**

Chair: William Carlezon

- 12:40 p.m. Oxytocin and Facial Expressivity in Patients with Schizophrenia and Healthy Participants Josh Woolley
- 12:50 p.m. Poverty and the Past: The Relation between Hippocampus Function and Memory Performance is linked to Childhood Poverty Elizabeth Duval
- 1:00 p.m. Rare Genetic Variants in VMAT1 (SLC18A1) Are Functional in Vitro and Associated with Bipolar Disorder *Falk Lohoff*
- 1:10 p.m. Real-time Functional MRI Feedback, Compared to Sham, Reduces Cue-Induced Nicotine Craving in Smokers: Results from the First Clinical Trial *Colleen Hanlon*
- 1:20 p.m. Substrate-selective COX-2 Inhibition Decreases Anxiety via Endocannabinoid Activation Sachin Patel

PL

#### **Can Serotonin Put Your Mind at Rest?**

Tuesday, Poster #192

Alexander Schäfer, Inga Burmann, Ralf Regenthal, Katrin Arelin, Andre Pampel, Arno Villringer, Daniel Margulies, <u>Julia Sacher</u> Max Planck Institute for Human Cognitive and Brain Sciences

**Background:** The serotonin transporter (5-HTT) is essential to maintaining adequate brain serotonin homeostasis, and alteration of its function has been linked to heightened susceptibility for depression and anxiety (Holmes et al. 2003). Differences in the 5-HTT genotype have also been recently related to variation in intrinsic functional brain organization (Li et al., 2012). While preliminary evidence supports a connection between the serotonergic system and intrinsic brain activity, the precise role of serotonin in modulating its functional organization is not known. Here we demonstrate that a single dose of a selective serotonin reuptake inhibitor (SSRI) dramatically alters intrinsic functional connectivity throughout the human brain.

**Methods:** Degree centrality (DC) mapping of resting-state functional magnetic resonance imaging (rs-fMRI) data was applied to twenty-one individual datasets of healthy, anti-depressant naïve participants following a single oral dose of the selective serotonin reuptake inhibitor (SSRI) escitalopram in a randomized placebo-controlled design. Degree centrality measures connectivity by counting the number of connections of each specific node. This number is then assigned as a centrality value to the given node (a node is defined by each separate voxel in gray matter resulting in a network of ~ 63000 voxels). rs-fMRI data was acquired on a Siemens Verio 3 tesla scanner equipped with a 32-channel head coil (410 volumes, TR=2000ms). Standard image preprocessing was performed using FSL and AFNI (Biswal et al., 2010).

**Results:** DC-analysis revealed a widespread decrease in connectivity in most cortical and subcortical areas (p=0.01, cluster corrected) following the oral intake of a single dose of 20 mg escitalopram. While the majority of functional connectivity decreased, localized increases were observed in cerebellar and thalamic regions. These connectivity changes could not be explained by alterations in the local signal properties, such as the amplitude of the resting state signal, and appeared to be specific to the correlation between regions which points towards

#### **Can Serotonin Put Your Mind at Rest?**

Tuesday, Poster #192 (continued)

#### Julia Sacher

an alteration in long range synchronization. It is noteworthy that these neural changes were also reflected in behavioral findings of significantly increased Visual Analogue Scores (VAS)-scores for concentration, alertness, attention and coordination (p<0.001, post-hoc Bonferroni corrected).

**Discussion:** The increase in connectivity found in the thalamus and cerebellum may be of particular relevance for the excitability of the many serotonergic projection neurons that terminate in the thalamus. By cerebellar modulation these neurons can turn from burst into tonic mode, a mechanism hypothesized to alert cortical networks. This conceptional framework is further supported by the reported increases in concentration, alertness, attention and coordination. Our findings are the first to directly link a single dose of an SSRI to such a substantial mechanism of modulating intrinsic functional connectivity in the human brain. The evidence we present for an acute and global change in connectivity following a single dose of escitalopram is a first step towards identifying noninvasive neural biomarkers for individual responsivity of the human brain to serotonergic modulation.

# **Effects of Pharmacogenetic Manipulation of the Nucleus Accumbens on Neuronal Activity and Alcohol-Related Behaviors**

Tuesday, Poster #53

<u>Angela Ozburn</u>, Ryan Logan, Puja Parekh, Jake Bosin, Colleen A. McClung University of Pittsburgh Medical Center

**Background:** Chronic alcohol intake leads to long lasting changes in rewardand stress-related neuronal circuitry. The nucleus accumbens (NAc) is an integral component of this circuitry. Promising clinical trials have shown that deep brain stimulation of the NAc decreases alcohol craving and relapse in alcohol dependent subjects. Here, we used a cutting edge pharmacogenetic approach to induce activity in the NAc to reduce alcohol-related behaviors in mice. We used the mutagenized muscarinic G protein-coupled receptors hM3Dq and hM4Di that are selectively activated by the pharmacologically inert and orally bioavailable drug, clozapine-N-oxide (CNO). We tested the ability of these channels to change NAc activity and assessed the effects of altered NAc activity to alter binge-like alcohol drinking, tastant intake, and reward.

**Methods:** Mice were stereotaxically injected with AAV2 hSyn-HA hM3Dq, -hM4Di, or -eGFP bilaterally into NAc. Experiments were carried out to verify CNO induced changes in NAc activity (via ex-vivo whole cell electrophysiological recordings). We tested the effect of altering NAc activity on binge ethanol intake (or intake of sucrose, quinine, and water) using the drinking in the dark paradigm (n=9-10/group). We also evaluated the effects of altering NAc activity on the rewarding properties of ethanol using conditioned place preference (n=7-10/group).

**Results:** CNO increased NAc firing in hM3Dq positive cells and decreased firing in hM4Di cells, confirming the ability of these channels to spatially and temporally alter neuronal activity. Increasing NAc activity significantly decreased binge drinking (p<0.05) without altering intake for other tastants. Increasing NAc activity is not rewarding and altering NAc activity does not change the rewarding properties of ethanol.

**Discussion:** These experiments demonstrate that neuronal activity can be controlled in a spatial and temporal manner using pharmacogenetics. We find that

# **Effects of Pharmacogenetic Manipulation of the Nucleus Accumbens on Neuronal Activity and Alcohol-Related Behaviors**

Tuesday, Poster #53 (continued)

#### Angela Ozburn

increasing NAc activity decreases binge drinking without altering the rewarding properties of ethanol. Ongoing experiments aim to identify the the effects of altering NAc activity on the aversive properties of ethanol, and identifying transcriptional changes induced by this pharmacogenetic manipulation. These findings could have promising implications for treatment.

PL

# Feedforward and Feedback Control Abnormalities During Precision Grasping Implicate Cerebellar Dysfunction in Autism Spectrum Disorder

Tuesday, Poster #182

<u>Matthew W. Mosconi</u>, Suman Mohanty, Rachel K. Greene, Lauren Schmitt, David E. Vaillancourt, John A. Sweeney University of Texas Southwestern

**Background:** Sensorimotor impairments are present in the majority of individuals with autism spectrum disorder (ASD). Yet, these deficits and their neurophysiological mechanisms have not been systematically assessed. In the present study, we examined visually guided fine motor control in individuals with ASD. The relative influence of the quality of sensory input on movement abnormalities in ASD was examined by varying the precision of visual feedback. Methods: Twenty-eight individuals with ASD and 29 healthy controls matched on age (range: 6-35 years), IQ and handedness performed precision grip force tasks in which they viewed a white FORCE bar on a screen that moved upwards with increased manual force toward a fixed green TARGET bar. In the first experiment, subjects were instructed to reach a target force level as fast as they could, and to sustain the target force level for the duration of the trial (15 sec). Target force levels were varied between 5-85% of each individual's maximum force across trials. To assess the integrity of feedforward control mechanisms, we examined the accuracy and force dynamics of initial, rapid force generation. To assess feedback control mechanisms, force accuracy and variability were measured during sustained force generation. The regularity of each subject's sustained force time series also was examined to determine the degree to which individuals made online adjustments to refine their performance. A second experiment was performed to assess the relative impact of changes in sensory input on sustained precision force generation. During this task, the vertical distance the FORCE bar moved per Newton of force was varied between .12-145 mm. Thus, in experiment 2, visual feedback precision was increased by moving the FORCE bar a greater distance for every Newton of force generated.

Results: Primary responses generated by feedforward mechanisms were

## Feedforward and Feedback Control Abnormalities During Precision Grasping Implicate Cerebellar Dysfunction in Autism Spectrum Disorder

Tuesday, Poster #182 (continued)

#### Matthew W. Mosconi

hypermetric for individuals with ASD compared to controls. Individuals with ASD also showed increased rates of force increase during their primary movements that were associated with the degree to which they overshot target force levels. During sustained force generation in which subjects attempted to maintain alignment of the force and target bars, individuals with ASD demonstrated increased force error and variability. These deficits implicating feedback control alterations were more severe at larger force levels, and at the most and the least precise visual feedback conditions. Spectral analyses showed that sustained force deficits in individuals with ASD were associated with reduced power in the 0-1 Hz frequency range and increased power at higher frequencies (1-3 Hz). Across all force levels and all levels of visual feedback precision, the force time series of subjects with ASD was less complex, or more regular.

**Discussion:** These studies identified three distinct sensorimotor deficits in individuals with ASD. First, individuals with ASD show reduced accuracy and alterations in their force dynamics during primary motor responses suggesting that forward control mechanisms are disrupted in this disorder. Second, reduced accuracy of sustained motor responses indicates that feedback control systems also are impaired in ASD. The severity of feedback control deficits covaried with changes in both the motor execution and sensory processing demands, implicating abnormalities in both input and output processes. Third, increased force variability and increased regularity in the time-dependent structure of sustained precision force suggest that individuals with ASD utilize fewer degrees of freedom to correct their force precision during goal-directed actions. Taken together, these behavioral findings are consistent with the hypothesis that dysfunctions within cerebellar circuitry in ASD lead to both hypermetric feedforward motor processes and more variable sensory feedback control of motor commands.

## Frequency and Characteristics of Isolated Psychiatric Episodes in Anti-NMDA Receptor Encephalitis

Tuesday, Poster #190

<u>Matthew Kayser</u>, Maarten Titulaer, Nuria Gresa-Arribas, Josep Dalmau Perelman School of Medicine at the University of Pennsylvania

**Background:** Anti-N-methyl-D-aspartate (NMDA) receptor encephalitis is an autoimmune disorder in which IgG antibodies are directed against the NR1 subunit of the NMDA receptor (NMDAR). The disorder includes a range of psychotic symptoms early in the course of the disease followed by neurologic involvement, and ultimately protracted cognitive and behavioral symptoms. The occurrence of severe behavioral changes reminiscent of a schizophrenia-like illness has fueled speculation that this disorder might define a subset of patients misdiagnosed with a primary psychiatric disease. Hoewever, the frequency and type of isolated psychiatric episodes (pure psychiatric symptoms without neurological involvement) either as initial presentation of the disease or as relapse are unknown. This work aims to determine the frequency, symptoms, and outcome of isolated psychiatric episodes in a large cohort of patients with anti-NMDAR encephalitis.

**Methods:** This was an observational cohort of patients diagnosed over a 5 year period (median follow-up 2 years). 571 patients with IgG antibodies against the NR1 subunit of the NMDAR were included in the study. Antibody studies were performed at the Universities of Pennsylvania and Barcelona, and clinical information was obtained by the authors or referring physicians. We measured frequency, type of symptoms, and outcome of patients with anti-NMDAR encephalitis and isolated psychiatric manifestations. All patients had a detailed work up to rule out other disorders, including brain MRI, and blood and CSF studies. Isolated psychiatric presentations were defined as episodes (either initial presentation or relapse) that occurred in association with NMDAR antibodies in serum or CSF without neurological involvement. Relapse was defined by the new onset or worsening of symptoms at least two months after improvement or stabilization, without any other etiology involved, and persistent detection of NMDAR antibodies.

### Frequency and Characteristics of Isolated Psychiatric Episodes in Anti-NMDA Receptor Encephalitis

Tuesday, Poster #190 (continued)

#### Matthew Kayser

**Results:** 23/571 patients (4%) developed isolated psychiatric episodes, 5 at disease onset and 18 during relapses. For all 23 patients, age (median 20 years), gender (91% female), and tumor association (43%, ovarian teratoma) were similar to the population at large. Predominant symptoms included, delusional thinking (74%), mood disturbances (70%, usually manic), and aggression (57%). Brain MRI was abnormal in 10/22 (45%) and CSF showed pleocytosis in 17/22 (77%). Eighty three percent of the patients had full/substantial recovery after immunotherapy and tumor resection when appropriate. After relapse, 17/18 (94%) patients returned to a similar or better pre-relapse functional level.

**Discussion:** We report 23 patients with anti-NMDAR encephalitis who developed isolated psychiatric symptoms either as initial episode of the disease (5 patients) or as relapse of encephalitis (18 patients). Predominant symptoms included delusional thinking, auditory or visual hallucinations, and manic and aggressive behavior. The fact that 5 patients had initial psychiatric presentations without neurologic symptoms or past history of encephalitis suggests that some cases of anti-NMDAR encephalitis can be mistaken for a primary psychiatric disorder. Therefore, isolated psychiatric episodes are rare but can occur as initial onset or relapse of anti-NMDAR encephalitis. Recognition of these episodes is important because they respond to immunotherapy. In patients with new onset psychosis, history of encephalitis, subtle neurological symptoms, and/or abnormal ancillary tests should prompt screening for NMDAR antibodies.

# Genetic Background Regulates the Effect of Antidepressant Treatment on Behavioral Despair and Hippocampal Neurogenesis in Mice

Tuesday, Poster #90

<u>Brooke H. Miller</u>, Thomas A. Lanz, Zane Zeier, Miguel Lopez-Teledono, Robin Kleiman, Mathew Pletcher, Claes Wahlestedt University of Florida College of Medicine

**Background:** There is strong evidence that chronic treatment with antidepressants such as fluoxetine induces an increase in adult hippocampal cell proliferation and neuronal differentiation, and that this effect may be associated with the behavioral response to antidepressants.

**Methods:** In order to test the association between antidepressant efficacy and hippocampal neurogenesis, we treated mice from 30 inbred strains with chronic oral fluoxetine and measured the effect of drug treatment on behavioral despair and hippocampal gene expression. The effect of fluoxetine on neurogenesis (BrdU labeling) was measured in a subset of the 30 strains.

**Results:** We found that approximately 60% of the strains showed a positive behavioral response to fluoxetine treatment, similar to the percent response observed in human cohorts. Gene expression analysis identified a set of approximately 100 genes, many of which have been associated with neurogenesis, that clustered based on the strain-specific behavioral response to fluoxetine. This gene set was found to reliably predict the effect of fluoxetine on cell proliferation (as measured by BrdU labeling) in the dentate gyrus of a subset of the inbred strains. Subsequent genome-wide association mapping (GWAS) identified several genetic loci associated with both the behavioral and neurogenic response to fluoxetine.

**Discussion:** These results suggest that the behavioral response to fluoxetine is under genetic regulation and associated with hippocampal neurogenesis: strains that show a positive behavioral response to fluoxetine also show an increase in hippocampal neurogenesis, whereas no change in neurogenesis is observed in strains that do not show a behavioral response. Additional genetic and genomic analysis was used to identify gene networks and genomic loci that may regulate antidepressant efficacy.

# High-throughput Behavior-based Neuroactive Drug Discovery in Zebrafish

Tuesday, Poster #20

David Kokel Massachusetts General Hospital

**Background:** Behavioral phenotyping is an effective way to discover novel neuroactive drugs. However, it has been difficult to develop efficient behavioral phenotyping assays for large-scale chemical screens. New technologies and behavioral phenotyping assays in the zebrafish are opening new opportunities to understand the central nervous system (CNS) and discover neuroactive drugs. These technologies are ushering in a new phase of discovery-based research in behavioral pharmacology. Neuroactive compounds with new structures, targets, mechanisms, and functions are being discovered. Given the fundamental differences between the human and zebrafish nervous systems it will be difficult to translate zebrafish discoveries to clinical medicine. However, given the molecular genetic similarities between humans and zebrafish, it is likely that some of the compounds being identified in the zebrafish will find translational utility in humans. The greatest new successes in CNS drug discovery will likely leverage the advantages of many model systems, including *in vitro*, cellular and rodent models, in addition to zebrafish.

**Methods:** To determine how small molecules affect zebrafish behavior, we have built a fully automated phenotyping system capable of tracking and quantifying zebrafish behaviors in HT, 96-well format. The platform is a high content imaging system that combines robotic stimulus presentation with high-quality digital video capture and image processing algorithms.

**Results:** Preliminary screening of 30,000 compounds in one behavioral assay has identified 44 'hit' compounds and zero false positives among 5,155 DMSO-treated negative control wells. Of these 44 hits, 9 are known bioactive compounds. All 9 of the known hits are annotated as GABA receptor agonists. These data suggest that some of the 35 other novel hit compounds from this screen may also target GABAergic and other anxiety-related pathways.

# High-throughput Behavior-based Neuroactive Drug Discovery in Zebrafish

Tuesday, Poster #20 (continued)

#### David Kokel

**Discussion:** The compounds identified in this screen may have utility beyond anxiety-related research. It is difficult to predict exactly how behavior-modifying compounds will work, or what their translational utility will be. However, history suggests that psychiatric medicines are often discovered based on phenotypic observations. Given the efficacy of behavior-based neuroactive drug discovery, and the historical lack of high-throughput phenotyping technologies, it is likely that new large-scale behavior-based screening efforts in the zebrafish will successfully identify new neuroactive compounds.

PL

#### Modulation of N-methyl-D-aspartate (NMDAR)-type Glutamate Receptors in Psychiatric Disorders

Tuesday, Poster #156

<u>Joshua T. Kantrowitz</u>, Michael Epstein, Odeta Beggel, Nayla Scaramello, Gail Silipo, Elisa Dias, Stephanie Rohrig, Batsheva Halberstam, Marlene Carlson, Daniel C. Javitt Columbia University

**Background:** Over the past 20 years, attention has turned increasingly to dysfunction of the N-methyl-D-aspartate (NMDAR)-type glutamate receptors as a fundamental deficit underlying pathophysiology in major psychiatric disorders such as schizophrenia and affective illnesses. In schizophrenia, a major focus has been on development of compounds to enhance NMDAR function. Proof-ofprinciple trials have been conducted with glycine-site agonists including glycine and D-serine, and with high affinity glycine transport inhibitors. Although significant improvement has been observed on negative and total symptoms in some, but not all, studies, effects of these compounds on cognition remain relatively understudied. Furthermore, relatively low doses of D-serine have been used because of concerns regarding nephrotoxicity. Two studies have been done to assess potential effects of D-serine on cognitive function. The first investigated effects of high dose (60 mg/kg/d) D-serine X 6 weeks on neurocognition as assessed both with neurophysiological and neurocognitive measures. A second piloted effects of acute D-serine treatment in the enhancement of cognitive plasticity during an auditory learning task. In affective disorders, NMDAR antagonists represent a potential treatment modality for treatment resistant major depression and bipolar depressive disorders. Acute treatment with intravenous ketamine induces near-immediate relief for treatment resistant depressive symptoms that last for up to 2 weeks. However, practical approaches to prolong this acute benefit are still being developed. D-Cycloserine (DCS) is an antibiotic hat cross reacts with the glycine site of the NMDAR, and, at high dose, acts as a net NMDAR antagonist. Antidepressant effects of DCS were first described in the 1950's and have recently been confirmed in a double blind RCT of treatment resistant MDD. The present study investigates utility of acute ketamine challenge,

#### Modulation of N-methyl-D-aspartate (NMDAR)-type Glutamate Receptors in Psychiatric Disorders

Tuesday, Poster #156 (continued)

#### Joshua T. Kantrowitz

added to ongoing treatment, to induce improvement followed by high-dose DCS to maintain improvement, as a potential near-term, practical approach for MDD treatment. A concern with use of NMDAR antagonists is a risk of treatment emergent psychosis. In this study, DCS is added to ongoing treatment with antipsychotics (Seroquel, fluoxetine/olanzapine, lurasidone) also approved for treatment of bipolar depression.

**Methods:** We will present data from three NMDAR modulator studies, as follows: 1. **<u>ERP biomarker</u>**: In this study, neurophysiological and neurocognitive data were collected during a double blind, crossover study of high dose (60 mg/ kg/d) D-serine vs. placebo added to existing antipsychotic medication (n=16). These neurophysiological data were combined with previously unpublished measures obtained as part of a previously reported open label dose finding study (Kantrowitz 2010) (n=19). Primary outcome measures for neurophysiology included amplitude of the mismatch negativity (MMN) and visual P1 potentials, analyzed as described previously (Friedman 2012). The primary neurocognitive outcome measure was composite score on the MATRICS neuropsychological battery. Clinical symptoms were assessed using the PANSS.

2. <u>D-serine + cognitive remediation</u>: In this study, MMN was measured before and after subjects completed an auditory training program (coinciding with peak d-serine levels) and post intervention ERP (NCT01474395). Cognitive remediation consisted of an auditory frequency discrimination task shown to promote learning in healthy controls (Ahissar 2006). Outcome measures included tone matching accuracy and MMN.

3. <u>DCS in bipolar depression</u>: DCS has been reported to be effective in treatment of refractory MDD. This study evaluates effectiveness of DCS as maintenance treatment for bipolar depression after acute ketamine administration. DCS (1000 mg) was added to standard treatment for 8 weeks in an open label tolerability investigation.

#### Modulation of N-methyl-D-aspartate (NMDAR)-type Glutamate Receptors in Psychiatric Disorders

Tuesday, Poster #156 (continued)

#### Joshua T. Kantrowitz

**Results:**ERP biomarker:33 subjects with a mean age of  $42.7\pm9.9$  and a mean chlorpromazine equivalent dose of 749±545 completed. 14 completed doubleblind, while 19 additional subjects received open label D-serine. There were no significant between design (open label vs. double-blind D-serine) differences across ERP, and results were combined across design. Comparisons between the D-serine and placebo groups found significant, moderate/large effect size difference in MMN to frequency deviants and a non-significant, but moderate effect size improvement in Visual P1. For cognitive outcomes, across all subjects, a moderate effect size, significant drug effect was seen for the MCCB composite and Visual learning domains. D-serine also induced a significant, moderate effect-size reduction in PANSS total symptoms. While no significant drug effect was seen across any individual factors, moderate effect size differences were seen for the negative and depressive factors that favored D-serine vs. placebo. **NMDA+cognitive remediation** ERP analysis is ongoing. 13 patients have completed, and after controlling for baseline pitch processing, significant drug by order effect was seen. A trend towards significance was seen for a drug effect for within visit improvement (p=0.063), primarily in subjects receiving d-serineon day 1. NMDA-Bipolar Enrollment is ongoing. 3 patients have received open label ketamine followed by NMDAR antagonist dose (1000 mg/ day) DCS. All three patients remitted with ketamine no significant side effects. As of this writing, remission was maintained in all subjects (3 to 6 weeks), with no significant treatment emergent side effects.

**Discussion:** The development of treatments targeted at the glutamatergic system remains novel. These findings represent the first double-blind data with 60 mg/kg D-serine in schizophrenia, and the first data on DCS in bipolar depression. No significant treatment-related side effects were observed, supporting viability of the NMDAR treatment approach in schizophrenia and affective illnesses.

## Oxytocin and Facial Expressivity in Patients with Schizophrenia and Healthy Participants

Tuesday, Poster #233

<u>Josh Woolley</u>, Chris Fussell, Wanda Lai, Olivia Lam, Brandon Chuang, Bruno Biagianti, Dan Fulford, Daniel H. Mathalon, Sophia Vinogradov UCSF

**Background:** Restricted expression of affect, including both reduced frequency and intensity of facial emotional expression, is a common negative symptom of schizophrenia that is correlated with worse functional outcomes and quality of life. Furthermore, there are currently no available treatments for these deficits. The neuropeptide oxytocin (OT) has multiple prosocial effects when administered intranasally in humans and offers a potential remedy for these expressivity deficits. OT has been implicated in bonding and has shown promise in enhancing social cognition in schizophrenia. However, the effects of oxytocin administration on facial expressivity have not been investigated in any healthy or patient population. Therefore, we investigated the effects of oxytocin on emotional expression in patients with schizophrenia and age-matched healthy controls while they observed emotionally provocative photos.

**Methods:** Twenty-five male individuals with SCID-confirmed schizophrenia and twenty-seven male, age-matched, healthy participants participated in the study. Testing was performed in a randomized, double-blind, cross-over, placebo-controlled, within-subject design, with the two testing days separated by one week. On each test day, 40 IU of oxytocin or placebo (PCB) was self-administered intranasally. Participants were video recorded while they performed a facial trustworthiness assessment task. During this task, participants were shown 49 faces, and 49 positive (e.g., sports), 49 negative (e.g., snakes), and 49 neutral (e.g., household objects) affective photos from the International Affective Picture System (IAPS). Positive, neutral, and negative photos were chosen from the IAPS based on published ratings of arousal and valence. Effectiveness of the photos to produce pleasant and unpleasant feelings in patients with schizophrenia was validated in a previous study. Participants' facial expressions were coded from the videos independently by two raters, who were blind to condition, using

## Oxytocin and Facial Expressivity in Patients with Schizophrenia and Healthy Participants

Tuesday, Poster #233 (continued)

#### Josh Woolley

the Facial Expression Coding System (FACES). FACES is a behavioral coding system validated for use in patients with schizophrenia based on a dimensional model of emotion, in which each expression is coded for valence (positive/ negative) and intensity (weak/strong). FACES ratings converge with ratings made using Ekman's Facial Action Coding System and with data from facial muscle activity, psychophysiology, and subjective report. Inter-rater agreement was excellent (correlation coefficients: 0.94 to 0.96). Given the preponderance of zeroes in the data (particularly for the schizophrenia group; i.e., lack of facial affect), we conducted non-parametric tests where possible.

Results: Healthy controls (HC) and individuals with schizophrenia (SZ) were well matched on age (Mean (SD) SZ: 43.2 (11), HC: 42 (13.7) p = 0.5). Mann-Whitney U tests revealed that, on the PCB day, individuals with SZ showed significantly lower number (SZ: 1.1 (2.4) vs. HC: 5.8 (7.6)) and intensity of facial expressions (SZ: 0.5 (0.9) vs. HC: 1.0 (0.7)) than HCs (all p's < 0.01) consistent with previous studies. To test the effects of intranasal OT administration on facial expressivity we performed a repeated-measures ANOVA with one within-subject factor, Drug (OT and PCB), and one between-subject factor, Group(SZ and HC), for number and intensity of facial expressions. For number of expressions, we found a significant Drug effect (SZ: PCB: 1.1 (2.4) vs. OT: 2.7 (6.5); HC: PCB: 5.8 (7.6) vs. OT: 8.4 (9.8); F(1) = 6.6, p = .01) but no significant Drug X Group interaction (F(1) = 0.4, p = 0.5). Looking separately at positive and negative expressions revealed a significant Drug effect for negative (SZ: PCB: 0.4 (1.4) vs. OT: 1.7 (4.9); HC: PCB: 3.0 (3.9) vs. OT 4.6 (5.6); (*F*(1) = 6.7, *p* = 0.01) but not positive (SZ: PCB: 0.7 (1.6) vs. OT: 1.0 (2.6); HC: PCB: 2.8 (5.1) vs. OT: 3.8 (7.9); (F(1) = 1.4, p = 0.2) expressions. We found no significant Drug or Drug X Group effects for intensity of facial affect (p's > 0.05). Looking separately by group, related samples Wilcoxon Signed Rank tests revealed that OT increased the total number of facial expressions significantly in SZ (p = 0.01); and

### Oxytocin and Facial Expressivity in Patients with Schizophrenia and Healthy Participants

Tuesday, Poster #233 (continued)

#### Josh Woolley

non-significantly in HC (p = 0.1) but had no effect on the intensity of facial expressions in either group.

**Discussion:** Our results suggest that administration of a single dose of oxytocin increases facial expressivity in SZ and HC during viewing of emotionally provocative photos. While OT appeared to selectively increase negative expressions this was likely due to our stimuli being more effective at inducing negative expressions. The mechanism of OT's effect on facial expressivity is unknown. OT may increase facial expressivity by increasing participants' psychological or physiological response to the emotional cues. Alternatively, facial expressivity is modulated by parasympathetic tone and OT is known to affect parasympathetic tone in humans and rodents. Therefore, OT administration may increase facial expressivity by directly altering parasympathetic regulation to facial musculature without affecting other responses to the emotional cues. Further research is necessary to explore these various hypotheses. In sum, the present study provides support for using OT as a pharmacological agent to remediate the facial expressivity deficits in SZ. Larger studies focused on patients with schizophrenia who have significant baseline negative symptoms are needed to confirm and extend our findings.

# **Poverty and the Past: The Relation Between Hippocampus Function and Memory Performance is Linked to Childhood Poverty**

Tuesday, Poster #193

Elizabeth R. Duval, Sarah N. Garfinkel, Chandra S. Sripada, James E. Swain, Gary W. Evans, Israel Liberzon University of Michigan Health System

Background: Childhood poverty is a risk factor for poorer cognitive performance both among children and possibly in adulthood, although most of the adult studies rely on retrospective estimates of childhood SES. While connections between poverty and cognitive deficits have been accumulating, the underlying neural mechanisms are undetermined. In order to investigate the neurobiological link between childhood poverty and memory deficits, we examined neural activity and working memory in a prospective design among young adults with and without childhood history of poverty. We predicted that memory recall would differ between the two groups, and that these differences would be related to differences in hippocampal activation during encoding.

Methods: Fifty four right handed healthy adults between the ages of 20 and 27 were divided into two groups based on family income to need ratio at age nine. Twenty-eight came from middle income families, and 26 were from households falling below the poverty line. Within the context of a larger study, participants underwent fMRI scanning while encoding line drawings of common objects and animals, followed by a memory recall task. Signal detection (d-prime) was the measure of performance. D-prime was entered as a regressor into fMRI analyses, to examine brain activations during encoding that predicted memory recall performance. The effects of childhood poverty were also examined with respect to memory related activations.

**Results:** Adults who grew up in middle income families performed significantly better than the poverty group (t(52) = 2.21, p < 0.05). A d-prime regressor in fMRI analysis demonstrated a significant positive relationship between activation in left hippocampal regions during encoding and memory recall performance (p < .005, >10 contiguous voxels). This relationship remained even after controlling

# **Poverty and the Past: The Relation Between Hippocampus Function and Memory Performance is Linked to Childhood Poverty**

Tuesday, Poster #193 (continued)

Elizabeth R. Duval

for current income to need ratios. The relationship between left hippocampal activation during encoding was significant in the middle income group (Pearson's r = .48, p < .01) but not in the poverty group (Pearson's r = .29, p > .05). **Discussion:** Our prospective results confirm previous retrospective studies that childhood poverty is associated with poorer memory performance during adulthood. Our findings indicate left hippocampal activation during encoding is related to performance on a subsequent recall task. More specifically, the degree of left hippocampal activation during encoding was associated with better memory recall, but these relationships were demonstrated in the middle income group only. Future studies should continue to investigate mediators and moderators of these relations, including chronic stress, parenting, and other factors related to poverty.

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## **Rare Genetic Variants in VMAT1 (SLC18A1) Are Functional in** Vitro and Associated with Bipolar Disorder

Tuesday, Poster #191

<u>Falk W. Lohoff</u>, Rachel Hodge, Sneha Narasimhan, Glenn Doyle University of Pennsylvania

**Background:** The gene encoding the vesicular monoamine transporter 1 (VMAT1) has recently emerged as a candidate gene for bipolar disorder (BPD), schizophrenia and emotional behavior. We have shown that the common amino acid substitution polymorphism Thr136Ile leads to increased monoamine transport *in vitro* and affects negative emotion processing *in vivo*. In this study we conducted deep sequencing of patients with BPD in order to detect rare VMAT1 variants and to determine their function *in vitro*.

**Methods:** Sanger sequencing of all VMAT1 exons was carried out in 196 BPD individuals and 196 Caucasian controls. Novel rare variants that are likely to change protein function were tested for functional relevance using monoamine reuptake assays in CV-1 cells. Missense SNPs that were functional *in vitro* were then genotyped in a large cohort of BPD (n=4023) and normal controls (n=3305) of European descent from the NIMH Genetic Initiative using standard ABI TaqMan genotyping protocols.

**Results:** Sequencing of BPD patients identified several novel and rare variants. Comparison of sequencing results of rare variants in BPD individuals with normal controls from the 1000 Genome project shows that the global burden of rare variants was increased in the BPD group. Interestingly, several novel variants were only detected in the BPD group but were absent in the controls. Monoamine uptake *in vitro* was carried out for Gln10Arg, Phe84Ser, Ala101Pro, Arg138Leu and Leu392Val. Phe84Ser robustly increased monoamine uptake in particular for DA (p<0.001) and the three variants, Ala101Pro, Arg138Leu and Leu392Val decreased uptake, with Arg138Leu showing the largest effect for DA (P<0.001), although similar results were also obtained for 5-HT and NE. Because of the robust functional effects of Phe84Ser and Arg138Leu, we genotyped these rare variants in a large sample of BPD cases and controls. The Ser84 allele was absent in controls but present in seven BPD individuals, including one homozygote

## **Rare Genetic Variants in VMAT1 (SLC18A1) Are Functional in** Vitro and Associated with Bipolar Disorder

Tuesday, Poster #191 (continued)

#### Falk W. Lohoff

and six heterozygotes (Fisher exact test, P=0.009). The Leu138 frequency did not differ statistically between cases and controls. Haplotype analysis of the individuals with the rare variant Phe84Ser showed that all subjects had almost exclusively the same halplotype Thr–Ser–Thr, indicative of a common origin and founder population effect.

**Discussion:** Sequencing detected several rare and novel missense variants in BPD patients. *In vitro* results show that rare variants lead to "hyper or hypo" transport of monoamines. Association analyses of the rare variants Phe84Ser and Arg138Leu show that the Ser84 allele was only present in BPD but not controls. Given that the common Thr136Ile was previously shown to increased monoamine transport and has an effect on interindividual responses to medial PFC activation of negative words and threat-related amygdala reactivity, the rare Phe84Ser variant may have similar effect on these brain circuits and may contribute to the pathophysiology of BPD. Future studies are needed to comprehensively investigate common and rare SNP-dosage effects on transporter function in vivo and risk for BPD.

## **Real-time Functional MRI Feedback, Compared to Sham, Reduces Cue-Induced Nicotine Craving in Smokers: Results from the First Clinical Trial**

Tuesday, Poster #216

<u>Colleen A. Hanlon</u>, Karen Hartwell, Jeffrey J. Borckardt, James J. Prisciandaro, Melanie Canterberry, Xingbao Li, Max Owens, Todd LeMatty, Michael Saladin, Megan Moran-Santa Maria, Mark S. George, Kathleen T. Brady Hanlon Lab

**Background:** Realtime functional MRI feedback (rtfMRI) is an emerging and innovative technique which allows an individual to receive ongoing feedback about their own neural activity while they perform a given task. Here we present data from the first single-blind, sham controlled clinical trial for rtfMRI as a means of lowering cue-induced craving among smokers.

**Methods:** Forty nicotine dependent smokers, who stated that they were motivated to quit, were enrolled in the rtfMRI protocol which consisted of 3 rtfMRI sessions (1 hour duration, 1 week apart), and 2 follow up visits (1 week, 1 month). Patients were randomized to either the real or sham feedback group. At each visit, a patient-tailored feedback region (craving ROI) was established through a "crave" run in which the participants were instructed to crave when viewing smoking related cues. This ROI, in the region of the anterior cingulate or medial prefrontal cortex, was then 'fed back' to the individuals on 3 subsequent "reduce" craving runs. During the reduce craving scans they were exposed to similar smoking and neutral cues while receiving visual feedback (a thermometer) of BOLD signal activity from the ROI. They were instructed to reduce their craving and the activity in the ROI. Smoking-cue reactivity was measured through psychophysiologic assessments and self-reported metrics before, during, and after each fMRI visit. The primary endpoint was a change in smoking cue-reactivity as measured by heart rate, skin conductance, and self-reported craving metrics.

**Results:** Individuals were unable to reliably identify if they were in the real or sham group, confirming that the integrity of the blind. The real and sham group did not differ in demographic or drug use variables (e.g. age, gender, smoking history, baseline carbon monoxide, FTND score). There was a significant effect

# Real-time Functional MRI Feedback, Compared to Sham, Reduces Cue-Induced Nicotine Craving in Smokers: Results from the First Clinical Trial

Tuesday, Poster #216 (continued)

#### Colleen A. Hanlon

of group for both <u>psychophysiologic parameters</u> (heart rate: F=14.13, p=0.0002; skin conductance:F=9.67, p=0.0019), with the "real feedback" group having a lower physiologic response to cues. There was also a prominent difference among the <u>self-reported craving</u> metrics (Questionnaire of Smoking Urges Factor 1: F=4.52, p=0.041, peak craving: F=4.00, p=0.053) with the "real feedback" group having a lower urge to smoke or peak craving than the sham group. Finally, there was a significant main effect of <u>percent BOLD signal change</u> in the craving ROI. That is, the real feedback group over time had a lower BOLD response in this region than did the sham feedback group.

**Discussion:** These data demonstrate that smokers who are motivated to quit can modulate their cue-induced craving and regional brain activity by using three sessions of realtime feedback training from a patient-tailored ROI. Interestingly, these effects translate into a reduction of psychophysiologic arousal by the cues an hour after the scan as well as a lower self-reported craving during the scans. Further work is needed to determine if these exciting findings can be translated into some form of therapy for treatment seeking smokers, or those with other addictions.

### Substrate-selective COX-2 Inhibition Decreases Anxiety via Endocannabinoid Activation

Tuesday, Poster #232

Daniel Hermanson, Nolan Hartley, Joyonna Gamble-George, Lawrence Marnett, <u>Sachin Patel</u> Vanderbilt University School of Medicine

**Background:** Augmentation of endogenous cannabinoid (eCB) signaling represents an emerging approach to the treatment of affective disorders. Cyclooxygenase-2 (COX-2) oxygenates arachidonic acid to form prostaglandins, but also inactivates eCBs *in vitro*. However, the viability of COX-2 as a therapeutic target for *in vivo* eCB augmentation has not been explored.

**Methods:** Here we utilized medicinal chemistry and *in vivo* analytical and behavioral pharmacological approaches to demonstrate a key role for COX-2 in the regulation of endocannabinoid (eCB) levels *in vivo*. A novel pharmacological strategy involving "substrate-selective" inhibition of COX-2 was developed used to augment eCB signaling without affecting related non-eCB lipids or prostaglandin synthesis.

**Results:** Administration of the substrate-selective inhibitor LM-4131 increased brain and peripheral anandamide levels without affecting prostaglandin levels or levels or other related non-endocannabinoid lipids. Behaviorally, LM-4131 reduced anxiety-like behaviors in a variety of pre-clinical models. These effects were mediated via activation of CB1 type cannabinoid receptors. LM-4131 also reduced stress-indcued anxiety states in animals.

**Discussion:** These data indicate that substrate-selective COX-2 inhibition represents a viable approach to enhance eCB signaling for the treatment of affective disorders. These data also highlight a key role for COX-2 in the regulation of central eCB signaling and validate COX-2 as a new molecular target for psychiatric drug discovery.

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

## Emerging Role of the Primary Cilium in Neuropsychiatric Disorders

Chair: Bernard Lerer

- 3:00 p.m. The Role of AHI1 in Regulating Primary Cilia Signaling *Russell J. Ferland*
- 3:25 p.m. Functional Significance of Primary Cilia to GPCR Signaling and Relationship to Neuropsychiatric Disease *Mark Von Zastrow*
- 3:50 p.m. Abnormal Response to Stress in Heterozygous AHI1 Knockout Mice: A Consequence of Primary Ciliary Dysfunction? Bernard Lerer

4:15 p.m. – 5:30 p.m. Mini-Panel Diplomat Ballroom 1 & 2

# Adolescent Brain Development and Affective Disorders: The Role of Reward and Threat Circuitry

Chair: Erika E. Forbes

| 4:15 p.m. | Adolescent VTA Neurons Exhibit Latent Neuronal Correlate of<br>Reward Opportunity<br><i>Bita Moghaddam</i>   |
|-----------|--|
| 4:40 p.m. | Adolescents' Neural Response to Personally Relevant Social<br>Reward Is Associated with Severity of Mania and Depression<br><i>Erika E. Forbes</i> |
| 5:05 p.m. | Neural Mechanisms of Frustration in Chronically Irritable Youth<br>Ellen Leibenluft  |

#### ACNP 52nd Annual Meeting • Final Program

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

# An Update from the Clinic on mGluR2/3 Approaches for Treating Schizophrenia – Understanding Human Circuit Engagement through to Recent Clinical Trials

Chair: Nicholas Brandon

- 3:00 p.m. The Development of Pomaglumetad Methionil as an Innovative Glutamate-based Pharmacotherapy for Schizophrenia *Bruce J. Kinon*
- 3:30 p.m. Discovery and Early Clinical Development of Novel mGlu2 Receptor Pams *Hilde Lavreysen*
- 4:00 p.m. AZD8529 An mglur2 Positive Allosteric Modulator for the Treatment of Schizophrenia *Alan Cross*
- 4:30 p.m. Efficacy of an mGluR2 Agonist (LY354740) and an mGluR2 Positive Allosteric Modulator (AZD8529) in Attenuating Ketamine Effects in Humans John H. Krystal

5:00 p.m. Discussant: Daniel R. Weinberger

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3:00 p.m. – 5:30 p.m. Panel Atlantic Ballroom 3

#### Anxiety and the Striatum, an Unusual Suspect

Chair: Monique Ernst

- 3:00 p.m. Neural Response in Striatum Varies by Reward Magnitude, Decision Making, and Anxiety Diagnosis in Adolescents *Amanda E. Guyer*
- 3:30 p.m. Cortico-amygdala Pathways form Hierarchical Networks that Predict Output to the Striatum Julie L. Fudge
- 4:00 p.m. The Impact of Induced Anxiety on Ventral Striatal Response to Aversive and Appetitive Prediction Error Signals *Oliver J. Robinson*
- 4:30 p.m. Endocannabinoids in the Dopaminergic Control of Punishment and its Avoidance Joseph Cheer
- 5:00 p.m. Discussant: *Mauricio Delgado*

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

#### **Biotypes of Psychosis**

Chair: Carol A. Tamminga Co-Chair: Godfrey D. Pearlson

- 3:00 p.m. Phenotypic Characterization of the Schizophrenia- bipolar Disorder Continuum Matcheri Keshavan
- 3:30 p.m. Identification of Distinct Psychosis Biotypes with Multivariate Taxometric Analyses of Neuro-pathologically Relevant Biomarkers Brett A. Clementz
- 4:00 p.m. Validating Psychosis Biotypes *Carol A. Tamminga*
- 4:30 p.m. Multivariate Fusion Methods Identify Gene Components Associated with Heritable Resting State fMRI Abnomalities in BSNIP Probands and Relatives *Godfrey D. Pearlson*
- 5:00 p.m. Discussant: Steven E. Hyman

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

## Pathophysiology and Treatment of Obesity and Glucose Dysregulation in Schizophrenia

Chair: Lars Fredrik Jarskog Co-Chair: Scott Stroup

- 3:00 p.m. Effect of Metformin on Weight in Patients with Schizophrenia with Impaired Fasting Glucose Scott Stroup
- 3:30 p.m. Dopamine, Clean Up Your "AKT"! Restoring Insulin Signaling in Brain Aurelio Galli
- 4:00 p.m. Dysglycemic Signals in Antipsychotic-Treated Children and Adolescents with Schizophrenia-Spectrum Disorders: Trajectories, and Moderating and Mediating Factors *Christoph Correll*
- 4:30 p.m. No Effect of Adjunctive, Repeated Dose Intranasal Insulin Treatment on Body Metabolism in Patients with Schizophrenia *Xiaoduo Fan*
- 5:00 p.m. Discussant: Robert W. Buchanan

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

#### **Posttraumatic Stress Disorder: From Markers to Mechanisms**

Chair: Murray B. Stein

- 3:00 p.m. Identification of Novel Gene Regulatory Networks Associated with PTSD: Evidence from Whole Genome Studies Examining DNA Methylation Douglas E. Williamson
- 3:30 p.m. Allele Specific Epigenetic Modifications A Molecular Mediator of Gene-environment Interactions in Stress Related Psychiatric Disorders? *Torsten Klengel*
- 4:00 p.m. Opioid Receptor-Like 1 (OPRL1) is Involved in Amygdaladependent Fear in Mice and Humans with PTSD *Kerry J. Ressler*
- 4:30 p.m. Contextual Processing Deficits in PTSD: Translational Studies Israel Liberzon
- 5:00 p.m. Discussant: Charles R. Marmar

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

# **Treating Addiction: Should We Aim High or Low?**

Chair: Marina E. Wolf

| 3:00 p.m. | Rapid LTP in Accumbens Is a Common Feature of Relapse to<br>Multiple Classes of Addictive Drug<br><i>Peter W. Kalivas</i>                   |  |
|-----------|---|--|
| 3:30 p.m. | Synaptic Depression via Positive Allosteric Modulation of<br>mGluR1 Suppresses Cue-induced Cocaine Craving<br>Marina E. Wolf                |  |
| 4:00 p.m. | Silent Synapse-based Circuitry Reorganization in Cocaine<br>Craving<br>Yan Dong   |  |
| 4:30 p.m. | .m. Withdrawal from Acute Amphetamine Potently Down-Regula<br>VTA Dopamine Neuron Activity: Reversal by Ketamine<br><i>Anthony A. Grace</i> |  |
| 5:00 p.m. | Discussant: Anissa Abi-Dargham  |  |

| A     | CNP 52nd Annual Meeting | Final Program |
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| Notes |                         |               |
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8:30 a.m. – 9:45 a.m. Mini-Panel Diplomat Ballroom 1-2

### Are the Putative Therapeutic Effects of Kappa-opioid Antagonists Explained by Anti-stress Actions?

Chair: William Carlezon

- 8:30 a.m. Disruption of Kappa-opioid Receptor Actions Reduces Stress Effects on Cognitive Function and Anxiety-like Behavior Ashlee Van't Veer
- 8:55 a.m. Dynorphin-kappa Systems in Compulsive-like Responding with Extended Access to Elicit Drugs *George F. Koob*
- 9:20 a.m. The Dissociable Effects of Kappa-opioid Receptor Activation on Intolerance to Delay and Response Inhibition Brendan Walker

9:45 a.m. – 11:00 a.m. Mini-Panel Diplomat Ballroom 1-2

#### Developing Imaging Biomarkers for Treatment Development: Beyond CNTRICS, CNTRaCs and NEWMEDS

Chair: Cameron S. Carter

| 9:45 a.m.  | New Neuroscience Based Cognitive Paradigms for Biomarker<br>Research in Schizophrenia                                 |   |
|------------|---|---|
|            | Deanna M. Barch   |   |
| 10:10 a.m. | Developing Imaging Biomarkers for Treatment Development:<br>Beyond CNTRICS, CNTRaCs and NEWMEDS<br>Angus W. MacDonald | M |
| 10:35 a.m. | Imaging Biomarkers for Psychiatric Disorders: The NEWMEDS<br>Experience<br>Andreas Meyer-Lindenberg                   |   |

8:30 a.m. – 11:00 a.m. Panel Regency Ballroom 3

#### Alterations of the Glutamate Cycle in Severe Mental Illness

#### Chair: Adrienne C. Lahti Co-Chair: Robert E. McCullumsmith

| 8:30 a.m.  | Glutamatergic Abnormalities in Medicated and Unmedicated<br>Patients with Schizophrenia<br>Adrienne C. Lahti   |
|------------|--|
| 9:00 a.m.  | Abnormalities of Glutamate Transporter Expression in<br>Schizophrenia: Evidence for Increased Glutamate Reuptake and<br>Altered Subcellular Partitioning of EAAT2 Interacting Proteins<br><i>Robert E. McCullumsmith</i> |
| 9:30 a.m.  | Psychosis and Cognition Related to Different Brain Glutamate<br>Pools in Schizophrenia<br>Juan Bustillo  |
| 10:00 a.m. | Functional Implications of Altered In Vivo Glutamate and GABA<br>Systems in Schizophrenia<br>Laura M. Rowland  |
| 10:30 a.m. | Discussant: Kelvin O. Lim  |

8:30 a.m. – 11:00 a.m. Panel Atlantic Ballroom 1

# **Epigenetic Mechanisms in Neuropsychiatric Disorders**

#### Chair: Paul Kenny Co-Chair: Claes Wahlestedt

| 8:30 a.m.  | MicroRNAs and Drug Addiction<br>Paul Kenny  |
|------------|---|
| 9:00 a.m.  | Global Transcriptome Analysis of Human Cerebrospinal Fluid<br>Claes Wahlestedt  |
| 9:30 a.m.  | Insights into the Roles of the Methyl-DNA Binding Protein<br>MeCP2 in Addictive-like Behaviors<br>Anne E. West                    |
| 10:00 a.m. | MicroRNA 135 Is Essential for Chronic Stress Resiliency,<br>Antidepressant Efficacy and Intact Serotonergic Activity<br>Alon Chen |
| 10:30 a.m. | Discussant: Eric Nestler  |

8:30 a.m. – 11:00 a.m. Panel Atlantic Ballroom 3

#### α4β2-Nicotinic Acetylcholine Receptors in Schizophrenia: Implications for Smoking Cessation and Therapeutics

Chair: A. Eden Evins Co-Chair: Irina Esterlis

- 8:30 a.m. In Vivo Evidence for β2\*-nAChR Upregulation in Smokers as Compared to Nonsmokers with Schizophrenia *Irina Esterlis*
- 9:00 a.m. Nicotinic CHRNA4 Exon 5 Genotype Predicts Clinical Outcome in Schizophrenia and Neuroleptic Drug Treatment-response *Georg Winterer*
- 9:30 a.m. Examining the α4β2 Nicotinic Partial Agnoist Varenicline on the Tobacco Abstinence Syndrome in Schizophrenia Versus Control Smokers *Victoria C. Wing*
- 10:00 a.m. Extended Duration Pharmacotherapy with Varenicline Prevents Relapse to Smoking in Adult Smokers with Schizophrenia

A. Eden Evins

10:30 a.m. Discussant: Tony P. George

8:30 a.m. – 11:00 a.m. Panel Atlantic Ballroom 2

### Manipulating BDNF-TrkB Signaling in Brain Disorders: Complex Regulation and Cellular & Systems Level Interactions as Novel Substrates for Translational Medicine

Chair: Keri Martinowich Co-Chair: Francis Lee

| 8:30 a.m.  | Differential Contribution of Individual BDNF Splice Variants to<br>Brain and Behavioral Functions<br><i>Keri Martinowich</i> |
|------------|--|
| 9:00 a.m.  | Role of Slitrk5 in Regulating BDNF Dependent Signaling<br>Francis Lee  |
| 9:30 a.m.  | Convergence of BDNF and Glucocorticoid Receptor Signaling<br>Moses V. Chao   |
| 10:00 a.m. | Synaptic Repair: Translating BDNF Biology into New Medicines<br>for Psychiatric Diseases<br><i>Bai Lu</i>                    |
| 10:30 a.m. | Discussant: Ronald S. Duman  |

8:30 a.m. – 11:00 a.m. Panel Regency Ballroom 2

#### New Directions for Optogenetics: Investigating Plasticity Mechanisms Underlying Psychiatric Disorders

Chair: Helen Blair Simpson Co-Chair: Susanne E. Ahmari

| 8:30 a.m. | Brief Repeated Cortico-striatal Stimulation Leads to Persistent<br>OCD-relevant Behaviors<br>Susanne E. Ahmari |
|-----------|--|
| 9:00 a.m. | Cortical Control of Brainstem Neuromodulatory Systems in<br>Motivated Behavior<br>Melissa R. Warden            |

#### 9:30 a.m. Different Patterns of Stimulation in Projections from VTA to PFC Exert Distinct Effects on Behavioral Flexibility *Vikaas S. Sohal*

- 10:00 a.m. Molecular and Circuit Basis of Impaired Hippocampal-prefrontal Synchrony in a Mouse Model of Schizophrenia Predisposition Joshua A. Gordon
- 10:30 a.m. Discussant: Karl Deisseroth

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8:30 a.m. – 11:00 a.m. Panel Regency Ballroom 1

### The Ventromedial Prefrontal Cortex in Conditioning and Extinction in Chronically Relapsing Disorders

Chair: Rita Goldstein Co-Chair: Karen K. Szumlinski

| 8:30 a.m.  | Factors that Impact the Functional Reactivity of the Fear<br>Extinction Network Across Disorders<br>Mohammed R. Milad  |
|------------|--|
| 9:00 a.m.  | Retention of Extinction Learning for Monetary Reward in Cocaine Addiction: Role of the Amygdala and VMPFC <i>Rita Goldstein</i>  |
| 9:30 a.m.  | Deficits in Ventromedial Prefrontal Cortex Group1 Metabotropic<br>Glutamate Receptors Underpin Cognitive Dysfunction during<br>Protracted Cocaine Withdrawal<br><i>Karen K. Szumlinski</i> |
| 10:00 a.m. | Role of Ventral Medial Prefrontal Cortex (Mpfc) and<br>Its Projections to Accumbens Shell on Context-induced<br>Reinstatement of Heroin Seeking in Rats<br>Jennifer M. Bossert             |
| 10:30 a.m. | Discussant: Gregory J. Quirk   |

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

#### Human Brain Evolution and Comparative Epigenomics

Chair: Schahram Akbarian

- 3:00 p.m. Decoding the Molecular Evolution of Cognition *Genevieve Konopka*
- 3:25 p.m. Divergent Whole Genome Methylation Maps of Human and Chimpanzee Brains Reveal Epigenetic Basis of Human Regulatory Evolution and Disease Susceptibility *Soojin V. Yi*
- 3:50 p.m. Neuronal Epigenome Mapping in Human and Non-human Primate Prefrontal Cortex Jogender Singh Tushir

4:15 p.m. – 5:30 p.m. Mini-Panel Diplomat Ballroom 1 & 2

#### Intergenerational Transmission of Trauma – From Animal Models to Humans

Chair: Kerry J. Ressler Co-Chair: Jacek Debiec

| 4:15 p.m. | Behavioral and Neural Mechanisms of the Intergenerational<br>Transmission of Trauma<br>Jacek Debiec        |
|-----------|--|
| 4:40 p.m. | Epigenetic Markers in the GR and FKBP5 Genes in Children of<br>Holocaust Survivors<br><i>Rachel Yehuda</i> |
| 5:05 p.m. | Transgenerational Imprints on Structure and Function in the<br>Mammalian Nervous System                    |

Brian Dias

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

### Legal Damages: New Insights into Chronic Marijuana Effects on Human Brain Structure and Function

Chair: Steven Grant Co-Chair: James M. Bjork

| 3:00 p.m. | Impact of Chronic Marijuana Use on Reward and Control Brain<br>Networks<br><i>Francesca M. Filbey</i>          |
|-----------|--|
| 3:30 p.m. | Effect of Long-term Cannabis Use on Axonal Fiber Connectivity<br>Andrew Zalesky                                |
| 4:00 p.m. | Unmotivated? Signatures of Blunted Dopaminergic<br>Responsiveness in Chronic Marihuana Abuse<br>Nora D. Volkow |
| 4:30 p.m. | Multimodal MR Imaging in Adolescent MJ Users<br>Deborah Yurgelun-Todd  |
| 5:00 p.m. | Discussant: Linda Porrino  |

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

#### **Early Stress and Emotion Dysregulation**

Chair: Christian Schmahl Co-Chair: Larry Siever

- 3:00 p.m. Weakening Fear Memories as a Potential Treatment for Posttraumatic Stress Disorder *Karim Nader*
- 3:30 p.m. Effect of Direct Eye Contact in PTSD Related to Interpersonal Trauma: An fMRI Study of Activation of an Innate Alarm System
  - Ruth Lanius
- 4:00 p.m. Examining the Genetic Underpinnings of the Amygdala Habituation Deficit in Borderline Personality Disorder *M. Mercedes Perez-Rodriguez*
- 4:30 p.m. Influence of Dissociation on Emotional Distraction in Borderline Personality Disorder *Annegret Krause-Utz*
- 5:00 p.m. Discussant: Andreas Meyer-Lindenberg

3:00 p.m. – 5:30 p.m. Panel Atlantic Ballroom 3

# **Glutamate-dopamine Interactions in Nicotine and Cocaine Dependence: Biomarkers and Therapy Opportunities**

Chair: Dean F. Wong Co-Chair: Gerhard Gründer

| 3:00 p.m. | In Vivo Imaging of Human mGluR5 and nAch Receptors with<br>PET: Dynamic Duo for Abuse Studies and Drug Occupancy?<br>Dean F. Wong   |
|-----------|---|
| 3:30 p.m. | Dopamine Activity and Reward Processing in Smokers Before<br>and After Smoking Cessation: Combined [18F]FDOPA/fMRI<br>Studies<br>Gerhard Gründer                                  |
| 4:00 p.m. | Reduced mGluR5 Receptor Binding in Smokers and Ex-smokers<br>Determined by [11C]ABP688 Positron Emission Tomography:<br>Clinical and Scientific Relevance<br><i>Gregor Hasler</i> |
| 4:30 p.m. | Understanding Glutamate, Acetylcholine and Dopamine<br>Interactions in Nicotine Dependence Using Animal Models<br>Manoranjan S. D'Souza   |
| 5:00 p.m. | Discussant: Athina Markou   |

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

### Multidimensional Data Integration and Causality: A Systems Approach for Unraveling the Molecular Architecture of Mental Disorders

Chair: Thomas Lehner

| 3:00 p.m. | Elucidating the Complexity of Psychiatric Disorders via the<br>Integration of High-dimensional, Multiscale Data<br>Eric E. Schadt |
|-----------|---|
| 3:30 p.m. | Cis and Trans Data Integration to Find Mechanisms Causing<br>Psychiatric Disorders<br>Edwin van den Oord                          |
| 4:00 p.m. | The ENIGMA Consortium: Meta-analyzing Neuroimaging and Genetic Data from 125 Institutions <i>Paul Thompson</i>                    |
| 4:30 p.m. | Computational Analysis of Complex Human Disorders<br>Andrey Rzhetsky  |
| 5:00 p.m  | Discussant: Steven E. Hyman   |

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

# Neurobiological Regulation of Palatable Food Binging and Seeking

Chair: Jacqueline F. McGinty

| 3:00 p.m. | Extended Amygdala-hypothalamic Inhibitory Circuits Regulate<br>Feeding<br>Garret D. Stuber  |
|-----------|---|
| 3:30 p.m. | Cholinergic Control of Food Intake: Mechanisms Highjacked by<br>Nicotine<br>Marina Picciotto  |
| 4:00 p.m. | Serotonin Control in the Proclivity for High Impulsive Action<br>and Binge Eating<br><i>Kathryn Cunningham</i>                          |
| 4:30 p.m. | Neural Correlates of Craving, Cognitive Control and Reward<br>Processing in Obesity and Binge-eating Disorder<br><i>Marc N. Potenza</i> |
| 5:00 p.m. | Discussant: Jacqueline F. McGinty   |

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

# Public-private Repositioning Partnerships: A New Path to Achieve Target Validation and Proof of Concept for Novel CNS Indications

Chair: Linda S. Brady Co-Chair: Jeffrey S. Nye

| 3:00 p.m. | Drug Repositioning Through Open Innovation - An Industry<br>Perspective<br>Donald Frail                                   |
|-----------|---|
| 3:30 p.m. | The MRC/AstraZeneca Mechanisms of Disease Compound<br>Sharing Initiative<br><i>Christopher Watkins</i>                    |
| 4:00 p.m. | NCATS/NIH-Industry Pilot Program on Drug Repositioning<br>Christine Colvis  |
| 4:30 p.m. | Open Innovation and Mobile Health Technology to Improve and<br>Accelerate Clinical Development<br><i>Tomasz Sablinski</i> |
| 5:00 p.m. | Discussant: Jeffrev J. Nve  |

|       | ACNP 52nd Annual Meeting • Final Program |
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8:00 a.m. – 10:30 a.m. Panel Atlantic Ballroom 1

# Brain on Fire: Inflammation in Neurological and Psychiatric Illness

Chair: Scott Russo Co-Chair: Danielle L. Graham

| 8:00 a.m.  | Pruning Developing CNS Synapses: An Active Role for Glia and<br>Immune Molecules<br>Beth Stevens                                  |
|------------|---|
| 8:30 a.m.  | Monitoring Neuroinflammation and Demyelination Using<br>Magnetic Resonance Imaging in a Preclinical Setting<br>Danielle L. Graham |
| 9:00 a.m.  | Role of the Peripheral Immune System in Stress-induced<br>Depressive Behavior<br><i>Georgia E. Hodes</i>                          |
| 9:30 a.m.  | Testing the Cytokine Hypothesis of Depression: Trials and<br>Tribulations<br>Andrew H. Miller                                     |
| 10:00 a.m. | Discussant: Christopher McDougle  |

8:00 a.m. – 10:30 a.m. Panel Regency Ballroom 2

#### Broadening the Trajectories of Risk: Specific and Non-specific Markers of Risk of Psychopathology

Chair: Kathleen Ries Merikangas

| 8:00 a.m. | Comorbidity of Medical and Psychiatric Disorders in the |
|-----------|---|
|           | Neurodevelopmental Genomics Cohort Study                |
|           | Kathleen Ries Merikangas                                |

- 8:30 a.m. The Study of Developmental Trajectories in Autism Spectrum Disorders; Lessons Learned *Peter Szatmari*
- 9:00 a.m. Clinical, Neurobiological and Circadian Correlates of the Onset and Course of Major Mood Disorders: From Childhood Risk to Adolescent-onset and Persistence into Adulthood *Ian Hickie*
- 9:30 a.m. Brain-behavior Measures in Psychosis Spectrum Youths of the Philadelphia Neurodevelopmental Cohort Raquel E. Gur
- 10:00 a.m. Discussant: Patrick McLzorry

8:00 a.m. – 10:30 a.m. Panel Atlantic Ballroom 3

# **Building a More Predictive Mouse: Humanized Mouse Models for Neuropsychiatric Disorders**

Chair: Mark A. Geyer

| 8:00 a.m.  | The Tryptophan Hydroxylase Arg439His Knockin (Tph2KI)<br>Mouse: A Naturalistic Model of 5-HT Deficiency<br>Jacob Jacobsen             |
|------------|---|
| 8:30 a.m.  | Modeling the Contributions of Dopamine to ADHD via a Novel,<br>Knock-in Mouse Model<br><i>Maureen K. Hahn</i>                         |
| 9:00 a.m.  | Development and Characterization of Mice Humanzied for the<br>COMTval158met Polymorphism<br>Victoria Risbrough                        |
| 9:30 a.m.  | DISC1-Boymaw Fusion Gene May Contribute to Major<br>Psychiatric Disorders in the Scottish Schizophrenia Family<br><i>Xianjin Zhou</i> |
| 10:00 a.m. | Discussant: John Talpos   |

8:00 a.m. – 10:30 a.m. Panel Atlantic Ballroom 2

# Melatonin and Its Receptors: Important Players in Major Depressive Disorder

Chair: Pierre Blier

| 8:00 a.m.  | Melatonin-mediated Potentiation of Physical Activity-induced<br>Neurogenesis in the Dentate Gyrus of the C3H/HeN Mouse.<br><i>Margarita L. Dubocovich</i> |
|------------|---|
| 8:30 a.m.  | Interactions between Melatonin and 5-HT Receptors to Enhance<br>Monoaminergic Transmission in the Rat Brain<br><i>Pierre Blier</i>                        |
| 9:00 a.m.  | Impact of Hippocampal Neurogenesis on Cognition and Mood<br>Rene Hen  |
| 9:30 a.m.  | A Pilot, Placebo-controlled Study of Buspirone Plus Melatonin in<br>Major Depressive Disorder<br><i>Maurizio Fava</i>                                     |
| 10:00 a.m. | Discussant: Gabriella Gobbi   |

8:00 a.m. – 10:30 a.m. Panel Diplomat Ballroom 1-2

# Molecular Regulation and Clinical Applications of Phosphodiesterase 4, the Major Enzyme for Degrading cAMP

Chair: Robert Innis Co-Chair: Akira Sawa

| 8:00 a.m.  | PDE4 in Huntington's Disease: Pathology of Cross-seeding of<br>Huntingtin and Amyloidogenic DISC1<br>Koko Ishizuka   |
|------------|--|
| 8:30 a.m.  | Control of Mood by Selective Potentiation of cAMP Signaling in<br>Ventral Striatum<br>James A. Bibb  |
| 9:00 a.m.  | Structural and Pharmacological Studies of PDE4 Subtype<br>Selective Allosteric Inhibitors<br><i>Mark Gurney</i>  |
| 9:30 a.m.  | Binding of 11C-(R)-rolipram to Phosphodiesterase 4 is<br>Downregulated in Major Depressive Disorder and Normalized<br>with Antidepressant Treatment<br><i>Robert Innis</i> |
| 10:00 a.m. | Discussant: Carlos Zarate, Jr.   |

8:00 a.m. – 10:30 a.m. Panel Regency Ballroom 1

# Naltrexone Revisited: New Findings Beyond Mu, Beyond Dopamine and Beyond Addiction

Chair: Robert M. Swift

| 8:00 a.m.  | Naltrexone Effects on GABAergic Neuroactive Steroids:<br>Associations to Subjective Responses and Pharmacogenetics<br><i>Lara Ray</i> |
|------------|---|
| 8:30 a.m.  | Neurocognitive Effects of Naltrexone<br>Charlotte A. Boettiger  |
| 9:00 a.m.  | Naltrexone Pharmacotherapy for Adverse Metabolic Outcomes<br>of Second Generation Antipsychotic Agents<br>Igor Elman                  |
| 9:30 a.m.  | Microglial Activation Alters Reward Circuitry in Chronic Pain<br>States<br><i>Catherine Cahill</i>                                    |
| 10:00 a.m. | Discussant: Stephanie O'Malley  |

8:00 a.m. – 10:30 a.m. Panel Regency Ballroom 3

# Understanding Neurodevelopmental Risk Factors Leading to Anxiety and Depression to Inform Novel Early Interventions in Vulnerable Children

Chair: Ned H. Kalin

- 8:00 a.m. Neurobiology of Trauma and Infant Attachment: Short-term Benefits and Long-term Costs *Regina M. Sullivan*
- 8:30 a.m. Primate Anxious Temperament and Amygdala Metabolism are Environmentally Sensitive and Associated with Amygdalar Gene Expression

Andrew S. Fox

9:00 a.m. Glucocorticoid Receptor Activation Induced Epigenetic Changes and Their Moderation by Genetic Variants as Potential Mediators of Risk and Resilience to Early Trauma-associated Psychiatric Disorders

Elisabeth B. Binder

- 9:30 a.m. The Pervasive and Persistent Neurobiological Consequences of Child Abuse and Neglect; Clinical Implications *Charles B. Nemeroff*
- 10:00 a.m. Discussant: Bruce McEwen

12:00 p.m. – 2:30 p.m. Panel Atlantic Ballroom 3

#### Applying Animal and Human Models of Risk Avoidance and Impulsivity to Understanding Eating Disorders

Chair: Walter Kaye Co-Chair: Barry Setlow

- 12:00 p.m. A Translational Assessment of Reward-based Learning in Adolescents with Bulimia Nervosa Rachel Marsh
- 12:30 p.m. Harm Avoidant Behaviors and Altered Limbic and Executive Neural Function in Anorexia Nervosa *Walter Kaye*
- 1:00 p.m. Striatal Dopamine D2 Receptor Modulation of Risky Decision Making Barry Setlow
- 1:30 p.m. D1- and D2-like Dopamine Receptors, Impulsive Temperament and Corticostriatal Function as Related to Risky Decisionmaking: Multimodal Imaging in Healthy Research Participants *Edythe D. London*
- 2:00 p.m. Discussant: Trevor Robbins

12:00 p.m. – 2:30 p.m. Panel Atlantic Ballroom 2

### Behavioral, Endocrine, and Neural Plasticity Changes Reflecting Stress Associated with Mouse and Monkey Models of Heavy Alcohol Drinking

Chair: Howard C. Becker Co-Chair: Kathleen A. Grant

- 12:00 p.m. Behavioral and Neural Adaptations Linked to Stress Associated with a Mouse Model of Ethanol Dependence and Relapse Drinking *Howard C. Becker*
- 12:30 p.m. Behavioral and Endocrine Adaptations in a Monkey Model of Heavy Alcohol Drinking *Kathleen A. Grant*
- 1:00 p.m. Chronic Ethanol Exposure Increases Output from the Sensorimotor Striatum in Mouse and Monkey Models via Changes in Neuronal Excitability and Synaptic Transmission David Lovinger
- 1:30 p.m. Similar Dopamine System Adaptations in Mouse and Monkey Models of Excessive Alcohol Exposure Sara R. Jones
- 2:00 p.m. Discussant: Antonio Noronha

12:00 p.m. – 2:30 p.m. Panel Regency Ballroom 1

#### Cognition, Biomarkers, and Longitudinal Outcomes in Geriatric Mood Disorders

Chair: Helen Lavretsky Co-Chair: Charles Reynolds

- 12:00 p.m. Longitudnial BDNF Levels in Both an Elderly Cohort and an Inflammatory Cytokine-exposed Cohort: Risk for Cognitive Deficits *Francis E. Lotrich*
- 12:30 p.m. Late-life Depression May Increase Risk of Dementia but Does Not Increase Risk of Developing Mild Cognitive Impairment *Meryl Butters*
- 1:00 p.m. Cognitive Control Network: Motivational Disturbances and Treatment Response of Late Life Depression *George S. Alexopoulos*
- 1:30 p.m. Combination of Methylpehnidate with Citalopram is Superior to Either Drug Alone in Improving Clinical and Cognitive Outcomes in Geriatric Depression *Helen Lavretsky*
- 2:00 p.m. Discussant: Wesley Thompson

12:00 p.m. – 2:30 p.m. Panel Regency Ballroom 2

### Experimental Therapeutics and Drug Development Targeting Inflammation in Developmental Disorders

Chair: Eric Hollander Co-Chair: Carlos A. Pardo

| 12:00 p.m. | Gastrointestinal Symptoms in a Mouse Model of an<br>Environmental Risk Factor for Autism and Schizophrenia<br>Paul H. Patterson  |
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| 12:30 p.m. | Immune System as a Target for Therapeutic Intervention in<br>Neurodevelopmental Disorders: Lessons from the Rett Syndrome<br>Jonathan Kipnis   |
| 1:00 p.m.  | PET Imaging of Microglial Activation in Young Adults with<br>Autism Spectrum Disorder<br><i>Kazuhiko Nakamura</i>  |
| 1:30 p.m.  | Translational Experimental Therapeutics of Inflammation and<br>Fever in Autism Spectrum Disorder : Hot Tubs, Locus Coeruleus<br>Modulation and Helminth Therapy<br><i>Eric Hollander</i> |
| 2:00 p.m.  | Discussant: Carlos Pardo   |

12:00 p.m. – 2:30 p.m. Panel Diplomat Ballroom 1-2

#### Novel Molecules and Mechanisms in Vulnerability and Resilience Throughout Life

Chair: Marcelo A. Wood Co-Chair: Tallie Z. Baram

- 12:00 p.m. miRNA Programming in Neurodevelopment: Epigenetic Targets in a Dynamic Landscape *Tracy Bale*
- 12:30 p.m. Epigenetic Pathways during Early Postnatal Life: How Does a Neuron "Know" to Modulate Its Epigenetic Machinery in Response to Early-life Experience? *Tallie Z. Baram*
- 1:00 p.m. How Neocortical Tet-mediated DNA Hydroxymethylation Regulates Memory *Timothy W. Bredy*
- 1:30 p.m. Neuron-specific Nucleosome Remodeling: A Missing Link in Our Understanding of Epigenetic Mechanisms Underlying Intellectual Disability Disorders *Marcelo A. Wood*
- 2:00 p.m. Discussant: Farah Lubin

12:00 p.m. – 2:30 p.m. Panel Atlantic Ballroom 1

# Strategies for the Development of Novel Therapies for Schizophrenia: From Clinic to Laboratory (And Back Again)

Chair: Joel E. Kleinman

| 12:00 p.m. | Psychiatric GWAS Consortium Triples Schizophrenia GWAS<br>Sample-size to 31,000 Cases and 37,000 Controls<br>Stephan Ripke                           |
|------------|--|
| 12:30 p.m. | Integrating the Genome, Epigenome, and Transcriptome in<br>the Human Brain: Accounting for Biological and Technical<br>Heterogeneity<br>Andrew Jaffe |
| 1:00 p.m.  | Understanding ZNF804A: Allelic Variation, Alternative<br>Transcripts, Brain Development and Schizophrenia<br><i>Thomas M. Hyde</i>                   |
| 1:30 p.m.  | Applying Lessons from DISC1 to Convert Gene Discoveries into<br>Drug Discoveries<br><i>Nicholas Brandon</i>  |
| 2:00 p.m.  | Discussant: Paul Harrison  |

12:00 p.m. – 2:30 p.m. Panel Regency Ballroom 3

# The Insula Salience Network: Alterations in Its Connectivity in Developmental, Anxiety, Mood and Personality Disorders

Chair: Harold W. Koenigsberg

- 12:00 p.m. Insula Conceptualizing Its Architecture, Function and Connectivity, with Applications to Understanding Large-scale Brain Networks in Psychopathology and Autism *Vinod Menon*
- 12:30 p.m. Insula-amygdala Function and Connectivity in Trauma-related Disorders: Relationship to Childhood Maltreatment *Murray B. Stein*
- 1:00 p.m. Borderline Personality Disorder Patients Show Reduced Insulaamygdala Functional Connectivity and Fail to Habituate When Viewing Repeated Negative Emotional Pictures *Harold W. Koenigsberg*
- 1:30 p.m. Elevated Posterior Insula–ventral Striatal Connectivity to Reward in Youth with Bipolar Spectrum Disorders Relative to Youth with Other Behavioral and Emotional Dyregulation Disorders: A Potential Neural Marker of Heightened Reward-related Perceptual Salience in Bipolar Youth *Mary L. Phillips*
- 2:00 p.m. Discussant: *Kevin Ochsner*

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#### Poster Session I – Monday, December 9, 2013



Advocacy Affiliate – Cure Alliance for Mental Illness Research Parity for Mental Illness

Robin Cunningham and Hakon Heimer

M1. 2013 Report of the Membership Advisory Task Force

Linda Carpenter, Lisa Monteggia, Margaret Haney, Katherine Burdick, Jennifer Bartz, Elisabeth Binder, Paul Holtzheimer, Amit Etkin, Erica Forbes, Marlene Freeman, Thomas Schulze, Christina Barr, Gregory Light, Vaishali Bakshi, Raymond Cho, Cynthia Crawford, Philip Szeszko

M2. Elevations of Brain Kynurenic Acid in Prenatal Rats Result in Longlasting Impairments in Cortical Development and Cognitive Flexibility: Implications for Schizophrenia

> John P. Bruno, Sarah Vunck, Michelle Pershing, Ana Pocivavsek, Dave Bortz, Christinna Jorgensen, Peter Fredericks, Benedetta Leuner, Robert Schwarcz

M3. Evidence for Both an Alpha7 Nicotinic and a Glycine B Receptor Mediation of Working Memory in the Rat

> <u>Sarah Vunck</u>, Michelle Pershing, Dave Bortz, Christinna Jorgensen, Robert Schwarcz, John P. Bruno

M4. Attenuation of Metabolic Consequences from Atypical Antipsychotic Use in Schizophrenia: Folate Supplementation and the Role of Pharmacogenomics

Vicki Ellingrod, Tyler Grove, Stephan F. Taylor, Kyle Burghardt

M5. Weight Gain Independent, Centrally-mediated Effects of Olanzapine on Glucose Metabolism

Margaret Hahn, Araba Chintoh, Gary Remington, Celine Teo, Steve W. Mann, Paul Fletcher, Jose Nobrega, Adria Giacca

#### Poster Session I—Monday

M6. Disturbances of Tryptophan Metabolism and Risk of Depression in Hev Patients Treated with Ifn-alpha

<u>Gregory F. Oxenkrug</u>, Waldemar Turski, Wojciech Zgrajka, Joel Weinstock, Paul Summergrad

M7. Sensitive Biomarkers of Metabolic Risk in Children Treated with Antipsychotics

<u>Ginger E. Nicol</u>, Michael D. Yingling, Julia A. Schweiger, John W. Newcomer

M8. Increased Rate of Chiari I Malformation in Children of Depressed Mothers Treated with Selective Serotonin Reuptake Inhibitors during Pregnancy

> <u>Rebecca Knickmeyer</u>, Samantha Meltzer-Brody, Sandra Woolson, Robert M. Hamer, Keith Smith, Kenneth Lury, John Gilmore

M9. Olanzapine and Diet Affect CNS and Peripheral Metabolic Outcomes in a Non-human Primate

Cynthia L. Bethea, Oleg Varlamov, Paul Kievit, Arubala P. Reddy, Charles T. Roberts

M10. The Risk of Switch to Mania in Patients with Bipolar Disorder during Treatment with Antidepressants Alone and in Combination with a Mood Stabilizer

Mikael Landén, Michael E. Thase

M11. Do α2-containing Nicotinic Acetylcholine Receptors Play a Role in Baseline and Nicotine-modulated Behaviors in Mice?

<u>Shahrdad Lotfipour</u>, Janet Byun, Prescott Leach, Christie D. Fowler, Niall Murphy, Paul Kenny, Thomas J. Gould, Jim Boulter

M12. Intraaccumbal Administration of Zeta Inhibitory Peptide (ZIP) Erases Drug Memory and Prevents Cocaine Reinstatement

Lisa A. Briand, Chris Pierce

#### **Poster Session I—Monday**

M13. Cocaine Sensitivity Is Regulated by Striatal α5-containing Nicotinic Acetylcholine Receptors

Christie D. Fowler, Brian Lee, Paul Kenny

M14. Exome Sequencing in Rhesus Macaques Exhibiting Individual Differences in Aggression

<u>Carlos Driscoll</u>, Kevin Blackistone, Jessica Clemente, Stephen Lindell, Stephen Suomi, Christina Barr

M15. Conditional Elimination of the Interleukin-1 Receptor for the Study of the Impact of Inflammatory Cytokines on Brain and Behavior

Matthew J. Robson, Chong-Bin Zhu, Kathryn M. Lindler, Nicole Baganz, Jane Wright, William Hewlett, Randy D. Blakely

M16. Genes Harboring Addiction-related Variants Alter Dose-response Relationships for Stimulant Reward

<u>George Uhl</u>, Frank S. Hall, Barbara Ranscht, Noriko Uetani, Jana Drgonova

M17. Mother's 5-HTTLPR Genotype X Infant's Genotype Interact to Affect Mother-infant Interactions and Developmental Outcomes: Aggression, Anxiety, and Social Behavior

> Patrick O'Connell, Jenna Jackson, Stephen Lindell, Andrea Sorenson, Courtney Lindell, Melanie L. Schwandt, Stephen J. Suomi, Christina Barr, J. Dee Higley

M18. Genome-wide Mapping of Complex Psychiatric Traits in Commercially Available Outbred Mice

Clarissa C. Parker, Natalia M. Gonzales, Abraham A. Palmer

M19. A Cross-species Investigation into the Role of Lhx6 in Cortical Inhibitory Circuitry Disturbances in Schizophrenia

David W. Volk, Jessica R. Edelson, David A. Lewis

#### Poster Session I—Monday

M20. DNA Methylation Network Dysregulation Expressed in Lymphocytes of Schizophrenic Patients

<u>Robert C. Smith</u>, James Auta, Henry Sershen, Abel Lajtha, Sylvia Boules, Patricia L. Gerbarg, , Richard Brown , John M. Davis, Alessandro Guidotti

- M21. Region-specific Alteration of Wnt Signaling in Bipolar Disorders <u>Ghanshyam N. Pandey</u>, Xinguo Ren, Hooriyah S. Rizavi, Yogesh Dwivedi
- M22. An Integrated-omics Approach to Understanding Psychoneuroimmunology Crosstalk

Anil G. Jegga, Gayle Wittenberg, Xiang Yao, Lynn Yieh, <u>Guang</u> <u>Chen</u>, Vaibhav Narayan

M23. Key Role of Decreased Vesicular Uptake in the Profound Myocardial Norepinephrine Depletion in Parkinson's Disease

Irwin J. Kopin, Patti Sullivan, David S. Goldstein

M24. Chondroitin Sulfate Proteoglycan Abnormalities in Schizophrenic and Bipolar Disorder Subjects

<u>Harry Pantazopoulos</u>, Florian Jaquet, Doel Ghosh, Anna Wallin, Bruce Caterson, Sabina Berretta

M25. CSF and Plasma Interleukin-6 and Personality Traits in Suicide Attempters

<u>Jussi Jokinen</u>, Josef Isung, Shahin Aeinehband, Fariborz Mobarrez, Peter Nordström, Bo Runeson, Fredrik Piehl, Marie Åsberg

M26. Body Mass Index Affects Brain Dopaminergic Signaling After Glucose Ingestion

<u>Gene-Jack Wang</u>, Nora D. Volkow, Dardo Tomasi, Antonio Convit, Christopher Wong, Elena Shumay, Joanna Fowler
M27. Abnormal Bioenergetics in Schizophrenia and Bipolar Disorders Studied by Dynamic 31P-MRS

Fei Du, Cagri Yuksel, Scott Lukas, Bruce Cohen, Dost Ongur

M28. Expression of CHRNA7 and the Chimeric Gene CHRFAM7A are Altered in the Postmortem Dorsolateral Prefrontal Cortex in Major Psychiatric Disorders

Yasuto Kunii, Thomas M. Hyde, Amy Deep-Soboslay, Daniel R. Weinberger, Joel E. Kleinman, <u>Barbara Lipska</u>

M29. Neural Correlates of Response Inhibition and Concentration of Glutamate/GABA in the Anterior Cingulate Cortex in Borderline Personality Disorder

Annegret Krause-Utz, Julia van Eijk, Sylvia Cackowski, Traute Demirakca, <u>Christian Schmahl</u>, Gabriele Ende

M30. Measuring the Effects of Acute Alcohol Infusion on Human Brain Metabolites: An MR Spectroscopy Study

Claire Mann, Caitlin Durkee, Erica N. Grodin, Vijay A. Ramchandani, <u>Reza Momenan</u>

M31. Morphological Alterations in Layer 3 Pyramidal Cells of the Dorsolateral Prefrontal Cortex in Schizophrenia: Role of Actin Cytoskeleton

Dibyadeep Datta, Dominique Arion, David A. Lewis

M32. Impact of DOPA Decarboxylase Genetic Variation on Its In Vivo Enzymatic Activity in Humans

Daniel P. Eisenberg, Joseph C. Masdeu, Philip Kohn, Bhaskar S. Kolachana, Daniel R. Weinberger, Karen F. Berman

M33. Synaptophysin, vGlut1, Mitofusin2 and Calcineurin Protein Levels in the Anterior Cingulate Cortex in Schizophrenia: Relation to Treatment and Treatment Response

Rosalinda C. Roberts, Keri A. Barksdale, Adrienne C. Lahti

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#### **Poster Session I—Monday**

M34. Neurochemistry of First-hospitalization Manic Youth

<u>Marguerite R. Schneider</u>, Tessa Benanzer, Wade Weber, Luis R. Patino Duran, Jeffrey Strawn, Jeffrey Welge, Caleb Adler, Strakowski Stephen, Melissa DelBello

M35. Cortical Thickness in Individuals with Subclinical and Clinical Psychotic Symptoms

Iris Sommer, Marieke Begemann, Remko van Lutterveld

M36. Brain Activation to Natural Cues and Drug Cues and Dopamine Receptors in Cocaine Addicts

<u>Dardo Tomasi</u>, Gene-Jack Wang, Elisabeth Caparelli, Nora D. Volkow

M37. Brain-derived Neurotrophic Factor and Deficient Amygdala Habituation in Borderline Personality Disorder: A Research Domain Criteria Imaging Genetics Study

> <u>M. Mercedes Perez-Rodriguez</u>, Antonia S. New, Kim Goldstein, Qiaoping Yuan, Zhifeng Zhou, Colin A. Hodgkinson, David Goldman, Larry Siever, Erin Hazlett

M38. The Spatiotempotal Organization of Subcortical Anatomy in Human Development

<u>Armin Raznahan</u>, Shaw Phillip, Clasen Liv, Deanna Greenstein, Jason Lerch, Mallar Chakravarty, Jay Giedd

M39. Parametric Modulation of Neural Activity during Face Emotion Processing in Unaffected Youth at Familial Risk for Bipolar Disorder

> Melissa A. Brotman, Christen M. Deveney, Laura A. Thomas, Kendra Hinton, Jennifer Yi, Daniel S. Pine, Ellen Leibenluft

M40. Non-smoking Chronic Alcoholics Following Withdrawal Show Increased Cerebral Blood Flow and Altered Brain Docosahexaenoic (DHA) Metabolism on Partial Volume Error-corrected PET

> John C. Umhau, Weiyin Zhou, Shantalaxmi Thada, Peter Herscovitch, Norman Salem Jr,, Joseph R. Hibbeln, Jussi Hirvonen, <u>Stanley Rapoport</u>

M41. Developmental Differences in Resting-state Network Connectivity in Autism Spectrum Disorder

Dienke Bos, Tamar van Raalten, Anouk Smits, Janna van Belle, Serge Rombouts, <u>Sarah Durston</u>

M42. Abnormal Functional Brain Network Organization for Visual Processing of Non-appearance Stimuli in Body Dysmorphic Disorder

Teena Moody, Jesse Brown, Alex D. Leow, Liang Zhan, Jamie Feusner

M43. Reward-based Spatial Learning in Unmedicated Adults with Obsessivecompulsive Disorder

> <u>Rachel Marsh</u>, Yuankai Huo, Ge Lui, Mark J. Packard, Gregory Z. Tau, XueJun Hao, Bradley S. Peterson, Zhishun Wang, Helen Blair. Simpson

M44. Alterations of Cortical Thickness Related to Clinical Severity, but Not the Untreated Disease Duration in Schizophrenia

Su Lui, Yuan Xiao, Li Yao, Yong He, Qiyong Gong

M45. Differential Effects of Estrogen Hormone Therapy on CA1 Hippocampal Subfield Volume Change Over a 2-Year Observation Period in Postmenopausal Women at Risk for Alzheimer's Disease: Conjugated Equine Estrogen Versus Estradiol

Heather Kenna, Kristen Sheau, Tonita Wroolie, Ryan G. Kelley, Katherine Williams, Allan Reiss, <u>Natalie Rasgon</u>

M46. Effects of Serotonin Depletion on Punishment Processing in the Orbito Frontal and Anterior Cingulate Cortices in Healthy Women

> <u>Katrin Helmbold</u>, Michael Zvyagintsev, Brigitte Dahmen, Sarah Bubenzer, Tilman J. Gaber, Molly Crockett, Martin Klasen, Cristina L. Sanchez, Albert Eisert, Kerstin Konrad, Ute Habel, Beate Herpertz-Dahlmann, Florian Daniel. Zepf

M47. Serotonin and Affect Regulation in Humans: A Combined 5-HT1A [11C] CUMI-101 PET and FMRI Study

> <u>Sudhakar Selvaraj</u>, Elias Mouchlianitis, Paul Faulkner, Federico Turkheimer, Philip Cowen, Jonathan Roiser, Oliver Howes

M48. Dysregulated Neural Response to Social Evaluation in Bullied Adolescents: A Potential Mechanism that Promotes Risk for Social Anxiety Disorder

> Johanna M. Jarcho, Megan Davis, Ellen Leibenluft, Nathan Fox, Tomer Shechner, Daniel S. Pine, Eric Nelson

M49. Prenatal Exposure to Maternal Infection Alters Neonatal Brain Structure

John H. Gilmore, Mark Connelly, Philip Nielsen, Sandra Woolson, Robert Hamer, Rebecca Knickmeyer, Sarah Short, Xiujuan Geng

M50. Memory Retrieval of Addiction-related Images Induce Greater Insular Activation as Revealed by an fMRI Based Delayed Matching to Sample Task

<u>Amy Janes</u>, Robert Ross, Stacey Farmer, Blaise Frederick, Lisa Nickerson, Scott Lukas, Chantal Stern

M51. Nicotinic Acetylcholine Receptor Density as a Predictor of Quitting Smoking with Treatment

<u>Arthur Brody</u>, Alexey Mukhin, Michael Mamoun, Trinh Luu, Meaghan Neary, Lidia Liang, Jennifer Shieh, Catherine A. Sugar, Jed Rose, Mark Mandelkern

M52. Striatal Activation Induced by mGluR2 Positive Allosteric Modulation Correlates with Negative Symptom Reduction in Schizophrenia

> Daniel Wolf, Kosha Ruparel, Bruce Turetsky, Christian Kohler, Theodore D. Satterthwaite, Mark Elliott, Mary March, Alan Cross, Mark Smith, Stephen R. Zukin, Ruben C. Gur, Raquel E. Gur

M53. Olfactory Functional Magnetic Resonance Imaging (fMRI) in Combat Veterans: Brain Reactivity to Trauma-related Odor Cues

Bernadette M. Cortese, Qing X. Yang, Ron Acierno, Kimberly Leslie, Thomas W. Uhde

- M54. Methylphenidate and Brain Activity in a Reward/Conflict Paradigm <u>Iliyian Ivanov</u>, Xun Liu, Suzanne Clerkin, Kurt Schulz, Jin Fan, Jeffrey Newcorn
- M55. Being Liked Increases Social Motivation, but Not in Depressed Individuals: A μ-Opioid Positron Emission Tomography (PET) Study of the Ventral Striatum

<u>David T. Hsu</u>, Benjamin J. Sanford, Kortni Meyers, Kathleen E. Hazlett, Tiffany Love, Brian J. Mickey, Scott Langenecker, Jon-Kar Zubieta

M56. Prediction Error Reactivity and Its Relation to Reward Expectancy Are Altered in Major Depressive Disorder: Preliminary Findings from the EMBARC Study

> <u>Tsafrir Greenberg</u>, Henry Chase, Jorge Almeida, Richelle Stiffler, Carlos R. Zevallos, Haris Aslam, Thilo Deckersbach, Sarah Weyandt, Crystal Cooper, Benji T. Kurian, Patrick J. McGrath, Maurizio Fava, Myrna M. Weissman, Ramin V. Parsey, Madhukar Trivedi, Mary L. Phillips

M57. In Vivo Neurochemical Effects of Ketamine in OCD: A Pilot Proton Magnetic Resonance Spectroscopy Time-course Study of Cortical Glutamate-glutamine and GABA

> <u>Carolyn I. Rodriguez</u>, Lawrence S. Kegeles, Amanda Levinson, Todd Ogden, Xiangling Mao, Matthew Milak, Dikoma Shungu, Helen Blair. Simpson

M58. Subcortical Biophysical Abnormalities in Patients with Mood Disorders

<u>Anand Kumar</u>, Shaolin Yang, Olusola Ajilore, Minjie Wu, Rebecca Charlton, Jamie Cohen, Melissa Lamar

M59. Accounting for Dynamic Fluctuations across Time When Examining Test-retest Reliability: Analysis of a Reward Paradigm in the EMBARC Study

> <u>Henry Chase</u>, Jay Fournier, Tsafrir Greenberg, Jorge Almeida, Richelle Stiffler, Crystal Cooper, Thilo Deckersbach, Sarah Weyandt, Philips Adams, Maurizio Fava, Patrick J. McGrath, Myrna M. Weissman, Ramin V. Parsey, Benji T. Kurian, Madhukar Trivedi, Mary L. Phillips

M60. Increased Serotonin Transporter Binding Is Associated with Depression Development during Interferon-alpha Exposure in Humans

Francis E. Lotrich, Rajesh Narendran

M61. Naloxone-reversible Modulation of Pain Circuitry by Left Prefrontal Repetitive Transcranial Magnetic Stimulation

Joseph J. Taylor, Jeffrey J. Borckardt, Melanie Canterberry, Xingbao Li, Colleen A. Hanlon, Truman Brown, Mark S. George

M62. Abnormalities of Two Distributed Brain Networks in Major Depression

Alexander Petti, Daniel Kessler, Mary Heitzeg, Scott Langenecker, Tiffany Love, Kenneth Silk, Jon-Kar Zubieta, Chandra Sripada, Brian J. Mickey

M63. Contrasting Gray Matter Volume Biomarkers by Diagnosis and Biotype Across Schizophrenia - Bipolar Disorder Psychosis Dimension

<u>Elena I. Ivleva</u>, Anup S. Bidesi , Brett A. Clementz, Matcheri S. Keshavan, Shashwath A. Meda, Godfrey D. Pearlson, John A. Sweeney, Gunvant K. Thaker, Carol A. Tamminga

M64. Neuroimaging Abnormalities in Borderline Personality Disorder: MRI, MRS, fMRI and DTI Findings

Courtney McKenzie, Henry Nasrallah

M65. Cerebral Blood Flow Differences in Major Depressive Disorder Using Arterial Spin Labeling: Preliminary Results from the EMBARC Study

> <u>Crystal Cooper</u>, Hanzhang Lu, Jorge Almeida, Henry Chase, Thomas Carmody, Maurizio Fava, Tony Jin, Benji T. Kurian, Patrick J. McGrath, Melvin McInnis, Maria Oquendo, Ramin V. Parsey, Myrna M. Weissman, Sarah Weyandt, Mary L. Phillips, Madhukar Trivedi

M66. Exposure to Regional Anesthesia during Labor and Delivery and Its Effect on Neonatal Brain Morphology

Marisa N. Spann, Ravi Bansal, Tove Rosen, Bradley S. Peterson

M67. Abnormal Deactivation of Ventrolateral Prefrontal Cortex during Emotion Processing in Youth with Bipolar Disorder: Effects of Medication and Mood State

> Danella Hafeman, Genna Bebko, Michele A. Bertocci, Lisa Bonar, Susan Perlman, Vaibhav Diwadkar, Robert Kowatch, Boris Birmaher, Sarah Horwitz, Eugene Arnold, Mary Fristad, Eric Youngstrom, Robert Findling, Thomas Frazier, Wayne Drevets, Mary L. Phillips

M68. Kappa Opioid Receptor Systems and Threat, Loss, and Reward Responsiveness

Robert Pietrzak, Yiyun Huang, Mika Naganawa, Stefani Corsi-Travali, Richard E. Carson, <u>Alexander Neumeister</u>

#### ACNP 52nd Annual Meeting • Final Program

#### **Poster Session I—Monday**

M69. Pallidial Resting State Connectivity in Bipolar Disorder: Implications for Differences between Manic and Depressive States

Amit Anand, Harish Karne

M70. Hippocampus NAA as Biological Marker of Anhedonia in PTSD and Trauma-exposed Adults: Preliminary 1H-MRS Findings

Isabelle I. Rosso, David I. Crowley, Lily I. Preer, Marisa Silveri, J. Eric Jensen

M71. Equal HIV Risk Reduction with Buprenorphine-naloxone or Methadone

<u>George E. Woody</u>, Douglas Bruce, P. Todd Korthuis, Sumedha Chhatre, Maureen Hillhouse, James L. Sorensen, Andrew J. Saxon, Petra Jacobs, David S. Metzger, Sabrina Poole, Walter Ling

M72. Excellent Test-retest Reliability of Cerebral Blood Flow in Healthy Individuals Measured with Arterial Spin Labeling: EMBARC Study Preliminary Results

> Jorge Almeida, Hanzhang Lu, Henry Chase, Jay Fournier, Crystal Cooper, Thilo Deckersbach, Mohammad Zia, Maurizio Fava, Benji T. Kurian, Patrick J. McGrath, Maria Oquendo, Melvin McInnis, Ramin V. Parsey, Myrna M. Weissman, Madhukar Trivedi, Mary L. Phillips

M73. Cortico-striatal GABAergic and Glutamatergic Dysregulations in Subjects at Ultra-high Risk for Psychosis Investigated with 1H MRS

> <u>Camilo de la Fuente</u>, Pablo León-Ortiz, Xiangling Mao, Francisco Reyes-Madrigal, Oscar Rodríguez-Mayoral, Patricia Alvarado-Alanis, Rodolfo Solis-Vivanco, Rafael Favila, Ariel Graff, Dikoma Shungu

M74. Cortical Thickness as a Contributor to Abnormal Oscillations in Schizophrenia?

<u>José Cañive</u>, Yu-Han Chen, Breannan Howell, Cassandra Wootton, Michael Hunter, Mingxiong Huang , Gregory A. Miller, J Christopher Edgar

M75. Lithium and Brain Glucose Metabolism in Patients with Bipolar-I Disorder

<u>Abesh Bhattacharjee</u>, Monte S. Buchsbaum, Michael J. McCarthy, Anna DeModena, John Kelsoe

M76. FDG-PET Scans in Patients with Good and Poor Prognosis Schizophrenia

Monte S. Buchsbaum, Marie-Cecile Bralet, Serge Mitelman

M77. Tau PET Imaging of Neurocognitive Disorders Using Newly Developed Tau Ligand [11C]PBB3

> <u>Tetsuya Suhara</u>, Hitoshi Shimada, Masahiro Maruyama, Hitoshi Shinotoh, Bin Ji, Jun Maeda, Harumasa Takano, Naruhiko Sahara, Ming-Rong Zhang, Hiroshi Ito, Makoto Higuchi

M78. Food Reward Circuitry Hyperactivation, Acylated Ghrelin, and Hedonic Capacity in Women with Remitted Major Depressive Disorder

Laura M. Holsen, Kara Christensen, Priyanka Moondra, Anne A. Klibanski, Jill M. Goldstein

M79. Brain Injury in Battered Women and Its Relationship to Microstructural White Matter Alterations: A Diffusion Tensor Imaging Study

Eve Valera, Alan Francis, Nikos Makris, Zhi Li, Ezra Wegbreit, Margaret O'Connor

M80. Examining Fronto-striatal Circuit Structure and Function in Treatmentnaïve Children and Adolescents with Obsessive Compulsive Disorder

> <u>Stephanie Ameis</u>, Colleen Dockstader, Don Mabbott, Sandra Mendlowitz, Reva Schachter, Clara Tam, Fang Liu, Elysa Widjaja, Russell Schachar, Paul D. Arnold

M81. Multimodal Brain Connectivity Analysis Using Functional-by-Structural Hierarchical Mapping

<u>Olusola Ajilore</u>, Liang Zhan, Johnson Jonaris, Gad Elkarim, Aifeng Zhang, Jamie Feusner, Shaolin Yang, Paul Thompson, Anand Kumar, Alex D. Leow

M82. Modulation of Resting Brain Cerebral Blood Flow by the GABA B Agonist, Baclofen: A Longitudinal Perfusion fMRI Study in Marijuana Dependent Treatment Seeking Individuals

Kanchana Jagannathan, Reagan R. Wetherill, Julian Bender, Barbara Johnson, Joel Mumma, Kyle Kampman, Charles P. O'Brien, Anna Rose Childress, <u>Teresa R. Franklin</u>, Kimberly A. Young, Jesse J. Suh

M83. Chemokine-specific Relationships to AD Biomarkers in CSF in Healthy Older Adults

Nunzio Pomara, Davide Bruno, Chelsea Reichert, Jay Nierenberg, John J. Sidtis, Frank T. Martiniuk, Henrik Zetterberg, Kaj Blennow

M84. Varenicline Effects on Neural Reward Processing among Non-treatmentseeking Alcohol Dependent Individuals

> Joseph P. Schacht, Raymond F. Anton, Patrick Randall, Xingbao Li, Scott Henderson, Hugh Myrick

M85. PACAP Receptor (ADCYAP1R1) Genotype Associates with Fear Responses in the Amygdala and Hippocampus in Highly-traumatized Civilians

> <u>Kerry J. Ressler</u>, Jennifer Stevens, Lynn Almli, Negar Fani, David Gutman, Bekh Bradley, Seth D. Norrholm, Ebony Glover, Tanja Jovanovic

M86. Association Between Primary Insomnia and Major Depression: Distinct Entities or Spectrum Disorders?

<u>Ruth Benca</u>, Brady Riedner, Michael Goldstein, Lihong Cui, Anja Schmitz, Jihui Zhang, Kathleen Merikangas

M87. Neuroprotective Kynurenine Pathway Metabolites Are Associated with Larger Hippocampal and Amygdalar Volumes in Patients with Major Depressive Disorder

> <u>Jonathan Savitz</u>, Wayne C. Drevets, Teresa Victor, Jerzy Bodurka, Kent Teague, Robert Dantzer

M88. Evaluating the Impact of Early Life Stress on DLPFC Functional Connectivity in Healthy Adults: Informing Future Studies of Transcranial Magnetic Stimulation

<u>Noah S. Philip</u>, Thomas R. Valentine, Audrey R. Tyrka, Lawrence H. Price, Lawrence H. Sweet, Linda L. Carpenter

M89. Persistent Cannabis Use During Adolescence Is Linked to Thinner Hippocampal Cortex in Late Life After Decades of Abstinence

Alison Burggren, Brian Renner, Edythe D. London

- M90. Maternal Depression Affects Brain Responses to Baby Cry James E. Swain, S Ho, Katherine Rosenblum, Maria Muzik
- M91. Socially Rewarding Stimuli and Anhedonia Severity Among Depressed Adolescents

Vilma Gabbay, Sarah E. Henderson, Ana Vallejo, Rachel G. Klein

M92. Sleep Duration Contributes to Cortico-limbic Functional Connectivity, Emotional Functioning, & Psychological Health

William Killgore

M93. A Longitudinal MR Spectroscopy Study of the Anterior Cingulate Cortex and Hippocampus Before and After Antipsychotic Treatment in Patients with Schizophrenia

<u>Meredith A. Reid</u>, Nina V. Kraguljac, David M. White, Jan A. den Hollander, Adrienne C. Lahti

M94. Glutamate Levels Determined with Magnetic Resonance Spectroscopy (MRS) in the Medial Prefrontal Cortex of Patients with Psychosis as Compared to Healthy Volunteers

> <u>Stefano Marenco</u>, Yan Zhang, Anna Slagle, Susie Kuo, Christian Meyer, Adhiraaj Sethi, Alan S. Barnett, Jun Shen, Daniel R. Weinberger, Karen F. Berman

M95. Longitudinal Effects of Antipsychotic Treatment on Functional Connectivity of the Striatum in Patients with First-episode Psychosis

> <u>Deepak Sarpal</u>, Delbert G. Robinson, Todd Lencz, Toshikazu Ikuta, Miklos Argyelan, Katherine H. Karlsgodt, Juan A. Gallego, John Kane, Philip R. Szeszko, Anil Malhotra

M96. Multimodal Analysis of Brain Networks Structural and Functional Connectivity Changes in Non-medicated Late-life Depression

Reza Tadayon-Nejad, Shaolin Yang, Anand Kumar, Olusola Ajilore

M97. Neural Response during Indirect and Direct Processing of Emotional Faces Predicts Improvement Following Cognitive Behavioral Therapy in Generalized Social Anxiety Disorder

Heide Klumpp, Daniel Fitzgerald, David Post, K. Luan Phan

M98. The Effect of Electroconvulsive Therapy on Emotional Processing in Major Depressive Disorder: A Neuroimaging Study

> <u>Miklos Argyelan</u>, Styliani Kaliora, Harlington Hanna, Toshikazu Ikuta, Peter B. Kingsley, Deepak Sarpal, Philip R. Szeszko, Anil Malhotra, Georgios Petrides

M99. Categories and Dimensions of Anxiety and Depression in the Resting fMRI Signal

Desmond Oathes, Alan F. Schatzberg, Amit Etkin

M100. Changes in Cortical Thickness in Children of Parents with Bipolar Disorder

Roberto Sassi, Lindsay Hanford, Luciano Minuzzi, Geoffrey Hall

M101. Myelin and Axon Abnormalities in Schizophrenia and Bipolar Disorder Measured with Magnetization Transfer Ratio and Diffusion Tensor Spectroscopy

<u>Ann K. Shinn</u>, Fei Du, Thida Thida, Bruce M. Cohen, Dost Ongur, Kathryn E. Lewandowski

M102. Brain White Matter Development Is Associated with a Human-specific Haplotype Increasing the Synthesis of Long Chain Fatty Acids

> <u>Bart D. Peters</u>, Aristotle N. Voineskos, Philip R. Szeszko, Tristram Lett, Pamela DeRosse, Saurav Guha, Toshikazu Ikuta, Daniel Felsky, Majnu John, James L. Kennedy, Anil Malhotra

M103. Ketamine Reduces Left Nucleus Accumbens Volume within 24 Hours of Treatment of Major Depressive Disorder Patients

<u>Chadi Abdallah</u>, Andrea Jackowski, Ramiro Salas, Swapnil Gupta, João R Sato, Lee C. Chang, Xiangling Mao, Jeremy Coplan, Dikoma Shungu, Sanjay J. Mathew

M104. Visual Hallucinations in Patients with Schizophrenia Are Associated with Visual Cortex Hyperconnectivity to Amygdala and Hippocampus

<u>Judith M. Ford</u>, Vanessa Palzes, Brian J. Roach, Steven Potkin, Theo Van Erp, Jessica Turner, James Voyvodic, Bryon Mueller, Vincent Calhoun, Ayse Belger, Jatin Vaidya, Adrian Preda, FBIRN, Daniel H. Mathalon

M105. Baclofen Reduces Resting Blood Flow, and Correlations with Limbic Cue Reactivity, in the Ventral Striatum of Cocaine-dependent Men

<u>Kimberly A. Young</u>, Teresa R. Franklin, Yin Li, Kanchana Jagannathan, Reagan R. Wetherill, Jesse J. Suh, Zachary D. Singer, Samuel E. Davidson, Zachary A. Monge, Charles P. O'Brien, Anna R. Childress

M106. "Trouble Waiting to Happen?" Heightened Striatal Resting Perfusion in Cocaine Patients Predicts Limbic Vulnerability to Drug Cues

> <u>Anna R. Childress</u>, Kimberly A. Young, Teresa R. Franklin, Kanchana Jagannathan, Yin Li, Jesse J. Suh, Ronald Ehrman, Ze Wang, Zachary D. Singer, Zachary A. Monge, Daniel D. Langleben, Charles P. O'Brien

M107. Test-retest Reliability in Extinction Recall: A Neuroimaging Study of Healthy Adults

Jennifer Britton, Carolyn Spiro, Tomer Shechner, Gang Chen, Daniel S. Pine

M108. Hippocampal and Amigdala Volume Increase in Lithium-treated Bipolar I Patients Compared with Unmedicated Patients and Healthy Subjects

> <u>Carlos Lopez-Jaramillo</u>, Cristian David. Vargas Upegui, Juan Palacio, Gabriel Castrillon, Eduard Vieta, Carrie Bearden, Scott C. Fears, Nelson Freimer, Javier I. Escobar

M109. Greater Translocator Protein (TSPO) Distribution Volume During Major Depressive Episodes of Major Depressive Disorder

> <u>Jeffrey H. Meyer</u>, Elaine Setiawan, Romina Mizrahi, Pablo Rusjan, Alan A. Wilson, Sylvain Houle

M110. Distinct Patterns of Functional Connectivity in Patients with Childhoodonset Schizophrenia, Their Unaffected Siblings, and Healthy Controls

<u>Rebecca A. Berman</u>, Harrison McAdams, Deanna Greenstein, Nitin Gogtay, Judith L. Rapoport

M111. Alterations in Amygdala Functional Circuitry as a Neural Marker of Emotion Dysregulation in Young Children

<u>Amy K. Roy</u>, Rachel G. Klein, Clare Kelly, Francisco Xavier Castellanos

M112. Fear-potentiated Startle during Extinction Is Associated with Alterations in White Matter Connectivity

<u>Negar Fani</u>, Tricia King, Amita Srivastava, Ryan Brewster, Seth D. Norrholm, Kerry J. Ressler, Tanja Jovanovic

M113. Diffuse Tensor Imaging-based Brain Signatures Accurately Discriminate a Functional Pain from Health: Examining Central Mechanisms in Visceral Pain

> Jennifer Labus, John D. Van Horn, Carinna Torgerson, Cody Ashe-McNalley, Andrei Irimia, Micah C. Chambers, Arpana Gupta, Kirsten Tillisch, Emeran A. Mayer

M114. Clinical and Neuropsychological Correlates of DTI-derived Connectome Structure in Euthymic Bipolar I Disorder

> <u>Alex D. Leow</u>, Olusola Ajilore, Johnson Gadelkarim, <u>Jamie Feusner</u>, Teena Moody, Anand Kumar, Lori Altshuler

M115. Intrinsic Hippocampal Activity as a Biomarker for Cognition and Symptoms in Schizophrenia

Jason Tregellas, Jason Smucny, Josette Harris, Ann Olincy, Robert Freedman

M116. Morphometric and Volumetric Subcortical Differences in Alcoholics with and without Comorbid Drug Use Disorders

Erica N. Grodin, Reza Momenan

M117. A Preliminary Comparison of Methodologies for Quantifying Brain Gamma-aminobutyric-acid Concentrations In Vivo Using Proton Magnetic Resonance Spectroscopy

James J. Prisciandaro, Andrew Prescot, Joseph P. Schacht, Raymond F. Anton, Perry F. Renshaw, Truman Brown

M118. Prefrontal Cortex Activation during Safety Signal Processing in Generalized Anxiety Disorder as a Correlate of Overgeneralization

Katja Beesdo-Baum, Kevin Hilbert, Ulrike Lueken

M119. Longitudinal Change in Amyloid Deposition, Measured by PET and 11-C-PiB, in Older Adults

<u>Susan M. Resnick</u>, Murat Bilgel, Yang An, Madhav Thambisetty, Michael Kraut, Yun Zhou, Dean F. Wong

M120. Global Resting-state fMRI Analysis Identifies Frontal Cortex, Striatal, and Cerebellar Dysconnectivity in Obsessive-compulsive Disorder

Alan Anticevic, Sien Hu, Sheng Zhang, Patricia Gruner, Aleksandar Savic, Eileen Billingslea, Suzanne Wasylink, Grega Repovs, Michael Cole, Sarah Bednarski, John H. Krystal, Michael H. Bloch, Chiangshan Ray Li, <u>Christopher Pittenger</u>

M121. Examining Domains of Borderline Personality Disorder Using MRI

<u>S. Charles Schulz</u>, Kathryn R. Cullen, Bryon Mueller, Alaa Houri, Lizz Coykendall, Kelvin O. Lim

M122. The Norepinephrine Transporter: A Novel Target for Imaging Brown Adipose Tissue

<u>Yu-Shin Ding</u>, Janice Hwang, Catherine Yeckel, Jean-Dominique Gallezot, Renata Belfort-Deaguiar, Devrim Ersahin, Richard Carson, Robert Sherwin

M123. Measuring Smoking-induced Extrastriatal Dopamine Release: A [11C] FLB-457 PET Study

Victoria C. Wing, Doris E. Payer, Tony P. George, Isabelle Boileau

M124. A Longitudinal Mentoring and Training Program for Psychiatric Scientists

David J. Kupfer, Alan F. Schatzberg, Leslie Dunn, Melissa DeRosier, Helena Kraemer, Andrea Schneider

M125. EEG and fMRI Findings of Reduced Neural Synchronization during Visual Integration in Schizophrenia

Jonathan K. Wynn, Junghee Lee, William Horan, Brian J. Roach, Alexander S. Korb, Judith M. Ford, Michael F. Green

M126. Translating Functional Neuroimaging into Clinical Care by Modeling Normative Variance in Cognition and Neural Function: Insights from the Cognitive Connectome

<u>George A. James</u>, Jennifer S. Fausett, Jennifer L. Gess, Tonisha Kearney-Ramos, Clinton D. Kilts

M127. Low Fractional Anisotropy of the Right Ventral Anterior Cingulate Related to Depressive Symptoms in Atherosclerotic Vascular Disease

> Kelly Rowe Bijanki, Joy Matsui, Helen S. Mayberg, Vincent A. Magnotta, Stephan Arndt, Hans Johnson, Peggy C. Nopoulos, Sergio Paradiso, Laurie M. McCormick, Jess Fiedorowicz, Eric Epping, <u>David J. Moser</u>

M128. Moderate and Heavy Marijuana Use: Differences in Whole-brain Functional Network Structure that Underlie Iowa Gambling Task Performance

Malaak N. Moussa, Linda Porrino

M129. Executive Control Network Dysfunction in Major Depressive Disorder Patients with Early Life Stress: Preliminary Findings from the International Study to Predict Optimized Treatment in Depression

> <u>Shefali Miller</u>, Lisa McTeague, Anett Gyurak, Brian Patenaude, Leanne Williams, Amit Etkin

M130. Striatal Dopamine Transporter Availability in Obsessive-compulsive Disorder: A Randomized Clinical Trial Using [Tc99m]-TRODAT-1 SPECT

> <u>Marcelo Q. Hoexter</u>, Darin Dougherty, Roseli G. Shavitt, Juliana Belo. Diniz, Thilo Deckersbach, Ming Chi, João R Sato, Geraldo Busatto, Euripedes C Miguel, Rodrigo Bressan

M131. Cortico-amygdala Coupling as a Marker of Early Relapse Risk in Cocaine-addicted Individuals

<u>Meredith J. McHugh</u>, Demers Catherine, Braud Jacquelyn, Betty J. Salmeron, Michael D. Devous, Richard W. Briggs, N. Robrina Walker, Bryon Adinoff, Elliot A. Stein

M132. Distinct Types of Sensory Prediction-error Signals in Schizophrenia with Active Psychosis

Guillermo Horga, Anissa Abi-Dargham, Bradley S. Peterson

M133. Poor Amygdalofrontal Connectivity Predicts Symptomatic Deterioration in At-risk Youth

<u>Daphne J. Holt</u>, Emily A. Boeke, Avram J. Holmes, Garth Coombs, Amy Farabaugh, Maren Nyer, Maeve Ward, Susanna Crowell, Clair Cassiello, Angela Pisoni, Paola Pedrelli, Randy L. Buckner, Maurizio Fava

M134. Ketamine-induced Changes in [11C]ABP688 Binding in Healthy Human Subjects

<u>Irina Esterlis</u>, Nicole Dellagioia, Gerard Sanacora, Michael H. Bloch, Wendol Williams, Nabeel B. Nabulsi, John H. Krystal, Ramin V. Parsey, Richard E. Carson, Christine DeLorenzo

M135. Modafinil-induced Enhancement of Learning and Related fMRI Activation in Humans Reflects Individual Differences in Striatal Dopamine D2/D3 Receptor Availability

> <u>Dara Ghahremani</u>, Chelsea Roberson, Kenji Ishibashi, Golnaz Tabibnia, John Monterosso, Mark Mandelkern, Russell Poldrack, Edythe D. London

M136. Social Impairment Is Related to Frontolimbic Structural Connectivity and Functional Activity in Autism Spectrum Disorders

<u>Kimberly A. Stigler</u>, Tom A. Hummer, Yang Wang, Brenna C. McDonald, Andrew J. Saykin

M137. Watching Cerebral Blood Flow Using fMRI Yunjie Tong, Blaise Frederick

M138. Early Life Stress and Intra- and Extra-amygdaloid Effective Connectivity

<u>Merida Grant</u>, Kimberly Wood, Muriah Wheelock, Karthik R. Sreenivasan, Richard C. Shelton, David C. Knight, Gopikrishna Deshpande, Joshua R. Shuman

M139. Buspirone Blocks Dopamine D3 Receptors in the Non-human Primate Brain when Administered Orally

Sung Won Kim, Joanna Fowler, Phil Skolnick, Yeona Kang, Dohyun Kim, Nora D. Volkow

M140. A Novel fMRI Task to Evaluate Social Reward and Social Threat Hypersensitivity in Depressed Mothers of Psychiatrically III Children

> <u>Holly A. Swartz</u>, Jill M. Cyranowski, Jennifer Silk, Marlissa Amole, Marigrace Ambrosia, Susan Murphy, Stacy Martin, Judith Morgan, Samuel Musselman, Erika E. Forbes

M141. Disrupted Resting State Functional Connectivity in Unmedicated Patients with Schizophrenia

<u>Nina V. Kraguljac</u>, David M. White, Jennifer Hadley, Adrienne C. Lahti

M142. Connectivity Deficits in Chronic Stress and Depression: Resilience, Reversibility, and Clinical Implications

> Andrew T. Drysdale, Benjamin Zebley, Ashley C. Chen, Amit Etkin, Marc J. Dubin, <u>Conor Liston</u>

M143. Reduced Functional Connectivity in Executive Networks Associated with Cigarette and Alcohol Use

Barbara Weiland, Amithrupa Sabbineni, Vincent Calhoun, Robert Welsh, Angela Bryan, Kent Hutchison

M144. Functional Connectivity of the Intraparietal Sulcus Is Affected by Both Copy Number and Sequence Variation of the Williams Syndrome Gene LIMK1

> <u>Michael D. Gregory</u>, J. Shane. Kippenhan, Carolyn Mervis, Melanie Sottile, Jasmin Czarapata, Katherine Roe, Ena Xiao, Yunxia Tong, Bhaskar S. Kolachana, Daniel R. Weinberger, Venkata S. Mattay, Karen F. Berman

M145. In Anorexia Nervosa, Anxious Rumination Is Grounded in the Activation of Abnormal Interoceptive Insular Cortex

<u>William K. Simmons</u>, Kara Kerr, Scott Moseman, Jason Avery, Jennifer Dobson, Kaiping Burrows, Nancy Zucker

M146. First HDAC PET Radiotracer Ready for Human Translation

<u>Changning Wang</u>, Frederick A. Schroeder, Edward Holson, Stephen J. Haggarty, Jacob M. Hooker

M147. Cannabis Use Is Associated with Nucleus Accumbens and Amygdala Abnormalities in Young Adult Recreational Users

> Jodi M. Gilman, John Kuster, Sang Lee, Myung Joo Lee, Byoung Woo Kim, Nikos Makris, Andre van der Kouwe, Anne Blood, Hans C. Breiter

M148. Predictive Classification of Pediatric Bipolar Disorder Morphometric Features of the Amygdala

Benson Mwangi, Danielle Spiker, Giovana B. Zunta-Soares, Jair C. Soares

M149. Suicide Risk and Mood Regulation Deficits: Emotional Reactivity as an Exploratory Pathway

<u>Rebecca Bernert</u>, Melanie Hom, Madeleine Goodkind, Kathy Peng, Desmond Oathes, Michelle Primeau, Amit Etkin

M150. Relation of Diet, Exercise, and Body Mass Index to a Brain Imaging Biomarker of Plaques and Tangles in Non-demented Middle-aged and Older Adults

David A. Merrill, Prabha Siddarth, Cyrus A. Raji, Gary Small

M151. A Twin Study Identifying the Origin of Abnormal Automatic Responses to Threat Related Stimuli in PTSD

> <u>F. Caroline Davis</u>, Michael B. VanElzakker, Lindsay K. Staples, Natasha B. Lasko, Scott Orr, Roger K. Pitman, Lisa M. Shin

M152. Brain Morphology in Adolescents and Young Adults at High and Low Risk for Alcohol Dependence: Separating Cause and Consequence

Shirley Y. Hill, Wang Shuhui, Howard Carter, Robert Terwillinger

M153. Unsupervised Identification of Population Patterns in High-dimensional Multimodal Neuroimaging Scans: A Data-driven Machine Learning Approach

Benson Mwangi, Khader M. Hasan, Jair C. Soares

M154. Electrophysiological and Anatomical Evidence for Two Distinct but Interacting Neural Circuit Abnormalities in the Auditory Cortex in Schizophrenia

> <u>Yoji Hirano</u>, Naoya Oribe, Shigenobu Kanba, Toshiaki Onitsuka, Taiga Hosokawa, Martha Shenton, Robert W. McCarley, Kevin M. Spencer

M155. Dopaminergic Activity and Altered Insula Response to Sweet Taste Processing in Anorexia Nervosa

> <u>Ursula F. Bailer</u>, Julie L. Fudge, Julie C. Price, Carolyn C. Meltzer, Angela Wagner, Chester A. Mathis, Walter Kaye

M156. Reduced Prefrontal Gamma Band Power in Patients with Schizophrenia Studied with MEG during Working Memory

Dani Rubinstein, Daniel P. Eisenberg, Frederick W. Carver, Daniel R. Weinberger, Richard Coppola, Karen F. Berman

M157. Resting State Functional Connectivity of the Habenula as a Biomarker of Depression and Treatment Response

Philip Baldwin, Humsini Viswanath, Kenia Velasquez, Sanjay J. Mathew, <u>Ramiro Salas</u>

M158. Lower Limbic System mGluR5 Availability in Cocaine Dependent Subjects: A High-resolution PET [11C]ABP688 Study

> <u>Michele Milella</u>, Laura Marengo, Kevin Larcher, Aryandokht Fotros, Alain Dagher, Pedro Rosa-Neto, Chawki Benkelfat, Marco Leyton

M159. Prefrontal Response to Visual Drug Cues Predicts Adherence to Extended-release Injectable Naltrexone in Heroin-dependent Individuals

> <u>An-Li Wang</u>, Kanchana Jagannathan, Igor Elman, George E. Woody, Shira J. Blady, Emily D. Dowd, James W. Cornish, Anna R. Childress, Charles P. O'Brien, Daniel D. Langleben

M160. Amygdala Activation to Emotion Stimuli as a Predictor of Treatment Outcomes in Major Depressive Disorder: The International Study to Predict Optimized Treatment in Depression (iSPOT-D)

Leanne Williams, Mayuresh Korgaonkar, Stuart Grieve, Amit Etkin

M161. Relationship between Central Mu-opioid System Response and Affect to Feeding Is Altered by the Pathophysiology of Obesity

<u>Paul Burghardt</u>, Amy Rothberg, Kate Dykhuis, Charles Burant, Jon-Kar Zubieta

M162. Development of Cingulum Bundle White Matter in Pediatric Obsessive Compulsive Disorder

<u>Kate D. Fitzgerald</u>, Elyse Reamer, Yanni Liu, Robert C. Welsh, Stephan Taylor

M163. Prenatal Vigabatrin Exposure Attenuates Naloxone-induced Withdrawal Behaviors in Neonates

> Jakub Kaczmarzyk, Giovanni Santoro, Sandy Scherrer, Stergiani Agorastos, Jonathan D. Brodie, Joseph Carrion, Krishna Patel, Rebecca Silverman, Michelle Choi, Christina Veith, Danielle Mullin, <u>Stephen L. Dewey</u>

> > 188

# M164. Group ICA Analysis of Smokers and Controls

Philip Baldwin, Ramiro Salas

M165. Resting State Functional Connectivity of the Dorsal Attention, Frontoparietal, Cingulo-opercular, and Default Mode Networks in Children with a History of Depression and/or an Anxiety Disorder

<u>Chad Sylvester</u>, Deanna M. Barch, Jonathan Power, Michael Gaffrey, Bradley Schlaggar, Joan Luby

M166. Longitudinal fMRI Study of Quetiapine in Bipolar Mania

<u>Caleb Adler</u>, Andrew Davis, Melissa DelBello, Wade Weber, James Eliassen, Thomas Blom, Jeffrey Welge, David Fleck, Strakowski Stephen

M167. Increased Glutamate in the Dorsal Anterior Cingulate Cortex Is Associated with Anxiety Symptom Domain in MDD with High Inflammation

Ebrahim Haroon, Bobbi Woolwine, Xiangchuan Chen, Xiaoping Hu, Andrew H. Miller

M168. Intravenous Morphine Self-administration Reduces In Vivo Regional Glucose Utilization (18FDG-PET) and Accelerates Fear Extinction Behavior in Rats

Thien Le, Reed Selwyn, Robert Ursano, Kwang Choi

M169. Brain Diffusion Tensor Imaging and 31P Spectroscopy of In Vivo Tau P301L Toxicity Mechanisms

Naruhiko Sahara, Pablo Perez, Yan Ren, Huadong Zeng, Jada Lewis, <u>Marcelo Febo</u>

M170. In Vivo Diffusion Tensor Imaging Evidence for Reversible White Matter Microstructural Integrity Disruption with Binge but Not Chronic Ethanol Exposure

Natalie M. Zahr, Edith V. Sullivan, Adolf Pfefferbaum

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#### **Poster Session I—Monday**

M171. Differentiating Neural Networks Underlying Risk for Depression in Youth

Manpreet K. Singh, Ryan G. Kelley, Meghan Howe, Ian Gotlib, Kiki Chang

M172. Pre-symptomatic Functional Brain Changes in PS1 E280A Mutation Carriers Compared to Other Biomarkers: Pilot Data from the Alzheimer's Prevention Initiative Biomarker Project

> <u>Pierre Tariot</u>, Adam S. Fleisher, Kewei Chen, Jessica Langbaum, Auttawut Roontiva, Pradeep Thiyyagura, Ji Luo, Napatkamon Ayutyanont, Stephanie A. Parks, Francisco Lopera, Eric Reiman, Xiaofen Liu, Wendy Lee

M173. Multiscale Computer Modeling of Antipsychotic Targets: ER Parameters Modulate Calcium Wave Propagation

Mohamed A. Sherif, Robert McDougal, Samuel Neymotin, Michael Hines, William Lytton

M174. Association of Clinical Variables and Calf Arterial Compliance in Veterans with Psychiatric Diagnoses

Maju Koola, John Sorkin, William Brown, Bruce Cuthbert, Jeffrey Hollis, Ngoc-Anh Le, Jeffrey Raines, Erica Duncan

M175. High Variability and Lack of Change on the ADAS-Cog: Placebo Analyses of the CODR Database

Danielle Popp, Lori M. Garzio, Peter Boehm, Christopher Randolph

M176. Comparative Trials of Long-acting Injectable vs. Daily Oral Antipsychotic Treatment in Schizophrenia: Do Pragmatic vs. Explanatory Study Designs Matter?

Cynthia A. Bossie, Larry Alphs

M177. Mapping of the Brain-wide of the Glymphatic Waste Removal Pathway by MRI and PET Imaging

<u>Helene Benveniste</u>, Joanna Fowler, Paul Vaska, Maiken Nedergaard, Hedok Lee, Gene-Jack Wang, Nora D. Volkow

M178. Developing a Smart Phone App to Monitor Mood, Social Rhythms, Sleep and Social Activity: Technology to Support Effective Management of Bipolar Disorder

<u>Ellen Frank</u>, Mark Matthews, Tanzeem Choudhury, Stephen Voida, Saeed Abdullah

M179. Cognitive-affective Remediation Training Intervention in Anxiety and Depression

Anett Gyurak, James Gross, Larry Chan, Amit Etkin

M180. DREADDs in Drosophila: Pharmacogenetic Control of Neurons and Behavior in the Fly

<u>Charles D. Nichols</u>, Jaime Becnel, Oralee Johnson, Zana Majeed, Vi Tran, Bangning Yu, Bryan L. Roth, Robin L. Cooper, Edmund K. Kerut

M181. Forecasting Non-remitting PTSD Symptom Trajectory by Advanced Modeling Methods

> Isaac Galatzer-Levy, Karen-Inge Karstoft, Sara Freedman, Yael Ankri, Moran Gilad, Alexander Statnikov, <u>Arieh Y. Shalev</u>

- M182. Withdrawn
- M183. Olfactory Identification Deficits Predict Response to Cholinesterase Inhibitors in Patients with Mild Cognitive Impairment

Davangere P. Devanand, Gregory Pelton, Howard Andrews, Bruce Levin

M184. ALKS 5461, a Novel Opioid Modulator, Produces Remission and Decreases Core Depressive Symptoms and Anhedonia as an Adjunctive Treatment: A Sequential Parallel Comparison Design Trial in Inadequate Responders to Antidepressants

> Marlene P. Freeman, Randall Marshall, Asli Memisoglu, Richard Leigh-Pemberton, Elliot W. Ehrich, Michael E. Thase, Madhukar Trivedi, J. Alexander Bodkin, Maurizio Fava

M185. MicroRNA Dysregulation in Cerebrospinal Fluid in Patients with Schizophrenia

Juan A. Gallego, Todd Lencz, Brian Cantley, Anil Malhotra

M186. Pharmacometabolomics of Atypical Antipsychotics in Bipolar Disorder: An Untargeted Approach

Kyle Burghardt, Vicki Ellingrod

M187. Meta-analysis of the Discriminative Validity of Caregiver, Teacher, and Youth Checklists for Assessing Pediatric Bipolar Disorder

> Eric Youngstrom, Jacquelynne Genzlinger, Ericka McKinney, Greg Egerton, Anna Van Meter

M188. Development of a Lab-on-a-Chip Biosensor for Clozapine Monitoring

<u>Deanna L. Kelly</u>, Hadar Ben-Yoav, Veronika Stock, Thomas Winkler, Gregory Payne, Sheryl Chocron, Eunkyoung Kim, Gopal Vyas, Raymond Love, Heidi J. Wehring, Kelli M. Sullivan, Stephanie Feldman, Fang Liu, Robert P. McMahon, Reza Ghodssi

M189. Print 'Close the Use of a Novel Urine Drug Monitoring Test to Help Assess How Well Clinicians Predict Antipsychotic Medication Nonadherence

Matthew Keats, Harry Leider, Kathryn Bronstein, Mary Anne Lang

M190. Functional Capacity Assessment in Older Adults

Sara J. Czaja, Philip D. Harvey, David Loewenstein

M191. Estimating Endogenous Dopamine Levels at D2 and D3 Receptors in Humans Using the Agonist Radiotracer [11C]-(+)-PHNO

<u>Ariel Graff</u>, Fernando Caravaggio, Shinichiro Nakajima, Philip Gerretsen, David Mamo, Gary Remington, Alan A. Wilson

M192. Central 5-HT4 Receptor Binding as Biomarker of Serotonergic Tonus in Humans: A [11C]SB207145 PET Study

> Mette Haahr, Patrick Fisher, Christian Gaden, Vibe Frokjaer, Brenda McMahon, Karine Madsen, Wim Baaré, Szabolz Lehel, Anne Norremolle, Eugenii Alfredovich. Rabiner, <u>Gitte M. Knudsen</u>

M193. A Mixture Model Estimate of Time to Antidepressant Drug-effect in Association with Covariates Using the STAR\*D Sample

<u>Yin Yao</u>, Mengyuan Xu, Eleanor Murphy, Harold Wang, Francis J. McMahon

M194. Responses to Blocked Goal Attainment in Preschoolers at Risk for Bipolar Disorder

> <u>Wan-Ling Tseng</u>, Christen M. Deveney, Amanda E. Guyer, Jennifer Yi, Kimberly Espy, Lauren Wakschlag, Kenneth Towbin, Ellen Leibenluft, Melissa A. Brotman

M195. Assessing Effort-based Decision-making in Schizophrenia with Two Novel Behavioral Paradigms

> <u>Felice Reddy</u>, William Horan, Jonathan K. Wynn, Patricia Corey-Lisle, Gregory Maglinte, Deanna M. Barch, Robert W. Buchanan, James Gold, Jared Young, Michael F. Green

M196. MCI and Everyday Task Performance

Samir Sabbag, Sara J. Czaja, Philip Harvey

M197. Differential Prefrontal Control of Brainstem Neuromodulatory Systems in Depression-related Behavior

Melissa R. Warden, Emily Ferenczi, Karl Deisseroth

M198. Addiction-related Genes in Gambling Disorders: New Insights from Parallel Human and Pre-clinical Models

Daniela S. S. Lobo, Lily Aleksandrova, Jo Knight, David Casey, Nady el-Guebaly, Jose Nobrega, James L. Kennedy

M199. Effects of Combined Adrenoreceptor Antagonist Treatment with Prazosin and Propranolol on Alcohol Drinking in Humans and Rodents

Murray A. Raskind, Janice C. Froehlich, Elaine R. Peskind, Dennis D. Rasmussen

- M200. Postural Sway Abnormalities in Chronic Schizophrenia Brent G. Nelson, Kelvin O. Lim
- M201. Enhancement of rTMS Neuromodulatory Effects with Novel Waveforms Demonstrated via Controllable Pulse Parameter TMS (cTMS)

<u>Stefan M. Goetz</u>, Bruce Luber, Sarah Lisanby, Cassie I. Kozyrkov, Warren M. Grill, Angel V. Peterchev

M202. Computerized Cognitive Remediation for Geriatric Depression

Sarah Shizuko Morimoto, Bruce E. Wexler, Willie Hu, George S. Alexopoulos

M203. The Effect of Real Time fMRI Neurofeedback on Food and Cigarette Craving

Luke Stoeckel

M204. Orion Bionetworks: Causal Modeling Using Network Ensemble Simulations of Clinical, Imaging and Genetic Data to Predict Multiple Sclerosis

Magali Haas, Iya Khalil, Phil De Jager

M205. Specific Elevation of βCaMKII in the Lateral Habenula Lead to Core Symptoms of Depression

Hailan Hu, <u>Henn Fritz</u>, Kun Li, Tai Zhou, Zhongfei Yang, Lujian Liao, Roberto Malinow, John R. Yates III

M206. Mechanisms of Ventral Pallidal Enkephalin Regulation in Cocaine Addiction

Yonatan M. Kupchik, Peter W. Kalivas

M207. Abnormalities in Striato-pallidal-thalamic Surface Morphology as an Endophenotype for Obsessive-compulsive Disorder

Shaw Phillip, Wendy Sharp, Judith L. Rapoport

M208. High Blood Cytokine Levels Are Linked to Decreased Verbal Fluency and Broca's Area Volume Reduction in Schizophrenia

> <u>Thomas W. Weickert</u>, Stu Fillman, Rhoshel Lenroot, Jason Bruggemann, Maryann O'Donnell, Stanley V. Catts, Cynthia S. Weickert

M209. 5-HT3 Receptors Are Involved in the Mechanism of Action of the New Antidepressant Drug Vortioxetine

Francesc Artigas, Maurizio Riga, Pau Celada, Connie Sanchez

M210. Implications of the Human Mu Opioid Receptor (OPRM1 A118G) Polymorphism in the Neurobiology of Stress and Placebo Responses

> Marta Pecina, Tiffany Love, Colin A. Hodgkinson, David Goldman, Christian Stohler, Jon-Kar Zubieta

- M211. Pattern Classification Accuracy to Taste Stimulation in Eating Disorders <u>Guido K.W. Frank</u>, Carrie Keffler, Megan Shott
- M212. Whole-brain Dynamics Are Shifted in Animals with Learned Helplessness

Martine Mirrione, Bo Li, Stephen Shea, Henn Fritz

M213. Sleep Misperception in Bipolar Disorder: Are Our Patients Getting 7 Hours of Sleep?

> Erika F.H. Saunders, Scott Seaman , Julio Fernandez-Mendoza, Andrew Jacobs, Alan J. Gelenberg

M214. The Clinical Relevance of Neural Network Dynamics for Bipolar Disorder

Sophia Frangou, Danai Dima

M215. Early Adverse Life Events: Interaction with Glucocorticoid [NR3C1] and Proinflammatory Cytokine [IL-1 β] Polymorphisms to Influence Gray Matter Variations in Females with and without Chronic Abdominal Pain

> Arpana Gupta, Emeran A. Mayer, Mariam Bonyadi, Jennifer Labus, Cody Ashe-McNalley, Nuwanthi Heendeniya, Lin Chang, Lisa Kilpatrick

M216. Amygdalar Projections to Basilar Dendrites of mPFC Pyramidal Neurons Mediate CRF-induced EPSCs that Are Enhanced by Ketamine

Rong-Jian Liu, Kristie Ota, Ronald S. Duman, George Aghajanian

M217. Identification of Signaling Cascades Regulating the Extinction and Reconsolidation of Cocaine-associated Memories Using Phosphoproteomics

> Jane R. Taylor, Thomas Abbott, Erol E. Gulcicek, Kathryn Stone, Lisa Chung, Christopher Colangelo, <u>Mary M. Torregrossa</u>

M218. Role of a Beta-2 Adrenergic Receptor-regulated CRF-releasing Pathway from the BNST to the VTA in Stress-induced Relapse of Cocaine Use

John R. Mantsch, Oliver Vranjkovic, Jordan M. Blacktop

M219. Brain Glucose Metabolism Predicts Fear Extinction Recall and Global Functioning in Trauma-exposed Populations with and without PTSD

<u>Marie-France Marin</u>, Huijin Song, Lindsay K. Staples, Michael B. VanElzakker, Natasha B. Lasko, Roger K. Pitman, Lisa M. Shin, Mohammed R. Milad

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### **Poster Session I—Monday**

M220. Imaging Amino Acid Neurotransmitter Responses to a Single Subanesthetic Dose of Ketamine in Major Depressive Disorder Using Proton Magnetic Resonance Spectroscopy

> <u>Matthew Milak</u>, Stephanie Mulhern, Caitlin Proper, Amy Parter, Lawrence S. Kegeles, Todd Ogden, Xiangling Mao, Carolyn Rodriguez, Maria Oquendo, Raymond Suckow, Thomas Cooper, Dikoma Shungu, J. John Mann

M221. The Central Nucleus of the Amygdala Is Required for Habitual Cocaine Seeking through Functional Connectivity with the Dorsolateral Striatum

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

M222. Dopamine DREADDs: Chemicogenetic Control of VTA Dopamine Activity during Reinstatement of Cocaine Seeking

Stephen V. Mahler, Brittney M. Cox, Gary Aston-Jones

M223. Dorsal and Ventral Prefrontal Neuronal Activity and Cocaine Seeking: More Complex than Thought

David Moorman, Gary Aston-Jones

M224. Cytokine and Chemokine Profiling of Plasma and CSF Identifies the MCP-4/MCP-1 Ratio as a Novel Candidate Plasma Biomarker for Chronic Post-traumatic Stress Disorder

> <u>Harvey B. Pollard</u>, Clifton L. Dalgard, Catherine Jozwik, Meera Srivastava, Ofer Eidelman, Robert Ursano, David Jacobowitz, Omer Bonne

M225. Biomarkers Differentiating Major Depressive Disorder Subtypes

<u>Husseini K. Manji</u>, Lynn Yieh, Jieping Ye, Yashu Liu, Tao Yang, Michael Farnum, Xiang Yao, Willem Talloen, Thomas Steckler, Pim Drinkenberg, Pieter J. Peeters, Vaibhav Narayan, Gayle Wittenberg

M226. Contribution of a Mesocorticolimbic Subcircuit to Drug Contextinduced Reinstatement of Cocaine-seeking Behavior in Rats

> <u>Rita A. Fuchs</u>, Heather C. Lasseter, Xiaohu Xie, Amy A. Arguello, Audrey M. Wells, Matthew A. Hodges

M227. The Involvement of the Serotonergic System in the Nucleus Accumbens Shell on EtOH-seeking: Role of 5HT7 Receptors and Response to Conditioned Cues

<u>Gerald A. Deehan</u>, Sheketha R. Hauser, Eric A. Engleman, Jessica A. Wilden, William Truitt, William J. McBride, Zachary A. Rodd

M228. Maltreated Preschoolers: The Association of Stress Exposure with Adrenocortical and Behavioral Outcomes

<u>Audrey R. Tyrka</u>, Stephanie H. Parade, Nicole M. Eslinger, Brittney Shillan, Ashley Clement, Rebecca Berger, Susan Dickstein, Ronald Seifer

M229. Contributions of Ventral Dopamine Target Neurons to Risk-seeking Decisions in a Macaque Model of Compulsive Gambling

Brianna Sleezer, Benjamin Hayden

M230. Frontal and Subcortical Pathways Provide a Basis for Segmenting the Cingulum Bundle: Implications for Understanding the Default Mode Network, Diffusion Imaging, and Surgical Targets for Psychiatric Disorders

Sarah R. Heilbronner, Suzanne Haber

M231. Rules Prefrontal Pathways Use in the Anterior Limb of the Internal Capsule: Implications for Neuroimaging and Deep Brain Stimulation

Ziad Safadi, Suzanne Haber

M232. Exploring Side Effects Similarity as a Novel Approach for Inferring Shared Mechanisms and Targets among Antidepressant and Antiinflammatory Drugs

Vaibhav Narayan, Yu Sun, Gayle Wittenberg, Michael Farnum

M233. Functional Network Connectivity Dynamics in Schizophrenia and Bipolar Disorder

<u>Vincent Calhoun</u>, Barnaly Rashid, Eswar Damaraju, Godfrey Pearlson

M234. Withdrawn

M235. Treatment of Dopaminergic Dysfunction in Schizophrenia Using Nondopaminergic Mechanisms: A Computational Modeling Approach

Peter J. Siekmeier, David vanMaanen

M236. Retinoid-related Orphan Receptor Alpha: A Novel Candidate Gene for Psychiatric Disease

> Joseph I. Friedman, Sander Markx, Terry Vrijenhoek, Ronald Kim, Joris A. Veltman, Arthur Mikhno, James R. Moeller, Mala Ananth, David K. Leung, Han G. Brunner, Vincent Giguere, Panayotis K. Thanos

M237. Early Adverse Life Events: Interactions with Corticotropin Releasing Hormone Receptor 1 and Progestoerone Receptor Polymorphisms Healthy Controls and Patients with Chronic Abdominal Pain

Lisa Kilpatrick, Arpana Gupta, Nuwanthi Heendeniya, Jennifer Labus, <u>Emeran A. Mayer</u>

M238. Vocational Outcome in Patients with Psychotic and Affective Disorders: A Nation-wide, Historical-prospective Study

<u>Mark Weiser</u>, Ori Kapara, Nomi Werbeloff, Rinat Yoffe, Michael Davidson

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# Poster Session II – Tuesday, December 10, 2013



Advocacy Affiliate – Cure Alliance for Mental Illness Research Parity for Mental Illness

Robin Cunningham, Hakon Heimer

T1. 2013 Report of the Membership Advisory Task Force

Linda Carpenter, Lisa Monteggia, Margaret Haney, Katherine Burdick, Jennifer Bartz, Elisabeth Binder, Paul Holtzheimer, Amit Etkin, Erica Forbes, Marlene Freeman, Thomas Schulze, Christina Barr, Gregory Light, Vaishali Bakshi, Raymond Cho, Cynthia Crawford, Philip Szeszko

T2. Effects of CFA-induced Chronic Inflammatory Pain on Opioid Selfadministration and Accumbal Dopamine Release in Heroin Dependent Rats

Jose Moron-Concepcion, Lucia Hipolito-Cubedo

T3. Contributions of Glial Glutamate Transport and NMDA Receptors in Nicotine Relapse

<u>Cassandra Gipson</u>, Yonatan M. Kupchik, Neringa Stankeviciute, Peter W. Kalivas

T4. Behavioral and Molecular Consequences of GAD1 Downregulation in Cannabinoid Receptor 1 Expressing Interneurons

Jacquelyn Brown, Szatmar Horvath, Krassimira Garbett, Monica Everheart, Karoly Mirnics

T5. Modeling Fall Propensity in Parkinson's Disease: Deficits in the Attentional Control of Complex Movements in Rats with Cortical-cholinergic and Striatal-dopaminergic Deafferentation

Martin Sarter, Aaron Kucinski

T6. Different Adolescent Traumatic Stress Pre-exposures Differentially Modify Adulthood Predator Stress Responses in Rats

<u>Nicole L.T. Moore</u>, Daniel E. Altman, Sangeeta Gauchan, Raymond F. Genovese

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#### Poster Session II—Tuesday

T7. Genetic Interaction between Integrin β3 (Itgb3) and SerotoninTransporter (Slc6a4) Modifies Depressive-like Behaviors in the Mouse

Seth Varney, Alonzo Whyte, Tammy Jessen, Ana Carneiro

T8. Dopamine-independent Motor Control and Hyperactivity Involving Acetylcholine Systems

Kazutaka Ikeda, Yoko Hagino, Shinya Kasai

T9. A New Model for Studying Effects of Witnessing Traumatic Events in Rats

Samina Salim, Gaurav Patki, Naimesh Solanki, Farida Allam, Amber Ansari

T10. Identification of Early Risk for Substance Use: fMRI Responses to Cocaine-associated Cues in Juvenile Rats

Steven Lowen, Michael Rohan, Britta S. Thompson, Kai Sonntag, Susan L. Andersen

T11. Interaction between BDNF and Social Environment in Brain Physiology and Behavior

Robert Schloesser, Dennisse Jimenez, Julia Hill, Keri Martinowich

T12. Early Life FGF2 Treatment Alters Vasopressin and Oxytocin Gene Expression in Animals that Differ in Their Response to Novelty

<u>Cortney Turner</u>, Pamela Maras, Yoav Litvin, Stanley J. Watson, Bruce S. McEwen, Huda Akil

T13. Attitudes of Children and Adolescents and Their Caregivers Towards Long-acting Injectable Antipsychotics in a Cohort of Youth Initiating Oral Antipsychotic Treatment

> <u>Christoph U. Correll</u>, Owen Muir, Aseel Al-Jadiri, Sandeep Kapoor, Morgan Carella, Eva Sheridan, Lisa David, John Kane
T14. Comorbidity of PTSD and Alcoholism: A Rat Model of PTSD Leads to Escalated Ethanol Consumption

Jenica Tapocik, Jesse R. Schank, Cheryl Mayo, Courtney King, Jim Koenig, Markus Heilig, Greg I. Elmer

T15. Mouse Model of Chromosome 15q13.3 Microdeletion Syndrome Demonstrates Features of Autism Spectrum Disorder

> <u>Jeffrey Kogan</u>, Adam Gross, Rick Shin, Qian Chen, Noah Walton, Carrie Heusner, Amy Lin, Sosuke Miyoshi, Shintaro Nishimura, Shinichi Miyake, Katsunori Tajinda, Kouichi Tamura, Mickey Matsumoto

T16. Distinct Roles of PKC Signaling at Direct and Indirect Pathway Medium Spiny Neurons during Reinstatement of Cocaine-seeking

<u>Pavel I. Ortinski</u>, Lisa A. Briand, R. Christopher Pierce, Heath D. Schmidt

T17. Amygdala-ventral Pallidum Pathway Decreases Dopamine Activity Following Chronic Mild Stress in Rats

Chun-hui Chang, Anthony A. Grace

T18. Roles of Glucocorticoids in a Trajectory from Adolescent Social Stress to Adult Behavior

Minae Niwa, Akira Sawa

T19. High Traumatic Stress Reactivity Alters Behavior and Corticotropinreleasing Factor-1 (CRF1Rs) in Prefrontal Cortex-amygdala Circuitry

Nicholas W. Gilpin

T20. High-throughput Behavior-based Neuroactive Drug Discovery in Zebrafish

David Kokel

T21. Cortical Synaptic Alterations and Pharmacologic Rescue of Behavioral Changes in a Mouse Model of Bipolar Disorder with Conditional Forebrain Knockout of Ankyrin-G

Shanshan Zhu, Solange P. Brown, Vann Bennett, Mikhail V. Pletnikov, <u>Christopher A. Ross</u>

T22. Activity-based Anorexia in the Rat Induces Reward-related Alterations in Enkephalin Gene Expression and Dopamine Release in the Nucleus Accumbens

> <u>Nicole M. Avena</u>, Susan Murray, Nicole Barbarich-Marstellar, Pedro Rada

T23. Repeated Ketamine Exposure during Adolescence Produces Long Lasting Stress Resistance in Adulthood

> Eric M. Parise, Lyonna F. Alcantara, Brandon L. Warren, <u>Carlos A.</u> <u>Bolanos-Guzman</u>

T24. Susceptibility to Chronic Social Defeat Stress Increases Morphine Reward

Megan Kechner, Michelle Mazei-Robison

T25. The Contribution of Adult Hippocampal Neurogenesis to Fear Memory Generalization

Mazen A. Kheirbek, Liam J. Drew, Elizabeth Balough, Christine A. Denny, Rene Hen

T26. Individual Differences in Instrumental Performance in Naïve Rats Predict Distinctive Responses to Chronic Stress

Shigenobu Toda, Yoshio Iguchi, Yoshio Minabe

T27. Pay Attention: Modelling the Inattentive and Impulsive Subtypes of Adult ADHD in the Rat - Using the 5-Choice Continuous Performance Task (5C-CPT)

Anneka Tomlinson, Joanna Neill

T28. Lesions of the Basolateral Amygdala Induce Elevated Risk-taking in Rats

Caitlin Orsini, Barry Setlow

T29. Loss Estrogen-related Receptor Alpha Activity Affects Behaviors Related to Eating Disorders in Mice

Huxing Cui, Michael L. Lutter

T30. Chronic Phenytoin Administration Prevents Single Prolonged Stress Induced Extinction Retention Deficits and Glucocorticoid Upregulation

<u>Sophie A. George</u>, Dayan K. Knox, Mariana Rodriguez, John Riley, Israel Liberzon

T31. Independent Effects of Lps and Social Isolation on Forced Swim Behavior in Female Mice

> <u>Cristina L. Sanchez</u>, Nicole L. Schramm-Sapyta, Cynthia M. Kuhn, Florian Daniel. Zepf

- T32. Hippocampal-prefrontal BDNF Circuits in Fear Extinction Luis E. Rosas-Vidal, Fabricio H. Do Monte, <u>Gregory J. Quirk</u>
- T33. Adolescent Cannabinoid Treatment Leads to Persistent Increase in Frontostriatal CB1 Expression Associated with Upregulation of Class I HDACs

<u>Subroto Ghose</u>, Hersh Trivedi, Kelly Gleason, Marcus Shanks, Shari Birnbaum

T34. Determinants of Conditioned Reinforcing Effectiveness: Implications for Relapse to Cocaine-seeking

Gregory T. Collins, Charles P. France

T35. Ketamine Alters Socially-evoked Activity in the Amygdala

Takuma Mihara, Rosanna Sobota, Robert Lin, Robert Featherstone, Steven J. Siegel

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### **Poster Session II—Tuesday**

T36. Modulation of Fear-related Behaviours by Prefrontal Cortical Gabaergic Transmission and Its Relevance to Schizophrenia

Stan B. Floresco, Patrick B. Piantadosi

T37. Central CRTH2/GPR44, a Second Prostaglandin D2 Receptor, Mediates Emotional Impairment in the Lipopolysaccharide and Tumor-induced Sickness Behavior Model

> <u>Hitoshi Hashimoto</u>, Ryota Haba, Norihito Shintani, Yusuke Onaka, Hiroyuki Hirai, Kin-ya Nagata, Masataka Nakamura, Akemichi Baba

T38. Timing May Matter: Vulnerability and Resilience to Acute Trauma Vary According to the Circadian Phase at Which Exposure Occurs

Hagit Cohen, Shlomi Cohen, Aleksander Mathé, Joseph Zohar

T39. Variations in a Stress Paradigm on Top of Early Interference with the Expression of Multiple Genes Leads to Disparate Behavioral Responses in Rats - A Possible Role for Essential Amino Acids

Eyal Asor, Avi Avital, Ehud Klein, Dorit Ben-Shachar

T40. Neural Disconnectivity and Loss of Connections Symmetry in Brains of Mice Knockout for the Neurodevelopmental Gene Ahil

<u>Amit Lotan</u>, Tzuri Lifschytz, Omer Lory, Gadi Goelman, Bernard Lerer

T41. Epigenetic Regulation of Dopamine D2 Receptor in the Core of the Nucleus Accumbens Contributes to Addiction Liability

<u>Shelly B. Flagel</u>, Sraboni Chaudhury, Maria Waselus, Stanley J. Watson, Huda Akil

T42. Positive Allosteric Modulation of mGluR5 Reverses the Akt Signaling Deficits in Serine Racemase Knockout Mice, a Genetic Model of Schizophrenia Due to NMDA Receptor Hypofunction

> <u>Darrick T. Balu</u>, Shunsuke Takagi, Thomas Steckler, Jose Manuel Bartolome, Carrie K. Jones, Jeffrey Conn, Joseph T. Coyle

T43. Age-related Sperm DNA Methylation Changes Are Transmitted to Offspring and Associated with Abnormal Behavior and Dysregulated Gene Expression

> <u>Maria H. Milekic</u>, Yurong Xin, Anne O'Donnell, Victoria Fatemeh. Haghighi, Jay A. Gingrich, John Edwards, Timothy Bestor

T44. Single Prolonged Stress Decreases Sign-tracking Conditioned Responses and Attenuates Cue-induced Reinstatement of Cocaine-seeking Behavior

Christopher Fitzpatrick, Terry E. Robinson, Jonathan D. Morrow

T45. Empathic Fear Responses in Mice Are Triggered by Recognition of a Shared Experience

Jeff Sanders, Mark Mayford, Dilip V. Jeste

T46. Selective Removal of Parvalbumin Interneurons from Striatal Networks to Model the Pathophysiology of Tourette Syndrome

Meiyu Xu, Vladimir Pogorelov, Lina Li, Christopher Pittenger

T47. Effects of Prenatal and Postnatal Hypoxia on Brain Derived Neurotrophic Factor Signaling in Mice

Anilkumar Pillai, Kristy R. Howell, Sarah Mehta

T48. Cocaine-induced Adaptations in Alpha2delta-1 Calcium Channel Subunit in Nucleus Accumbens Contribute to Cocaine-induced Drug Seeking

<u>Sade Spencer</u>, Robyn M. Brown, Gabriel Quintero, Yonatan M. Kupchik, Kathryn Reissner, Peter W. Kalivas

T49. Chronic Prenatal Kynurenine Elevation in Rats: A Naturalistic Model of Schizophrenia with Biochemical Abnormalities and Deficits in Hippocampal-mediated Learning and Memory

Robert Schwarcz, Ana Pocivavsek, Greg I. Elmer, John Bruno

T50. Evidence for a Novel Role of Acid Sensing Ion Channel, Asic1a in the Molecular Biology of Mood and Anxiety

James R. Shoblock, Natalie Welty, Yi Liu, Changlu Liu, Timothy Lovenberg, Guang Chen

T51. Repeated Administration of an Acetylcholinesterase Inhibitor Attenuates Nicotine Taking in Rats

Adrian Arreola, Blake Kimmey, Laura Rupprecht, Alycia Lee, Matthew Hayes, <u>Heath D. Schmidt</u>

- T52. Withdrawn
- T53. Effects of Pharmacogenetic Manipulation of the Nucleus Accumbens on Neuronal Activity and Alcohol-related Behaviors

<u>Angela Ozburn</u>, Ryan Logan, Puja Parekh, Jake Bosin, Colleen A. McClung

T54. Re-exposure to Nicotine After Chronic Nicotine Exposure and Withdrawal Potentiates Reward Responsiveness in Rats: Implications for Relapse

<u>Andre Der-Avakian</u>, Manoranjan S. D'Souza, Diego A. Pizzagalli, Athina Markou

- T55. Effects of Maternal Separation on Depressive- and Anxiety- Like Behaviors and Cardiovascular Function in Stress-susceptible Rats Ilan A. Kerman, Samir Rana, Nateka Jackson, Phyllis C. Pugh
- T56. Spontaneous Nicotine Withdrawal Enhanced Anxiety-like Behavior in the Fear-potentiated Startle Procedure in Rats

Xia Li, Athina Markou, Victoria Risbrough

T57. Long-term Modulation of Memory and Emotion after a Systemic Inflammatory Event

Natalie Tronson, Ian Speirs

T58. Selective Enhancement of Cue-induced Motivation in Obesity Prone vs. Resistant Rats Is Accompanied by Sensitization to Cocaine and Increased Striatal AMPA Receptor Expression

Carrie R. Ferrario, Cameron Nobile, Michael J.F. Robinson, Kent Berridge

- T59. Withdrawn
- T60. Ultra-high Magnetic Field (9.4 Tesla) Magnetic Resonance Imaging Reveals Neuroanatomical and Neurochemical Homologies between Schizophrenia and the Serine Racemase Knockout Mouse

<u>Matthew D. Puhl</u>, Dionyssios Mintzopoulos, J. Eric Jensen, Timothy E. Gillis, Marc J. Kaufman, Joseph T. Coyle

T61. Loss of GFAP-positive Astrocytes in the Nucleus Accumbens Following Cocaine Self-administration and Extinction Is Associated with Increased IL-6 Expression

Phuong K. Tran, Heather A. Boger, Sade Spencer, Michael D. Scofield, Peter W. Kalivas, <u>Kathryn J. Reissner</u>

T62. Evaluation of an Electronic Information System to Enhance Practice at a Medication-assisted Opioid Treatment Program

Lawrence Brown, Steven Kritz, Melissa Lin, Ben Louie, Roberto Zavala, Charles Madray

T63. Effects of Social Defeat Stress on Anhedonia in the Intracranial Selfstimulation Test

<u>Rachel Donahue</u>, John Muschamp, Sam Golden, Scott Russo, Eric Nestler, William Carlezon Jr.

T64. Toward a Bidirectional Model Animal of Bipolar Disorder: Genetic Susceptibility to Conditions that Induce Cycling between Mania and Depression in Mice

> Jared W. Young, Davide Dulcis, Jordy van Enkhuizen, Nicholas Spitzer, Andrea Grim, Mark A. Geyer

T65. Adolescent Cannabis Exposure Interacts with a Glial Genetic Risk Factor to Produce Cognitive Deficits in Adulthood

> Bagrat Abazyan, Sofya Abazyan, Michael Ballinger, Atsushi Kamiya, <u>Mikhail V. Pletnikov</u>

T66. The Interplay of Cannabinoid Signaling and DISC1 during Adolescence: Effects on Prefrontal Cortex Function in Adulthood

> Michael Ballinger, Bagrat Abazyan, Yu Taniguchi, Atsushi Saito, Koki Ito, Mikhail Pletnikov, <u>Atsushi Kamiya</u>

- T67. A Zebrafish Model for the Functional Analysis of Genes in Autism <u>Ellen J. Hoffman</u>, Joseph M. Fernandez, Antonio J. Giraldez, Matthew State
- T68. Reduced Motivation to Consume Alcohol After an Extended Access <u>Eric Augier</u>, Ruslan Damadzic, Erick Singley, Alexandra Pincus, Markus Heilig
- T69. Effects of Perinatal and Adolescent Oxidative Stress "Double Hit" on GABAergic Interneurons and Behavior in Mice

Susan B. Powell, Loek deJong, Mary E. Kamenski, Jacinta Lucero, Jared W. Young, M. Margarita Behrens

T70. Hyperactivity and Cortical Disinhibition in Mice with Restricted Expression of Mutant Huntingtin to Parvalbumin-positive Cells

Sarah E. Dougherty, John J. Hollimon, Laura J. McMeekin, Andrew S. Bohannon, Andrew B. West, Mathieu Lesort, John J. Hablitz, <u>Rita</u> <u>M. Cowell</u>

T71. Overexpression of CRF in the Central Nucleus of the Amygdala Diminishes the Dysphoric-like State Associated with Nicotine Withdrawal in Rats

Xiaoli Qi, Zhiying Shan, Yue Ji, Valerie Guerra, Jon C. Alexander, Brandi Ormerod, <u>Adrie Bruijnzeel</u>

T72. G-protein-dependent Signaling in Corticostriatal Afferents Regulates Locomotor Sensitization, Drug-taking and Drug-seeking Behaviors

Kerry Kerstetter, Amanda Wunsch, Tess Donckels, John F. Neumaier, <u>Susan Ferguson</u>

T73. Christianson Syndrome Protein NHE6 Regulates Intra-endosomal pH, BDNF Signaling and Circuit Development

Eric M. Morrow, Julie Kauer, Qing Ouyang, Sofia Lizarraga

T74. Memory Enhancement by Targeting Cdk5 Regulation of the NMDA Receptor Subunit NR2B

> <u>Florian Plattner</u>, Adan Hernandez, Karine Pozo, Gabriel Mettlach, Tanvir Singh, Deena Sajitharan, Chunfeng Tan, James A. Bibb

- T75. Reduced Somatostatin and Vasoactive Intestinal Peptide mRNAs in the Frontal Cortex of Subjects with Schizophrenia and Bipolar Disorder Samantha J. Fung, Cynthia S. Weickert
- T76. Dopamine D1-D2 Receptor Heteromer Activation Induces Place Aversion and Abolishes Cocaine Reward via a Cyclin-dependent Kinase 5 Mechanism

Melissa L. Perreault, Ahmed Hasbi, Maurice Shen, Brian F. O'Dowd, Susan R. George

T77. Schizophrenia-associated Alterations of Microtubule-associated Protein 2 in Human Auditory Cortex

Micah Shelton, Jason Newman, Kenneth Fish, Matthew L. MacDonald, Peter Penzes, David A. Lewis, <u>Robert A. Sweet</u>

- T78. Novel Replacement Strategy for Dissecting NMDA Receptor Regulation John Gray, Roger Nicoll
- T79. Gene Expression Profiling of Stress-induced Changes in CA3 Neurons Using Translating Ribosome Affinity Purification (TRAP)

Jason Gray, Todd Rubin, Bruce S. McEwen

T80. Glutamatergic Neurons in the Ventral Tegmental Area: Properties & Physiological Role

<u>Thomas S. Hnasko</u>, Ji Hoon Yoo, Gregory Hjelmstad, Howard Fields, Robert Edwards

T81. Locus Specific Epigenetic Reprogramming: Bidirectional Regulation of the FosB Gene Using Synthetic Transcription Factors In Vivo

<u>Elizabeth Heller</u>, Hannah Cates, Haosheng Sun, Catherine Pena, Deveroux Ferguson, Scott Knight, H. Steve Zhang, Eric Nestler

T82. Revealing Lithium's Molecular Mechanisms in Bipolar Disorder: Using the Circadian Clock

<u>Michael J. McCarthy</u>, Hongbing Wei, Stephen Beesley, Bruce M. Cohen, Donna L. McPhie, David Welsh

T83. A Cross-sectional Examination of Telomere Length and Telomerase in a Well-characterized Sample of Individuals with Major Depressive Disorder Compared to Controls

> <u>Naomi M. Simon</u>, Zandra Walton, Jennifer Prescott, Elizabeth Hoge, Aparna Keshaviah, T.H. Eric Bui, Noah Schwarz, Taylor Dryman, Rebecca A. Ojserkis, David Mischoulon, John Worthington, Immaculata DeVivo, Maurizio Fava, Kwok-Kin Wong

T84. Effects of Early Life Stress on Adulthood Stress Reactivity and Its Mechanisms

<u>Li Li</u>

T85. Brain Region-specific Changes in Extracellular Signal-regulated Kinase (ERK)-5 Signaling in Suicide Subjects

Yogesh Dwivedi, Ghanshyam Pandey, Hui Zhang

T86. The Genome in Three Dimensions: A New Frontier in Human Brain Research

<u>Amanda Mitchell</u>, Rahul Bharadwaj, Catheryne Whittle, Karoly Mirnics, Yasmin Hurd, Schahram Akbarian

T87. Acute but Not Chronic Psychosocial Stress Alters the Density and Immune-phenotype of Microglia in Mouse Stress-responsive Brain Regions

Michael Lehmann, Miles Herkenham

T88. The Neuron-specific Chromatin Regulatory Subunit BAF53b is Necessary for Epigenetic Regulation of Synaptic Plasticity and Memory

Annie Vogel-Ciernia, Dina Matheos, Ruth Barrett, Marcelo A. Wood

T89. Nucleus Accumbens Medium Spiny Neuron Subtypes Differentially Mediate Susceptibility and Resilience to Social Defeat Stress

> T. Chase Francis, Ramesh Chandra, Julie Brooks, Genesis Dayrit, Eric Finkel, Jeffrey D. Lenz, Sergio Iñiguez, Patricio O'Donnell, <u>Mary Kay Lobo</u>

T90. Genetic Background Regulates the Effect of Antidepressant Treatment on Behavioral Despair and Hippocampal Neurogenesis in Mice

> Brooke H. Miller, Thomas A. Lanz, Zane Zeier, Miguel Lopez-Teledono, Robin Kleiman, Mathew Pletcher, Claes Wahlestedt

T91. Genetic Modulation of Neuronal Competition Homeostasis in the Adult Dentate Gyrus to Enhance Hippocampal Functions

> <u>Amar Sahay</u>, Kathleen McAvoy, Kimberly Scobie, Stefan Berger, Nannan Guo, Sreyan Choudhry, Sam Miake-Lye, Rene Hen, Mark Nelson

T92. Disrupting AMPA Receptor Endocytosis Restores the Ability to Form New, and Enables the Recovery of Old, Memories in Mice Genetically Designed to Mimic Alzheimer's Disease

> <u>Sheena Josselyn</u>, Adelaide Yiu, Valentina Mercaldo, Derya Sargin, Paul Frankland

T93. Fragile X Mental Retardation Protein (FMRP) –Metabotropic Glutamate Receptor 5 (mGluR5) Signaling in Schizophrenia and Autism

S. Hossein Fatemi, Timothy Folsom

T94. Orbitofrontal Cortical Dendritic Spines: Markers of Adolescent (Stressor) Experience and Determinants of Habit Formation

Elizabeth A. Hinton, Andrew M. Swanson, Shannon L. Gourley

T95. The Methyltransferase PRDM2 Regulates Escalated Alcohol Consumption

Estelle Barbier, Jenica Tapocik, Andrea L. Johnstone, Jesse Schank, Zhifeng Zhou, Qiaoping Yuan, David Goldman, Claes Wahlestedt, Markus Heilig

T96. Alterations in Telencephalic Neuronal Fate, Neuronal Calcium Signaling and Neurotransmitter Release in iPSC Models of Bipolar Disorder

<u>Melvin McInnis</u>, Monica Bame, Haiming Chen, Cynthia J. DeLong, Todd J. Herron, Omar Mabrouk, Robert Kennedy, K Sue O'Shea

T97. Modulation of Dopamine Transporter by DISC1 Assemblies: A Novel Pharmacological Target

Verian Bader, Svenja Trossbach, Ingrid Prikulis, Sandra Schäble, Angelica de Souza, Zoe A. Hughes, Nicholas Brandon, Joseph Huston, <u>Carsten Korth</u>

T98. Role of Hippocampal  $\Delta$ FosB in Associations of Cocaine with Environment

Andrew Eagle, Paula Gajewski, Pamela Kennedy, <u>Alfred Jay</u> <u>Robison</u>

T99. Overexpression of the Steroidogenic Enzyme Cytochrome P450 Side Chain Cleavage in the Ventral Tegmental Area Increases 3α,5α-THP and Reduces Long-term Operant Ethanol Self-administration in Alcohol Preferring Rats

> <u>A. Leslie Morrow</u>, Jason B. Cook, David F Werner, Antoniette M. Maldonado-Devincci, Maggie N. Leonard, Kristen R Fisher, Todd K. O'Buckley, Patrizia Porcu, Thomas J. McCown, Clyde Hodge, Joyce Besheer

T100. Increased Rage, TLRs, and HMGB1 Expression in the Human Alcoholic Orbitofrontal Cortex Is Linked to Adolescent Drinking

Ryan P. Vetreno, Liya Qin, Fulton T. Crews

T101. The Extent of the Incorporation of the G Protein, Gsα, in Lipid Raft Membrane Fractions from Erythrocyte Membranes May Provide a Biomarker for Major Depressive Disorder

> <u>Mark M. Rasenick</u>, Robert Donati, Cynthia Fu, Sergi Costafreda, Peng Liu, Lauren Marangell

T102. Epigenetic Enzyme Expression Changes Associated with Alcohol Dependence

<u>Andrea L. Johnstone</u>, Christopher A. Rienas, Estelle Barbier, Jenica Tapocik, Markus Meinhardt, Shaun P. Brothers, Wolfgang H. Sommer, Markus Heilig, Claes Wahlestedt

T103. Mechanisms of Focal Thalamic Degeneration in Thiamine Deficiency Induced Wernicke's Encephalopathy-korsakoff Syndrome (WE-KS)

Fulton T. Crews, Liya Qin

T104. Expression of VEGF Receptor Is Higher with SSRI Treatment in Depressed Individuals and Correlates with Number of Cells, Capillaries and Dendrite Length in the Hippocampal Neurogenic Niche

Adrienne N. Santiago, Yan Liu, Mihran J. Bakalian, Andrew J. Dwork, Gorazd B. Rosoklija, René Hen, Victoria Arango, J. John Mann, <u>Maura Boldrini</u>

T105. Stress-context Detecting Function of the Mesolimbic Reward Circuit: The Role of CRF in Gating BDNF Signaling

> Jessica Walsh, Allyson Friedman, Haosheng Sun, Stacy Ku, Elizabeth Heller, Barbara Juarez, Veronica Burnham, Michelle Mazei-Robison, Deveroux Ferguson, Sam Golden, Ja Wook Koo, Dipesh Chaudhury, Daniel J. Christoffel, Scott Russo, Eric Nestler, <u>Ming-Hu Han</u>

T106. Altered Synaptic Protein Expression and Co-expression Network Topology Linked to Spine Loss in the Auditory Cortex of Schizophrenia

<u>Matthew L. MacDonald</u>, Ying Ding, Jason Newman, Nathan Yates, David A. Lewis, Robert A. Sweet

T107. Neurotrophin Receptor TrkB Expression in Dentate Gyrus and Hilus of Treated and Untreated Subjects with Major Depression Correlates with Number of Neural Progenitor Cells and Neurons

> Giulia Bracci, Mihran J. Bakalian, Andrew J. Dwork, René Hen, Gorazd B. Rosoklija, Victoria Arango, J. John Mann, <u>Maura Boldrini</u>

T108. DNA Methylation and Dysregulation of the GABAergic Phenotype in Post-mortem Human Hippocampus in Schizophrenia and Bipolar Disorder

W. Brad Ruzicka, Francine M. Benes

T109. Using the Olfactory Epithelium as a Surrogate Tissue to Explore Dynamic Molecular Signatures for Brain Diseases

Soumya Narayan, Koko Ishizuka, Narayan Rai, Charlee McLean, Pearl K. Kim, Maria Hipolito, Youjin Chung, Sandra Lin, John Nurnberger, Nicola Cascella, Akira Sawa, Evaristus Nwulia

T110. Neurobiological Basis of Augmentation Strategy of Serotonin Specific Reuptake Inhibitor by Compounds Able to Limit High Affinity Nicotinic Acetylcholine Receptors

<u>Yann S. Mineur</u>, Emily Einstein, Mattis Wigenstrand, Sam Blakeman, Gianna Fote, Marina Picciotto

T111. Actin Cytoskeleton Dysregulation in Schizophrenia and Bipolar Disorder: Relevance to Dendritic Spine Pathology

<u>Glenn Konopaske</u>, Sivan Subburaju, Joseph T. Coyle, Francine M. Benes

T112. Depression Decreases CD4 and Chemokine Receptor Expression in T-lymphocytes and Macrophages

> Tami D. Benton, Kevin Lynch, Steven D. Douglas, Benoit Dubé, David Gettes, Nancy Tustin, David S. Metzger, Sergei Spitsin, Dwight L. Evans

T113. Nicotinic Modulators in Subtype-specific Cortical GABAergic Neurons: Implication for Critical Period Development

> Michael Demars, Noreen Bukhari, Poromendro Burman, Ayan Hussein, <u>Hirofumi Morishita</u>

T114. Regulation of Primary Cilia Morphology in Striatum by 5 HT6 Receptor Signaling

John F. Neumaier, Matthew Brodsky, Jane Sullivan

T115. PET/CT versus PET/MR for the Clinical Evaluation of Patients with Dementia

Yu-Shin Ding, Timothy Shepherd, Fernando Boada, Kent Friedman

T116. Neuroimaging Predictors of Clinical Response and Potential Markers of Treatment with Duloxetine in Major Depressive Disorder

> <u>Cynthia Fu</u>, Sergi Costafreda, Mark M. Rasenick, Robert Donati, Peng Liu, Lauren Marangell

T117. Whole Genome DNA Cytosine Methylation in a Rat Model of Fetal Alcohol Syndrome

> <u>Kornel Schuebel</u>, Kevin Blackistone, Isioma Mordi, Qiaoping Yuan, Jennifer Thomas, David Goldman

T118. Activity-dependent Phosphorylation of MeCP2 Regulates Interaction with NCoR

Daniel Ebert, Michael E. Greenberg

T119. Epigenetic and Behavioral Correlates of Adolescent Intermittent Ethanol Exposure at Adulthood

> Subhash C. Pandey, Amul J. Sakharkar, Lei Tang, Tara Teppen, Huaibo Zhang

T120. Group I Metabotrobic Glutamate Receptor Activation Negatively Regulates Glua2-lacking Ampa Receptors in Cultured Nucleus Accumbens Neurons

> Jessica A. Loweth, Jeremy M. Reimers, Kuei Y. Tseng, Marina E. Wolf

- T121. CSF from HD Subjects Can Seed Aggregation of Mutant Huntingtin Steven Potkin, Zhigun Tan, Leslie Thompson, Charles Glabe
- T122. Telomere Length in Schizophrenia as a Function of Age and Illness Duration

Owen M. Wolkowitz, Barton W. Palmer, Danielle Glorioso, Wesley Thompson, Elissa S. Epel, Jue Lin, Elizabeth Blackburn, Dilip V. Jeste

T123. Cognitive Dysfunction and Higher Levels of Autofluorescence (AF) in Schizophrenia (SZ) Patient-derived Cells and Animal Models

> Tsuyoshi Tsujimura, Chi Ying. Lin, Juan A. Gallego, Xela Indurkhya, Nao Gamo, Minori Koga, Tess Maseda, Tom Sedlak, Anil Malhotra, Carsten Korth, Koko Ishizuka, Akira Sawa

T124. Functional Analysis of the Schizophrenia-associated Gene, TCF4

Matthew D. Rannals, Andrew Jaffe, Ran Tao, Thomas M. Hyde, Joel E. Kleinman, Daniel Weinberger, Brady J. Maher

T125. Regulation of Tyrosine Hydroxylase by CLOCK: Potential Mechanisms Underlying the Circadian Control of Dopamine and Reward

Wilbur Williams, Angela Ozburn, Colleen A. McClung

T126. Rats Prone to Obesity Show 'Addiction-like' Deficits in Behavior and Synaptic Plasticity

<u>Robyn M. Brown</u>, Yonatan M. Kupchik, Sade Spencer, Constanza Garcia-Keller, Danielle Schwartz, Kelsey Jordan, Thomas C. Jhou, Peter W. Kalivas

T127. Analysis of the Pain Transcriptome Using RNA-Seq

Samridhi Goswami, Santosh Mishra, Mark Hoon, Andrew Mannes, Michael Iadarola

T128. Actigraphy Measured Sleep Disruption as a Predictor of Survival among Women with Advanced Breast Cancer

> <u>David Spiegel</u>, Oxana Palesh, Arianna Aldridge-Gerry, Jamie Zeitzer, Cheryl Koopman, Janine Giese-Davis, Booil Jo, Helena Kraemer, Eric Neri, Bita Nouriani

T129. Inflammation, Depression and N-3 Fatty Acids: A Case of Personalized Medicine

<u>Mark Hyman. Rapaport</u>, Pamela Schettler, Thaddeus W. Pace, Becky Kinkead, Andrew A. Nierenberg, David Mischoulon

- T130. The Opiate Antagonist, Naltrexone, in the Treatment of Trichotillomania: Results of a Double-blind, Placebo-controlled Study Jon E. Grant, Brian Odlaug, Suck Won Kim
- T131. Perinatal Choline Supplementation Is Associated with Earlier Maturation of P50 Sensory Gating and May Improve Preschool Attentional Function

<u>Randal Ross</u>, Sharon Hunter, Lizbeth McCarthy, Amanda Hutchison, Brandie Wagner, Sherry Leonard, Karen Stevens, Robert Freedman

T132. An International Study of the GRID-HAMD: Has It Fulfilled Its Promise?

Janet B.W. Williams, Matej Ondrus, Melanie Kitzinger, Jennie Persson, Marlene Popescu, Risto Valjakka

T133. Methadone and Suboxone for Subutex Injectors: Primary Outcomes of Pilot RCT

<u>George E. Woody</u>, David Otiashvili, Gvantsa Piralishvili, Zura Sikharulidze, George Kamkamidze, Sabrina Poole

T134. Treatment-related Improvement in Neuropsychological Functioning in Depressed Patients at High Risk for Suicidal Behavior: Paroxetine vs. Bupropion

Marianne Gorlyn, John Keilp, Ainsley Burke, Maria Oquendo, J. John Mann, <u>Michael Grunebaum</u>

T135. How Far Are Duplicate Subjects Willing to Go? Changing Indications and Identifiers in Order to Participate in Studies at Distant Sites

Lilit Gevorgyan, Zoe Shiovitz, Marlene Zarrow, Thomas Shiovitz

T136. Corticostriatothalamic Circuit Dysfunction in Major Depressive Disorder

Olusola Ajilore, Melissa Lamar, Jamie Cohen, Anand Kumar

T137. Aripiprazole Lauroxil (ALKS 9070), a Novel Once-monthly Prodrug of Aripiprazole, Achieves Therapeutically Relevant Levels and Is Well-tolerated in Adult Patients with Schizophrenia Following Deltoid Administration

> <u>Ryan Turncliff</u>, Marjie Hard, David Brown, Mark Lerman, Adam Lowy, Morteza Marandi, Yangchun Du, Robert Risinger, Elliot W. Ehrich

T138. Clinical Assessment of Lurasidone Benefit and Risk in the Treatment of Bipolar I Depression Using Number Needed to Treat, Number Needed to Harm, and Likelihood to be Helped or Harmed

Leslie Citrome, Terence A. Ketter, Josephine Cucchiaro, Antony Loebel

#### ACNP 52nd Annual Meeting • Final Program

### Poster Session II—Tuesday

T139. Intranasal Ketamine in Treatment-resistant Depression

<u>Kyle A. Lapidus</u>, Cara F. Levitch, Laili Soleimani, Andrew M. Perez, Jess W. Brallier, Michael K. Parides, Dan V. Iosifescu, Dennis S. Charney, James W. Murrough

T140. Do Structured, Taped and Reviewed Rating Interviews Improve Outcomes in Antidepressant Trials?

Arif Khan, James Faucett, Walter A. Brown

T141. Patient-reported Outcome of Antipsychotic Treatment - Relationships to Psychopathology, Compliance and Remission

Dieter Naber

T142. The Efficacy of Vortioxetine versus Placebo in the Treatment of Adults with Major Depressive Disorder: Patient Level Data from 10 Short-term Studies and a Meta-analysis

Michael E. Thase, Atul Mahableshwarkar, Marianne Dragheim

T143. Varenicline Effects on Smoking, Cognition, and Psychiatric Symptoms in Schizophrenia

<u>Robert C. Smith</u>, Revital Amiaz, Si.Tian Mei, Lawrence Maayan, Hua Jin, Sylvia Boules, Henry Sershen, Chunbo Li, Juanjuan Ren, Liu Yanhong, Harshita Ravishankar, Abel Lajtha, Alessandro Guidotti, Mark Weiser, John M. Davis

T144. Citalopram Decreases Acute CSF Aβ Production in Young Healthy Subjects

<u>Yvette Sheline</u>, Tim West, Kevin Yarasheski, Robert A. Swarm, John Cirrito, Jin-Moo Lee, Mateusz S. Jasielec, Christine Frederiksen, Robert Chott, John C. Morris, Mark A. Mintun

T145. The Differential Effect of Early and Efficient Interventions for Acute PTSD Declines with Time

<u>Arieh Y. Shalev</u>, Yael Ankri, Moran Gilad, Yossi Israeli-Shalev, Meng Qian, Isaac Galatzer-Levy, Sara Freedman

T146. Reduced p11 in Blood Cells Predicts Antidepressant Response to Citalopram

<u>Per Svenningsson</u>, Louise Berg, Daniel Matthews, Alan Malinger, Marisa Toups, Madhukar Trivedi, Carlos A. Zarate, Paul Greengard

T147. Effects of Acute and Sustained Administration of the Antidepressant Vilazodone on Monoaminergic Systems: In Vivo Electrophysiological Studies

Pierre Blier, Agnes Crnic, Mostafa El Mansari

T148. The Efficacy of Levomilnacipran ER in Patients with Prominent Fatigue Symptoms: Post Hoc Pooled Analyses of Double-blind Placebocontrolled Trials

<u>Carl Gommoll</u>, Adam Ruth, Changzheng Chen, William M. Greenberg, Maurizio Fava

T149. Efficacy and Safety of Intravenous Esketamine in Patients with Treatment-resistant Depression: A Double-blind, Double-randomization, Placebo-controlled Phase 2a Study

> Jaskaran Singh, Margaret Fedgchin, Ella Daly, Liwen Xi, Caroline Melman, Geert De Bruecker, Andre Tadic, Pascal Sienaert, Frank Wiegand , Husseini K. Manji, Wayne Drevets, Luc Van Nueten

T150. The Triple Reuptake Inhibitor Antidepressant Effects (TRIADE) Trial: Amitifadine for the Treatment of Major Depressive Disorder

> <u>Marlene P. Freeman</u>, Anthony McKinney, Mark Bradshaw, Pierre Tran, Timothy Hsu, Maurizio Fava

T151. Reliability of Behavioral Phenotyping Predictors of Treatment Response in the EMBARC Study

> Diego A. Pizzagalli, Daniel Dillon, Pia Pechtel, Phillip Adams, Thomas Carmody, Crystal Cooper, Patricia J. Deldin, Maurizio Fava, Benji T. Kurian, <u>Patrick J. McGrath</u>, Melvin McInnis, David W. Morris, Ramin V. Parsey, Madhukar Trivedi, Myrna M. Weissman, Gerard Bruder

T152. Can Oxytocin Enhance Learning during Social Cognitive Skills Training in Schizophrenia?

Michael C. Davis, Michael F. Green, Junghee Lee, William Horan, Jonathan K. Wynn, Stephen R. Marder

T153. Examining for Potential Duplicate Patients in Clinical Trials: CATIE Analysis

Jonathan Rabinowitz, Yaacov Z. Rabinowitz

T154. A Double-blind Placebo-controlled Study of Long-chain Omega-3 Fatty Acid Supplementation for Depression in Youth at Ultra-high Risk for Bipolar Disorder

> <u>Melissa DelBello</u>, Jeffrey Welge, Jeffrey Strawn, Luis R. Patino Duran, Lauren Stahl, Thomas Blom, Stephen Strakowski, Robert McNamara

T155. Reliability of Electrophysiological Predictors of Treatment Response in the EMBARC Study

<u>Diego A. Pizzagalli</u>, Craig E. Tenke, Jürgen Kayser, Pia Pechtel, Daniel Dillon, Crystal Cooper, Patricia J. Deldin, Maurizio Fava, Benji T. Kurian, Patrick J. McGrath, Ramin V. Parsey, Eva Petkova, Madhukar Trivedi, Myrna M. Weissman, Sarah Weyandt, Gerard Bruder

T156. Modulation of N-methyl-D-aspartate (NMDAR)-type Glutamate Receptors in Psychiatric Disorders

> <u>Joshua T. Kantrowitz</u>, Michael Epstein, Odeta Beggel, Nayla Scaramello, Gail Silipo, Elisa Dias, Stephanie Rohrig, Batsheva Halberstam, Marlene Carlson, Daniel C. Javitt

T157. Randomized Comparison of the Acute Effects of Olanzapine and Ziprasidone on Tissue-specific Insulin Sensitivity in Healthy Volunteers

John W. Newcomer, Karen Flavin, Michael D. Yingling, Julia A. Schweiger, Angie Stevens, Ginger E. Nicol

T158. Efficacy and Safety of Vilazodone in Major Depressive Disorder: A Randomized, Double-blind, Placebo-controlled Trial

> Nunzio Pomara, Carl Gommoll, Dalei Chen, Rene Nunez, Maju Mathews, Harry A. Croft

T159. Efficacy of Cariprazine Across Schizophrenia Symptoms: A Post Hoc Analysis of PANSS Data from a Phase III, Double-blind, Placebo- and Active-controlled Trial

<u>Stephen R. Zukin</u>, Jeffrey A. Lieberman, Andrew J. Cutler, Kaifeng Lu, Raffaele Migliore, István Laszlovszky, György Németh, Suresh Durgam

T160. Randomized, Double-blind, Placebo-controlled Study of the Efficacy of Vortioxetine on Cognitive Dysfunction in Adult Patients with Major Depressive Disorder (MDD)

Roger S. McIntyre, Soren Lophaven, Christina K. Olsen

T161. Cognitive Remediation in Bipolar Disorder: Efficacy and Neural Correlates of Treatment

> <u>Kathryn E. Lewandowski</u>, Matcheri Keshavan, Bruce M. Cohen, Sarah H. Sperry, Dost Ongur

T162. Automated Analysis of Disorganized Communication Predicts Transition to Psychosis among Clinical High Risk Patients

Gillinder Bedi, Facundo Carillo, Guillermo Cecchi, Diego Fernandez Slezak, Mariano Sigman, Jordan E. DeVylder, Felix M. Muchomba, <u>Cheryl M. Corcoran</u>

T163. Protocol Complexity and Enrollment Eligibility Exclusivity: Are Today's Depression Study Volunteers Truly Representative?

<u>Charles S. Wilcox</u>, Judy L. Morrissey, Nader Oskooilar, Mellissa M. Henry, Daniel E. Grosz, My-Linh Tong, Don F. De Francisco

T164. Auditory Steady State Evoked Potential Abnormalities in Schizophrenia Are Normalized by an mGluR2 Positive Allosteric Modulator

> <u>Bruce Turetsky</u>, Daniel Wolf, Christian Kohler, Mary March, Alan Cross, Mark Smith, Stephen R. Zukin, Raquel E. Gur

T165. Effects of Neurokinin 1 Receptor Antagonism on Brain Response to Emotional Visual Stimuli in Co-morbid Alcohol Dependence and Posttraumatic Stress Disorder

> <u>Primavera Spagnolo</u>, Laura Kwako, Reza Momenan, Melanie L. Schwandt, Vijay A. Ramchandani, Daniel W. Hommer, David T. George, Markus Heilig

T166. Baseline Characteristics that Result in Higher Placebo on the MCCB Using Regression Analysis

George Haig, Earle Bain

- T167. Long-term Follow-up of Gamma Capsulotomy for Intractable OCD Steven Rasmussen, Benjamin Greenberg
- T168. N-Acetylcysteine for the Treatment of Non-suicidal Self-injurious Behavior in Adolescents: A Preliminary Study

<u>Kathryn R. Cullen</u>, Bonnie Klimes-Dougan, Lori LaRiviere, Alaa Houri, Melinda Westlund, Bernard Lim, Ana Bortnova, Katharine Nelson, Michael J. Miller, S. Charles Schulz, Bryon Mueller, Lynn Eberly, Kelvin O. Lim

T169. Phase 2 Evaluation of ITI-007, a Novel Approach to the Treatment of Schizophrenia

<u>Kimberly E. Vanover</u>, Sharon Mates, Paul Greengard, Robert E. Davis

T170. Modafinil for the Treatment of Cocaine Dependence

Kyle M. Kampman, Jennifer G. Plebani, Kevin G. Lynch, Helen M. Pettinati, Elizabeth Mahoney, Mary Slome, Margo Hendrickson, Charles P. O'Brien

T171. Personality Predicts Dropout and Placebo Response Risk in Patients with Bipolar Depression

<u>Gary S. Sachs</u>, Cynthia Siu, Josephine Cucchiaro, Robert Silva, Fred Grossman, Jay Hsu, Amir Kalali, Antony Loebel

T172. Sleep Architecture Abnormalities as a Risk Factor for Elevated Suicidal Ideation: A Polysomnographic Investigation of Sleep in Treatment Resistant Unipolar and Bipolar Depression

<u>Rebecca Bernert</u>, David Luckenbaugh, Wallace C. Duncan, Carlos A. Zarate

T173. Benzoate, a D-amino Acid Oxidase Inhibitor, for Treatment of Earlyphase Alzheimer's Disease: A Randomized, Double-blind, Placebocontrolled Trial

Guochuan Emil Tsai

T174. Early Changes in Apathy Predicts Response to Adjunctive Oral Methylphenidate in Depressed Patients

Sidney Kennedy, Sakina Rizvi, Joseph Geraci, Arun Ravindran

T175. CONSORT-NP: Guidelines for Reporting of Neuropsyhological Test Results in Clinical Trials

Robert M. Bilder

T176. Effects of Antidepressant Medication on Emotion Regulation in Major Depressive Disorder: An iSPOT-D Report

Leanne Williams, Kateri McRae, William Rekshan, James Gross

T177. Trichuris Suis Ova (TSO) as an Immune-inflammatory Treatment for Repetitive Behaviors in ASD

Eric Hollander, Casara J. Ferretti, Bonnie P. Taylor, Rachel Noone, Jonathan Kirsch, Emma Racine

T178. Safety and Efficacy of Short-term Treatment with the Acetylcholinesterase Inhibitors Rivastigmine and Huperzine a to Reduce the Subjective and Reinforcing Effects Produced by Acute Cocaine Exposure

> <u>Christopher D. Verrico</u>, James J. Mahoney, Kimberly N. Cooper, Tabish Iqbal, Thomas F. Newton, Richard De La Garza

T179. The Establishing Moderators and Biosignatures of Antidepressant Response for Clinical Care (EMBARC) Study: Rationale, Design and Progress

> <u>Madhukar Trivedi</u>, Patrick J. McGrath, Maurizio Fava, Ramin V. Parsey, Marisa Toups, Benji T. Kurian, Mary L. Phillips, Maria Oquendo, Gerard Bruder, Diego A. Pizzagalli, Sarah Weyandt, Randy Buckner, Philips Adams, Thomas Carmody, Eva Petkova, Myrna M. Weissman

T180. Safety and Effectiveness of Aripiprazole Once-monthly for the Treatment of Schizophrenia: A Pooled Analysis of Two Double-blind, Randomized, Controlled Trials (246 and 247)

> <u>W. Wolfgang Fleischhacker</u>, Raymond Sanchez, Tsai Lan-Feng, Timothy Peters-Strickland, Ross A. Baker, Anna Eramo, Dusan Kostic, John Kane

T181. Neural Response in Visual Cortex to Emotional Stimuli Predicts Clinical Outcome across Rapid Antidepressant Agents

> <u>Maura L. Furey</u>, Joanna Szczepanik, Allison C. Nugent, Nancy Brutsche, David Luckenbaugh, Carlos A. Zarate

T182. Feedforward and Feedback Control Abnormalities during Precision Grasping Implicate Cerebellar Dysfunction in Autism Spectrum Disorder

> <u>Matthew W. Mosconi</u>, Suman Mohanty, Rachel K. Greene, Lauren Schmitt, David E. Vaillancourt, John A. Sweeney

T183. Elevated Maternal C-reactive Protein and Schizophrenia in a National Birth Cohort

> <u>Alan Brown</u>, Sarah E. Canetta, Helja-Marja Surcel, Susanna Hinkka-Yli-Salomäki, Andre Sourander

T184. Childhood Maltreatment and the Structure of the Incidence of Psychiatric Disorders: A National Study

Carlos Blanco, Melanie Wall, Chelsea Jin

T185. Factors Impacting Functional Disability and PTSD Symptoms in OEF/ OIF/OND Veterans with PTSD

> <u>F. Andrew Kozel</u>, Jeffrey Spence, Christina Bass, Cassie Rae. Morgan, Penelope Jones, Caitlin D. Schraufnagel , Mary B. Turner, John Hart

T186. Early-onset and Very-early-onset Bipolar Disorder: Distinct or Similar Clinical Conditions?

<u>Lukas Propper</u>, Claire O'Donovan, Martina Ruzickova, Cynthia Calkin, Tomas Hajek, Abigail Ortiz, Claire Slaney, Julie Garnham, Martin Alda

T187. Obsessive-compulsive Symptom Dimensions in School Age Children and Their First Degree Relatives: Results from a Large Communitybased Study

> <u>Pedro de Alvarenga</u>, Raony Cassab Cezar, Tais Moriyama, Marcelo Hoexter, Gisele Manfro, James Leckman, Euripedes Constantino. Miguel, Maria Conceicao Rosario

T188. Premorbid Impairments in Childhood-onset Schizophrenia

<u>David I. Driver</u>, Deanna Greenstein, Madison Farmer, Judith L. Rapoport, Nitin Gogtay

T189. Replication and Refinement of the Predictive Value of Cognitive Markers in ADNI: Four Year Follow-up Data

Jesus J. Gomar, Concepcion Conejero-Goldberg, Peter Davies, Terry E. Goldberg

T190. Frequency and Characteristics of Isolated Psychiatric Episodes in Anti-NMDA Receptor Encephalitis

<u>Matthew Kayser</u>, Maarten Titulaer, Nuria Gresa-Arribas, Josep Dalmau

T191. Rare Genetic Variants in VMAT1 (SLC18A1) Are Functional In Vitro and Associated with Bipolar Disorder

Falk W. Lohoff, Rachel Hodge, Sneha Narasimhan, Glenn Doyle

T192. Can Serotonin Put Your Mind at Rest?

Alexander Schäfer, Inga Burmann, Ralf Regenthal, Katrin Arelin, Andre Pampel, Arno Villringer, Daniel Margulies, <u>Julia Sacher</u>

T193. Poverty and the Past: The Relation between Hippocampus Function and Memory Performance Is Linked to Childhood Poverty

<u>Elizabeth R. Duval</u>, Sarah N. Garfinkel, Chandra S. Sripada, James E. Swain, Gary W. Evans, Israel Liberzon

T194. Therapeutic Benefits of Dutasteride, a 5 Alpha-reductase Type I Inhibitor, in PMDD: Results of a Pilot Study

> <u>Pedro Martinez</u>, Lynnette K. Nieman, Leslie Morrow, Dahima Cintron, Karla Thompson, David Rubinow, Peter Schmidt

T195. The Hypothalamic-pituitary-adrenal Axis, Reproductive Aging, and Depression: Results of Cortisol and ACTH Response to Dex/CRH Testing in Women with and without Perimenopause-related Depression

> <u>Gioia Guerrieri</u>, Rivka Ben Dor, Leslie Smith, Karla Thompson, Pedro Martinez, David Rubinow, Peter Schmidt

T196. Impaired Glycemic Control in Urban African Americans with Type 2 Diabetes: Depression and Deficits in Functional Capacity

> Dominique L. Musselman, David Ziemer, Julia Seay, Marcia McNutt, Erica Royster, Bridget Larsen, Terrika Barham, Angelo Brown, Octavia Vogel, Lawrence Phillips, Philip Harvey

T197. Mu Opioid Receptor A118G Polymorphism Alters Venous Plasma Cortisol Prolactin and Heart Rate during Stress

Edward F. Domino, Keeley Erhardt, Mika Fujita-Hirasawa

T198. Inflammation Is Heightened in Iraq and Afghanistan Veterans with Posttraumatic Stress Disorder

<u>Aoife O'Donovan</u>, Beth Cohen, Karen Seal, Daniel Bertenthal, Shira Maguen, Mark Pacult, Thomas Neylan

T199. Reduced Function of Cacna1c Encoded Cav1.2 during a Hormone Sensitive Period in Brain Development Leads to Sex-dependent Resilience to Despair in Adulthood

Michal Arad, Margaret M. McCarthy, Todd D. Gould

T200. Fasting-induced Increase in Plasma Ghrelin Is Blunted by Intravenous Alcohol Administration: A Within-subject Placebo-controlled Study

Lorenzo Leggio, Melanie L. Schwandt, Emily N. Oot, Alexandra A. Dias, Vijay A. Ramchandani

T201. Early Life Adversity Increases Risk of New Onset Depression during the Menopause Transition

<u>C. Neill Epperson</u>, Mary Sammel, Stephanie Scalice, Sarah Conlin, Ellen Freeman

T202. Cortisol Response to Psychosocial Stress during Depression and Remission

Uma Rao, Matthew C. Morris

T203. Salivary Cortisol Response to Trier Social Stress Test in Healthy Third Trimester Pregnant Women and Third Trimester Pregnant Women at Elevated Risk of Developing Postpartum Depression

<u>Kristina M. Deligiannidis</u>, Aimee R. Kroll-Desrosiers, Bruce A. Barton, Anthony J. Rothschild

T204. Direct Comparison of the Psychometric Properties of Multiple Interview and Patient-rated Assessments of Suicidal Ideation and Behavior in a Large Inpatient Sample

> Eric Youngstrom, Ahmad Hameed, Michael Mitchell, Andrew Freeman, Anna Van Meter, Guillermo Perez Algorta, Alan J. Gelenberg, Roger Meyer

T205. Methylation of the Leukocyte Glucocorticoid Receptor: Early Adversity and HPA Axis Function

<u>Audrey R. Tyrka</u>, Lawrence H. Price, Carmen J. Marsit, Noah S. Philip, Linda L. Carpenter

T206. Third Trimester Free Thyroxine and Thyroid Binding Globulin Predict Subsequent Perinatal Depression and Anxiety Symptoms as Well as Syndromal Depression

> <u>Cort Pedersen</u>, Jacqueline Johnson, Nacire Garcia, Melissa Stansbury, Jane Leserman

T207. Dexamethasone Attenuates Impaired Fear Inhibition in PTSD in a Double-blind Placebo-controlled Study

<u>Tanja Jovanovic</u>, Seth D. Norrholm, Jennifer Stevens, Karin M. Nylocks, Kimberly Kerley, Bekh Bradley, Kerry J. Ressler

T208. Next-generation Psychiatric Drugs: Acetyl-L-carnitine

Carla Nasca, Danielle Zelli, Per Svenningsson, Aleksander Mathé, Bruce S. McEwen

T209. Sex Steroid Receptor Gene Expression Correlates with the Expression of Neurodevelopmental Genes and Modulates Gray Matter Volume in the Human Brain

> <u>Tuong-Vi Nguyen</u>, Peter Schmidt, J. Shane. Kippenhan, Melanie Sottile, Victor Ekuta, Bhaskar S. Kolachana, Beth A. Verchinski, Venkata S. Mattay, Joel E. Kleinman, Barbara Lipska, Daniel R. Weinberger, Karen F. Berman

T210. Disruption of Early Maternal Care Results in Epigenetic Regulation of the Oxytocin Receptor (OXTR) Gene in Rhesus Macaques

Maggie B. Baker, Stephen Lindell, Qiaoping Yuan, Zhifeng Zhou, J. Dee Higley, Stephen Suomi, <u>Christina Barr</u>

T211. Consumption of a High-fructose Diet during Adolescence Alters HPA Axis Function, Increases Anxiety-like and Depressive-like Behaviors, and Remodels Hypothalamic Gene Expression

Gretchen Neigh, Zachary Johnson, Constance Harrell

T212. Use of Gonadotropin-releasing Hormone Agonist Experimental Model to Isolate Predictors of Depressive Symptoms in Menopause: Role of Nocturnal Hot Flashes and Sleep Disturbance

> <u>Hadine Joffe</u>, Semmie Kim, Sybil Crawford, Marlene P. Freeman, Nicole Economou, David White, Lee S. Cohen, Janet E. Hall

T213. Menopause and Metabolic Function: Interactive Influences on Depression Symptoms and Emotional Regulation

<u>Alison Berent-Spillson</u>, Courtney Marsh, Carol Persad, Jon-Kar Zubieta, Yolanda Smith

T214. Effects of Intranasal CRF on Brain Response to Threat in Humans Royce J. Lee, Jayant Pinto, Eryka Nosal, Vernon Towle

T215. Vasopressin and CRF Regulation of Hypothalamic-pituitary-adrenal Axis Responsivity in Healthy Volunteers and Drug Free Former Cocaine Dependent Participants

> <u>Brian Reed</u>, Elizabeth Ducat, Brenda Ray, Molly Deutsch-Feldman, Mary Jeanne Kreek

T216. Real-time Functional MRI Feedback, Compared to Sham, Reduces Cueinduced Nicotine Craving in Smokers: Results from the First Clinical Trial

<u>Colleen A. Hanlon</u>, Karen Hartwell, Jeffrey J. Borckardt, James J. Prisciandaro, Melanie Canterberry, Xingbao Li, Max Owens, Todd LeMatty, Michael Saladin, Megan Moran-Santa Maria, Mark S. George, Kathleen T. Brady

T217. Selective Suppression of α-synuclein in Monoaminergic Neurons of Mice by Intranasal Delivery of Targeted Small Interfering RNA or Antisense Oligonucleotides: Potential Therapy for Parkinson's Disease

> Ariadna Recasens, Mireia Galofré, Iria Carballo-Carbajal, A. Ferrés-Coy, Jordi Bové, Celine Perier, María del Carmen Carmona, M. I. Santos, S. Baena, M Rosario Chica, <u>Andrés P. Montefeltro</u>, R. Revilla, Analia Bortolozzi

T218. Autonomic Responses to Intraoperative Subcallosal Cingulate DBS

<u>Patricio Riva Posse</u>, Cory Inman, Stephan Hamann, Steven Garlow, Robert Gross, Helen S. Mayberg

T219. A Two-site Pilot Study Suggests that Three Days (9 Sessions) of High Dose Left Prefrontal Repetitive Transcranial Magnetic Stimulation (rTMS) Is Feasible, Safe, and Reduces Suicidal Thinking in Suicidal Inpatients

> <u>Mark S. George</u>, Rema Raman, Sonia Jain, David M. Benedek, Christopher G. Pelic, Geoffrey G. Grammer, Karen Stokes, Matthew Schmidt, Chad Spiegel, Nancy DeAlmeida, Kathryn Beaver, Jeffrey J. Borckardt, Xiaoying Sun, Murray B. Stein

T220. Anxiety Moderates Response to Psychosocial Treatment for Depression in Bipolar Disorder: Results from Systematic Treatment Enhancement Program for Bipolar Disorder

> <u>Thilo Deckersbach</u>, Amy Peters, Navneet Kaur, Andrew K. Corse, Amanda R. Arulpragasam, Louisa Sylvia, Pedro Vieira da Silva, Michael Otto, Ellen Frank, David Miklowitz, Michael Berk, Darin Dougherty, Andrew A. Nierenberg

T221. Addition of a Comprehensive, Individualized, Person Centered Management Program, to Memantine Alone Produces a 900% Increment in a Pivotal Trial Global Measure over Medication Treatment Alone in Advanced Alzheimer's Disease

Barry Reisberg, Sunnie Kenowsky, Sloane Heller, Istvan Boksay, James Golomb, Santosh Ghimire, Carol Torossian, Iryna Lobach

T222. Neuropeptide-S Microinfusion into Basolateral Amygdala Rescues Behavior in a Rat Model of Posttraumatic Stress Disorder by Increasing Expression of BDNF and Neuropeptide Y-Y1 Receptor

Aleksander Mathé, Joseph Zohar, Hagit Cohen

T223. Monitoring Depression Severity Using the Patient Health Questionnaire during a Treatment Course of Transcranial Magnetic Stimulation

Umut Ozbek, Jonathan Cohen, Rebecca Gordon, Marc J. Dubin

T224. Electroconvulsive Therapy Pre-treatment with Low Dose Propofol: Comparison with Unmodified Treatment

Adarsh Tripathi, Nathan Winek, Kapil Goel, Douglas D'Agati, Jesus Gallegos, Geetha Jayaram, Thai Nguyen, Punit Vaidya, Peter Zandi, Jitendra Trivedi, <u>Irving M. Reti</u>

T225. The Effects of an Index Course of Magnetic Seizure Therapy and Electroconvulsive Therapy on Verbal Learning and Memory

<u>Shawn M. McClintock</u>, Mustafa Husain, C. Munro Cullum, Angel V. Peterchev, Ira Bernstein, Louis Stool, Bruce Luber, Paul Croarkin, Elisabeth Bernhardt, Kenneth Trevino, Sarah Lisanby

T226. Rapid Reduction in Suicide Risk in Depressed Elderly Treated with Electroconvulsive Therapy (ECT): Data from Phase I of the PRIDE Study

Charles Kellner, <u>Georgios Petrides</u>, Rebecca Knapp, W Vaughn McCall, Mustafa Husain, Robert Young, Sarah Lisanby

- T227. Transcendental Meditation for the Treatment of PTSD in Veterans Kelvin O. Lim, Amy Moran, Michael Kuskowski, Gregory Lamberty
- T228. Modifiable Risk Factors and Reports of Depression in Young, Middleaged, and Older Adults

Prabha Siddarth, Aaron Kaufman, David A. Merrill, Cyrus A. Raji, Fernando Torres-Gil, <u>Gary W. Small</u>

- T229. Electroconvulsive Therapy Response and Resting State Functional Connectivity in Older Patients with Major Depressive Disorder Chris Abbott, Thomas Jones, Nicholas T. Lemke, Shruti Gopal
- T230. Clinical Experience of Seven DBS for OCD Patients at an Academic Medical Center

Laurie M. McCormick, James Beeghly, Jeremy Greenlee

T231. Hyperthermia and the Improvement of ASD Symptoms

<u>Casara J. Ferretti</u>, Bonnie P. Taylor, Rachel Noone, Emma Racine, Jonathan Kirsch, Eric Hollander

T232. Substrate-selective COX-2 Inhibition Decreases Anxiety via Endocannabinoid Activation

> Daniel Hermanson, Nolan Hartley, Joyonna Gamble-George, Lawrence Marnett, <u>Sachin Patel</u>

T233. Oxytocin and Facial Expressivity in Patients with Schizophrenia and Healthy Participants

<u>Josh Woolley</u>, Chris Fussell, Wanda Lai, Olivia Lam, Brandon Chuang, Bruno Biagianti, Dan Fulford, Daniel H. Mathalon, Sophia Vinogradov

- T234. Withdrawn
- T235. Endothelial Function in Schizophrenia

Bernard A. Fischer, William R. Keller, Robert P. McMahon, Walter Meyer, Michael Miller, Robert W. Buchanan

T236. New Insight into How Ventral Tegmental Area Neurons Encode Action Sequence and Outcome Associations

Jesse Wood, Nicholas Simon, Frederick S. Koerner, Robert E. Kass, Bita Moghaddam

T237. Adolescent Ventral Tegmental Area Neurons Maintain Cue Evoked Responding After Extinction: A Mechanism for Adolescent Behavioral Flexibility?

Nicholas Simon, Yunbok Kim, Jesse Wood, Bita Moghaddam

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|       | ACNP 52nd Annual Meeting | Final Program |
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# Poster Session III – Wednesday, December 11, 2013



Advocacy Affiliate – Cure Alliance for Mental Illness Research Parity for Mental Illness

Robin Cunningham, Hakon Heimer

W1. 2013 Report of the Membership Advisory Task Force

Linda Carpenter, Lisa Monteggia, Margaret Haney, Katherine Burdick, Jennifer Bartz, Elisabeth Binder, Paul Holtzheimer, Amit Etkin, Erica Forbes, Marlene Freeman, Thomas Schulze, Christina Barr, Gregory Light, Vaishali Bakshi, Raymond Cho, Cynthia Crawford, Philip Szeszko

W2. Interoceptive Awareness in Meditators during Cardiorespiratory Deviations in Body Arousal

Sahib Khalsa, David Rudrauf, Richard Davidson, Daniel Tranel

W3. Neural Mechanisms of Extinction Learning for Monetary Reward in Health and Cocaine Addiction

Anna Konova, Muhammad Parvaz, Nelly Alia-Klein, Rita Goldstein

- W4. Withdrawn
- W5. BDNF, Synaptic Function and Cognitive Decline in Healthy Subjects and Pre-clinical Alzheimer's Disease

Pradeep J. Nathan

W6. Prolonged Temporal Interaction for Peripheral Visual Processing in Schizophrenia: Evidence from a Three-flash Illusion

Yue Chen, Daniel Norton, Charles Stromeyer

#### Poster Session III—Wednesday

W7. Social Isolation Stress Markedly Reduces the Response of Cortical Dopaminergic Neurons to Pleasurable Stimuli.

Giovanni Biggio, Laura Dazzi

W8. Impaired Neural Functioning Following Internally Focused Cognition in Obsessive-compulsive Disorder

Emily R. Stern, Alexandra F. Muratore, Stephan F. Taylor, James L. Abelson, Patrick R. Hof, Wayne K. Goodman

W9. Hyperconnectivity of Default Mode and Emotion-salience Neural Networks in Late-adolescent, Remitted Depression

<u>Rachel Jacobs</u>, Laura Gabriel, Kelly Ryan, Sara Weisenbach, Amanda M. Baker, Amy T. Peters, Rachel Ringrose, Gloria Harrington, Jon-Kar Zubieta, K. Luan Phan, Scott Langenecker, Robert Welsh

W10. Altered GABAergic Signaling in Prefrontal Cortex Contributes to Impaired Working Memory in Aged F344 Rats

> Jennifer Bizon, Cristina Banuelos, Sofia Beas, Joseph McQuail, Ryan Gilbert, Barry Setlow

W11. Negative Symptoms in the Early Course of Schizophrenia: Their Longitudinal Stability and Relationship to Early Cognitive Processes

<u>Joseph Ventura</u>, Kenneth Subotnik, Michael J. Gitlin, Denise Gretchen-Doorly, Gerhard S. Hellemann, Kathleen F. Villa, Keith H. Nuechterlein

W12. Error Monitoring in Autism: Correlates to Symptom Severity

<u>Melisa Carrasco</u>, Gregory L. Hanna, Catherine Lord, William J. Gehring

W13. Prospective Examination of Prepulse Inhibition in OIF/OEF Marines Suggests Reduced Sensorimotor Gating Is a Pre-existing Factor in Those that Develop PTSD after Combat Deployment

Victoria Risbrough, <u>Dean Acheson</u>, Dewleen G. Baker, Caroline Nievergelt, Kate Yurgil, Mark A. Geyer

W14. Relationship between Peritraumatic Distress and Attentional Avoidance of Trauma-relevant Threat in the Prediction of Posttraumatic Stress Disorder: Preliminary Results from a Prospective Study

Charmaine Thomas, Christopher Sears, Etienne Very, Juliette Salles, <u>T.H. Eric Bui</u>

W15. Reduced Impairment, Yet Increased Reliability of Cognitive Control Measurements in Remitted MDD

> <u>Scott Langenecker</u>, Rachel Jacobs, Natania Crane, Kelly Ryan, Sara Weisenbach, Olusola Ajilore, Michelle Kassel, Laura B. Gabriel, Jon-Kar Zubieta

W16. Neural Substrates of Spatial Working Memory Deficits in Patients with Neurofibromatosis 1 (NF1)

Amira Ibrahim, Caroline A. Montojo, Tena Rosser, Nicole Enrique, Katherine H. Karlsgodt, Alcino Silva, <u>Carrie Bearden</u>

W17. Positive and Negative Symptom Correlates of Second-generation Antipsychotic Adherence in Recent-onset Schizophrenia

> <u>Kenneth Subotnik</u>, Joseph Ventura, Denise Gretchen-Doorly, Gerhard S. Hellemann, Elisha R. Agee, Laurie R. Casaus, John S. Luo, Kathleen F. Villa, Keith H. Nuechterlein

W18. Early Changes in Neural Responses to Emotional Information Predict Clinical Response to SSRI Treatment in Depression

Beata Godlewska, Ray Norbury, Philip Cowen, Catherine J. Harmer

W19. Differential Alterations of Internally-generated Behavioral Responses and Dose-dependent Treatment Effects in Antipsychotic Naïve First Episode Schizophrenia and Psychotic Mood Disorder Patients

<u>Sarah Keedy</u>, James Reilly, Jeffrey R. Bishop, Margret Harris, Peter J. Weiden, John A. Sweeney

W20. Progressive Reduction of Visual P300 Amplitude in Patients with First Episode Schizophrenia

<u>Robert McCarley</u>, Naoya Oribe, Yoji Hirano, Shigenobu Kanba, Elisabetta del Re, Larry J. Seidman, Raquelle Mesholam-Gately, Martha Shenton, Jill M. Goldstein, Kevin M. Spencer, Margaret Niznikiewicz

W21. Adolescents' Neural Response to Personally Relevant Social Reward: A Novel Paradigm with Relevance to Affective Symptoms and Sensation Seeking

Erika E. Forbes, Lisa Sheeber, Nicholas Allen, Jennifer Silk, Marigrace Ambrosia

W22. Startle Latency and Magnitude Predict Clinical Outcome in the Psychosis Prodrome: Findings from the North American Prodromal Longitudinal Study (NAPLS)

> <u>Kristin Cadenhead</u>, Jean Addington, Carrie Bearden, Tyrone Cannon, Barbara A. Cornblatt, Daniel H. Mathalon, Thomas McGlashan, Diana Perkins, Larry J. Seidman, Ming Tsuang, Elaine Walker, Scott Woods

W23. Developmental Trajectory of Facial Emotion Recognition in Normal Development and Across Stages of Schizophrenia

<u>Cheryl M. Corcoran</u>, Pamela Butler Kahn, Elisa Dias, Daniel C. Javitt

W24. Adults Recovered from Anorexia Nervosa Show Altered Brain Response during Delayed Discounting in Fasted and Fed States

<u>Christina Wierenga</u>, Amanda Bischoff-Grethe, Andrew Melrose, Laura Torres, Laura Irvine, Ursula F. Bailer, Walter Kaye

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W25. Implicit Cognition towards Self-injury Among Teenage Suicide Attempters vs. Teens Engaged in Non-suicidal Self-injury

> Daniel P. Dickstein, Megan Puzia, Grace Cushman, Kerri Kim, Karen Seymour, Alexandra Weissman, Matthew Nock, Anthony Spirito

W26. Decision Making in Avoidance-reward Conflict: Behavioral Performance in Non-human Primates and Humans

<u>Darin Dougherty</u>, Amanda R. Arulpragasam, Andrew K. Corse, Navneet Kaur, Demetrio Sierra-Mercado, Tina Chou, Alexandra Rodman, Amanda Duffy, Eric J. McDonald, Christine A. Eckhardt, Emad N. Eskandar, Thilo Deckersbach

W27. The Effects of Tobacco Smoking Status on Theta-gamma Coupling in Patients with Schizophrenia and Non-psychiatric Controls

<u>Mera S. Barr</u>, Kristen M. Mackowick, Reza Zomorrodi, Victoria C. Wing, Caroline Wass, Zafiris J. Daskalakis, Tony P. George

W28. Baseline Neurocognitive Predictors of Responders to Treatment at 12 Month Follow-up for Major Depressive Disorder by Deep Brain Stimulation

> Shane McInerney, Sakina Rizvi, Heather McNeely, Helen S. Mayberg, Lozano Andres, Peter Giacobbe, Joseph Geraci, <u>Sidney</u> <u>Kennedy</u>

W29. Relationship between Frontal P300 Event-related Potential and Brain Glutamine/Glutamate Ratio Measured In Vivo

> <u>Mei-Hua Hall</u>, Jordan W. Smoller, Deborah L. Levy, Mary Lohan, Kevin M. Spencer, Dost Ongur

W30. Ethnic Differences in Physiological Responses to Fear Conditioned Stimuli

Karen G. Martinez, Jose A. Franco, Mohammed R. Milad, Gregory J. Quirk

W31. Preventing the Return of Fear Using a Retrieval + Extinction Reconsolidation Update Mechanism: The Integration of Fear-potentiated Startle and US-expectancy Ratings

> <u>Seth D. Norrholm</u>, Victor T. Warren, Kemp Anderson, Cliffe Kwon, Lauren Bosshardt, Tanja Jovanovic, Bekh Bradley, Kerry J. Ressler

W32. Neural Correlates of Relational and Item-specific Encoding and Retrieval (RiSE) in Schizophrenia

<u>J. Daniel Ragland</u>, Charan Ranganath, Deanna M. Barch, James M. Gold, Michael P. Harms, Evan Layher, Angus W. MacDonald, Joshua Phillips, Andrew Poppe, Steve M. Silverstein, Dennis Thompson, Cameron S. Carter

W33. Differential Effects of Aging and High Fat Diet on Cognitive Function in Mice

James Kesby, Svetlana Semenova, Jane J. Kim, Jerrold M. Olefsky, Cristian L. Achim, Virawudh Soontornniyomkij, Benchawa Soontornniyomkij, Dilip V. Jeste

W34. Age-at-Onset and Cognitive Control-related Brain Circuitry in First Episode Schizophrenia

<u>Tara Niendam</u>, Emilio Ferrer, Tyler A. Lesh, Stefania Ashby, Marjorie Solomon, J. Daniel Ragland, Jong Yoon, Michael J. Minzenberg, Cameron S. Carter

W35. Ecologically-valid Assessment of Attention in Schizophrenia in a Virtual Environment

<u>George Foussias</u>, Ishraq Siddiqui, Krysta McDonald, Eliyas Jeffay, Konstantine K. Zakzanis, Gary Remington

W36. Effects of Amphetamine on Encoding and Retrieval of Memory for Emotional Stimuli

Jessica Weafer, David Gallo, Harriet de Wit

W37. Training the Brain to Abstain from Alcohol: Towards the Neural Basis of Approach/Avoidance Training Effects in Hazardous Drinkers

<u>Charles Taylor</u>, Akanksha Shukla, Karalani Cross, Nader Amir, Murray B. Stein, Martin Paulus

W38. Steady-state Gamma-band Responses in Children with Autism Spectrum Disorders during an Auditory Oddball Task

<u>Kristina L. McFadden</u>, Sarah Steinmetz, Susan Hepburn, Jason Tregellas, Donald Rojas

W39. Variation in Serotonin Transporter Gene Predicts Neural Activation in a Response Inhibition Task

Ranjani Prabhakaran, Roberta Rasetti, Ena Xiao, Bhaskar S. Kolachana, Daniel R. Weinberger, Venkata S. Mattay, Karen F. Berman

W40. Fear Learning Deficits in Women with PTSD: Estrogen Is Associated with Extinction of Fear-potentiated Startle but Not Dark-enhanced Startle

<u>Ebony Glover</u>, Vas Michopoulos, Kristina B. Mercer, Seth D. Norrholm, Bekh Bradley, Kerry J. Ressler, Tanja Jovanovic

W41. Pattern Separation Bias Deficit in Patients with Schizophrenia

<u>Theo Van Erp</u>, Adrian Preda, Steven Potkin, Lawrence Faziola, Lisa Thom, Dana Nguyen, Andrea Weideman, Charles Kessler, Craig Stark

W42. Change in Functional Activation during Cognitive Control Across Childhood and Adolescence as Related to Risk Taking Behavior

> <u>Katherine H. Karlsgodt</u>, Bart D. Peters, Toshikazu Ikuta, Pamela DeRosse, Kimberly Cameron, Angelica Bato, Philip R. Szeszko, Anil Malhotra

W43. Learning in the Absence of Intact Cognitive Control: The Neural Substrates of Transitive Inference in Adolescents with Autism Spectrum Disorders

> Marjorie Solomon, Tyler A. Lesh, Tara Niendam, Jonathan Beck, John Matter, Nathan Whitmore, Cameron S. Carter, J Daniel Ragland

W44. Attenuated Behavioral and Brain Responses to Trust Violations among Assaulted Adolescent Girls

Josh Cisler, Jennifer Lenow, Scott Steele, Clinton D. Kilts

W45. Independent and Additive Impact of Co-occurring Anxiety Disorders on Elevated Trait Impulsivity in Bipolar Alcoholics: Implications for Alcoholism Severity and Bipolar Course of Illness

Bryan K. Tolliver, James J. Prisciandaro, Delisa G. Brown, Helena Brenner

W46. Fronto-parietal Dysfunction and Cognitive Control Deficits in Firstepisode Psychosis Patients with Schizophrenia and Bipolar Disorder

> <u>Tyler A. Lesh</u>, Benjamin Geib, Tara Niendam, Michael J. Minzenberg, Jong Yoon, J Daniel Ragland, Marjorie Solomon, Cameron S. Carter

W47. Mismatch Negativity Predicts Psychosis Onset and Is Associated with Plasma Markers of Inflammation in Youth at Clinical High Risk for Psychosis

> Daniel H. Mathalon, Diana Perkins, Kristin Cadenhead, Gregory A. Light, Peter Bachman, Jason Johannesen, Aysenil Belger, Margaret Niznikiewicz, Erica Duncan, Ricardo Carrion, Jean Addington, Tyrone Cannon, Barbara A. Cornblatt, Larry J. Seidman, Elaine Walker, Scott Woods

W48. High-resolution fMRI Reveals Reward Anticipation Signaling of the Substantia Nigra that Is Modulated by Reward Magnitude in Healthy Subjects and Blunted Responses in Schizophrenia

Jong Yoon, Anthony Grandelis, Edward D. Cui, Michael J. Minzenberg, Tara Niendam, J Daniel Ragland, Tyler A. Lesh, Marjorie Solomon, Cameron S. Carter

W49. Acute and Non-acute Effects of Cannabis on Neurocognitive Functioning

April D. Thames, Natalie Arbid

W50. Anger in Body and Brain: Elevated Blood Pressure Impedes Reaction Time and Diminishes Neural Activity in Attention and Visual Areas during a Decision Making Task

Sarah N. Garfinkel, Jos Brosschot, Julian Thayer, Hugo D. Critchley

W51. Input-information Processing during Fear Acquisition in PTSD Using Dynamic Causal Modeling and fMRI

Huijin Song, Mohammed R. Milad

W52. Deficits in Reward Prediction Error Signaling in Cocaine Addiction: Evidence from the Feedback Negativity and Relationship to Recency of Cocaine Use

> <u>Muhammad Parvaz</u>, Anna Konova, Jonathan P. Dunning, Greg H. Proudfit, Pias Malaker, Scott J. Moeller, Nelly Alia-Klein, Rita Goldstein

W53. Genetic Factors Contributing to Body Weight in Anorexia Nervosa and Bulimia Nervosa

<u>Allan S. Kaplan</u>, Zeynep Yilmaz, Arun K. Tiwari, Robert D. Levitan, Jo Knight, Sara Piran, Sarah Gagliano, Andrew Bergen, Walter H. Kaye, James Kennedy

W54. FKBP5 Moderates Alcohol Withdrawal Severity: Human Genetic Association and Functional Validation in Knockout Mice

Ming-Chyi Huang, <u>Melanie L. Schwandt</u>, Julie A. Chester, Aaron M. Kirchhoff, Chung-Feng Kao, Tiebing Liang, Jenica Tapocik, Vijay A. Ramchandani, David T. George, Colin A. Hodgkinson, David Goldman, Markus Heilig

W55. Haplotype and SNP Variation in Genes Implicated in GABA Synthesis, Synaptic Transmission and Re-uptake are Predictors for Alcoholism

<u>Mary-Anne Enoch</u>, Colin A. Hodgkinson, Elena Gorodetsky, Cheryl Marietta, Alec Roy, David Goldman

W56. Role of Genetic Variation in Host-parasite Interaction Associated with Major Mental Illness

Shinichi Kano, Colin A. Hodgkinson, Lorraine V. Jones-Brando, Sharon Eastwood, Koko Ishizuka , Minae Niwa, Alec Roy, Nicola Cascella, Faith Dickerson, Anil Malhotra, David Goldman, Paul J. Harrison, Robert Yolken, Akira Sawa

W57. Genome-wide Association Study of Superior Frontal Volumes in Schizophrenia

<u>Ryota Hashimoto</u>, Masashi Ikeda, Fumio Yamashita, Kazutaka Ohi, Hedenaga Yamamori, Yuka Yasuda, Michiko Fujimoto, Masaki Fukunaga, Kiyotaka Nemoto, Kiyoto Kasai, Norio Ozaki, Nakao Iwata, Masatoshi Takeda

- W58. Withdrawn
- W59. A Trial Matching and Mismatching Ondansetron and Sertraline to 5-HTTLPR Alleles in Non-treatment Seeking Alcohol Dependent Individuals

<u>George Kenna</u>, Lorenzo Leggio, Robert M. Swift, William H. Zywiak, John McGeary, James Clifford, Jessica R. Shoaff, Samuel R. Fricchione, Michael Brickley, Kayla Beaucage, Carolina Haass-Koffler, Cynthia Vuittonet

W60. Monoamine Polygenic Liability in Health and Cocaine Addiction: Imaging Genetics Study

> <u>Scott J. Moeller</u>, Muhammad Parvaz, Elena Shumay, Salina Wu, Nicassia Beebe-Wang, Anna Konova, Michail Misyrlis, Nelly Alia-Klein, Rita Goldstein

W61. Brain eQTLs Shared by Multiple Psychiatric Diseases

<u>Chunyu Liu</u>, Chunling Zhang, Chao Chen, Judith Badner, Ney Alliey-Rodriguez, Elliot Gershon, Eric Gamazon, IOCDF-GC, TSAICG, Nancy J. Cox

W62. Combined Brain Transcriptome Meta-analysis and Genome-wide Association Studies Provide Evidence for Shared Genetic Risk between Depression and Other Brain Disorders

Etienne Sibille, Ying Ding, Lun-Ching Chang, Xingbin Wang, Jean-Philippe Guilloux, Jenna Parrish, Hyunjung Oh, David A. Lewis, George C. Tseng

- W63. A Candidate Gene Analysis of Acoustic Startle Latency and Psychosis Lauren Gensler, Tanja Jovanovic, Alicia K. Smith, Lynn Almli, Seth D. Norrholm, Ebony Glover, Kerry J. Ressler, Bekh Bradley, <u>Erica</u> <u>Duncan</u>
- W64. DSM-IV and DSM-5 Alcohol, Cannabis, and Methamphetamine Use Disorders: Rates, Heritability, and Co-morbidity in an American Indian Community Sample

David Gilder, Ian Gizer, Cindy L. Ehlers

W65. A Second Large-scale Candidate Gene Analysis of Endophenotypes for Schizophrenia Further Implicates the Glutamate and Neuregulin-ErbB4 Signaling Pathways

<u>Tiffany A. Greenwood</u>, Gregory A. Light, Neal R. Swerdlow, David L. Braff

#### Poster Session III—Wednesday

W66. An Interactive Effect of the Two SNPs in the Catechol-O-Methyltransferase (COMT) Gene on Dopamine Concentration in the Prefrontal Cortex

Elena Shumay, Joanna Fowler, Nora D. Volkow

W67. Differential Allelic Expression and cis-regulatory Sites at Human Neuronal Genes

<u>Qiaoping Yuan</u>, Seungeun Yeo, Zhifeng Zhou, Colin A. Hodgkinson, David Goldman

W68. Application of Sequencing, Fatty Acid Profiling, and Metabolomics Investigations to Explore Pathogenesis and Treatment Strategy for Anorexia Nervosa

> <u>Pei-an Betty Shih</u>, Jun Yang, Christophe Morisseau, Ashley Van Zeeland, Toni-Kim Clarke, Andrew W. Bergen, Pierre Magistretti, Katherine Ann Halmi, Wade Berrettini, Nicholas Schork, Walter H. Kaye, Bruce D. Hammock

W69. Evidence for Epistatic Interactions between ANK3 and Voltage Gated Ion Channels in Influencing Schizophrenia Risk

Rebecca Birnbaum, Fengyu Zhang, Daniel Weinberger

W70. Suicidal Ideation and Suicidality in an American Indian Community: Comorbidity with Trauma Exposure, ASPD, Affective Disorders and Drug Dependence

Cindy L. Ehlers, David Gilder

W71. Whole Genome Sequencing of Schizophrenia in a Founder Population <u>Todd Lencz</u>, Semanti Mukherjee, Shai Carmi, Anil Malhotra, Itsik Pe'er, Ariel Darvasi

W72. A Genome-wide Association Study on Antipsychotic-induced Body Weight Gain Dissecting the CATIE Sample

> Daniel J. Mueller, Eva J. Brandl, Arun K. Tiwari, Clement C. Zai, Nabilah I. Chowdhury, Tamara Arenovich, Jiangshan J. Shen, James L. Kennedy

W73. Pharmacogenetics of Obsessive-compulsive Disorder Candidate Genes and Antidepressant Response

> <u>Gwyneth Zai</u>, Clement C. Zai, Vanessa Goncalves, Eva J. Brandl, Karen Wigg, James L. Kennedy, Peggy M.A. Richter

W74. The Genetic Basis of Neurocognitive Decline and Reduced White-matter Integrity in Normal Human Brain Aging

<u>David C. Glahn</u>, Jack Kent, Rene L. Olvera, Laura Almasy, Peter Kochunov, Ravi Duggirala, John Blangero

W75. Variation in the ZNF804A Gene Is Associated with Striatal Presynaptic Dopamine Function

<u>Catherine E. Hegarty</u>, Daniel P. Eisenberg, Philip D. Kohn, Daniel R. Weinberger, Joseph C. Masdeu, Karen F. Berman

W76. Pharmacoepigenetics of Depression - A Role of Monoamine Oxidase A DNA Hypomethylation?

<u>Katharina Domschke</u>, Nicola Tidow, Jürgen Deckert, Volker Arolt, Peter Zwanzger, Bernhard Baune

W78. High Transcriptional Plasticity of the AKT1 Gene Is Revealed by RNA Sequencing Analysis in the Brain

<u>Gianluca Ursini</u>, Joo Heon Shin, Bin Xie, Giovanna Punzi, Yuan Gao, Joel E. Kleinman, Thomas M. Hyde, Keri Martinowich, Daniel R. Weinberger

W79. Oxytocin and Vasopressin Peptide Gene Region: Associations with Autism Related Phenotypes

Sunday Francis, Emily Kistner-Griffin, Guter Stephen, Edwin H. Cook, <u>Suma Jacob</u>

W80. Association of SCN2A Variants with Cognitive Ability in Schizophrenia, and Additional Support from Analyses of Unaffected Siblings, Independent Schizophrenia Samples, fMRI, and mRNA Expression in Brain

> <u>Dwight Dickinson</u>, Richard Straub, Joey W. Trampush, Yuan Gao, Ningping Feng, Gianluca Ursini, Kristin Bigos, Bhaskar Kolachana, Ryota Hashimoto, Masatoshi Takeda, Dan Rujescu, Joseph H. Callicott, Thomas M. Hyde, Karen F. Berman, Joel E. Kleinman, Daniel R. Weinberger

W81. Variation in the Williams Syndrome GTF2i Gene and Anxiety-proneness Interactively Predict DLPFC Response to Aversive Social Stimuli in Humans

> <u>Mbemba Jabbi</u>, Qiang Chen, Nicholas Turner, Michael White, J. Shane. Kippenhan, Philip Kohn, Dwight Dickinson, Bhaskar Kolachana, Venkata Mattay, Daniel R. Weinberger, Karen Berman

W82. Generation of Serotonin Transporter Knock-in Mice Carrying Ile425Val Coding Variant Associated with Obsessive-compulsive Disorder and Tourette Disorder

> Sammanda Ramamoorthy, Padmanabhan Mannangatti, Kamalakkannan Naidu, Lankupalle Jayanthi, Dennis L. Murphy

W83. New Insight into Genetic Mechanism Underlying the Treatment Effect of Obsessive-compulsive Disorder Using SSRIs

Haide Qin, Jack Samuels, Ying Wang, Gerald Nestadt, Yin Yao

W84. The Utility of DNA Extracted from Saliva for Methylation Studies of Psychiatric Traits

> <u>Alicia K. Smith</u>, Varun Kilaru, Torsten Klengel, Kristina B. Mercer, Karen Conneely, Kerry J. Ressler, Elisabeth Binder

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W85. ADRB2 Gene Polymorphism Interacts with Childhood Trauma in Conveying Risk for Adult Posttraumatic Stress Disorder (PTSD)

<u>Anthony P. King</u>, Kerry J. Ressler, Lynn Almli, Greg Cohen, Marijo Tamburrino, Sandro Galea, Joseph R. Calabrese, Israel Liberzon

W86. Variation in the Human Fatty Acid Amide Hydroxylase (FAAH) Gene and Threat Processing

Francisco J. Amador, Andrew Holmes, Carmen L. Cadilla, Mohammed R. Milad, Karen G. Martinez, Gregory J. Quirk

W87. Telomere Length Measurements in Post-mortem Human Brain in Major Depressive Disorder

<u>Firoza Mamdani</u>, Brandi Rollins, William E. Bunney, Richard M. Myers, Jack David Barchas, Alan F. Schatzberg, Stanley J. Watson, Huda Akil, Marquis P. Vawter, Pedro A. Sequeira

W88. Dynamics of Transcriptional Coexpression Brain Networks Over the Human Lifespan

<u>Claudia C. Wehrspaun</u>, Wilfried Haerty, Danielle Bassett, Joo Heon Shin, Daniel R. Weinberger, Chris Ponting

W89. Large-scale RNA-sequencing of Schizophrenia Brains by the CommonMind Consortium

Pamela Sklar

W90. Pharmacogenetics of Growth Effects Complicating ADHD Treatment

<u>Erika L. Nurmi</u>, Allyson Mallya, Karyn S. Mallya, Gerhard S. Hellemann, James McGough, Sandra K. Loo, Robert M. Bilder, James T. McCracken

W91. Obsessive-compulsive Traits in Children and Adolescents from the General Population: A Genome-wide Association Study

<u>Paul D. Arnold</u>, Christine Burton, Laura Park, Bingbin Li, S-M Shaheen, Vanessa Sinopoli, Annie Dupuis, Andrew Paterson, Jennifer Crosbie, Russell Schachar

W92. Leveraging Hyperserotonemia and Whole Exome Sequencing in Autism Families to Identify Genetic Risk Factors

James S. Sutcliffe, Nicholas Campbell, Emily L. Crawford, Bingshan Li, Lea K. Davis, Nancy J. Cox, Edwin H. Cook

W93. A Genome-wide Association Study Identifies a Genetic Locus in the GRB10-amino Acid Decarboxylase Region of 7p12.2 Associated with Caucasian Treatment Resistant Schizophrenia Patients

Herbert Y. Meltzer, Jiang Li

W94. Characterization of Autism-associated De Novo Mutations Impacting Integrin Receptor Subunit Genes

James S. Sutcliffe, Emily L. Crawford, Keaton Wadzinski, Ana Carneiro

W95. RNA Editing Levels of 5-HT2C and GluA2 Are Increased in Suicides with Major Depression

Monsheel Sodhi, Thomas M. Hyde, Stefan Green, Joel E. Kleinman

- W96. Deep RNA Seq Characterization of Novel Transcripts in CACNA1C <u>Joo Heon Shin</u>, Dewey Kim, Joshua Hurtado, Bin Xie, Thomas M. Hyde, Joel E. Kleinman, Daniel R. Weinberger
- W97. Genetic Pathway Analyses of the Endocannabinoid System in a Sample of Social Drinkers and Treatment-seeking Alcoholics

<u>Jia Yan</u>, Bethany L. Stangl, Mark A. Reimers, Melanie L. Schwandt, Hui Sun, Colin A. Hodgkinson, David Goldman, Daniel W. Hommer, David T. George, Kenneth S. Kendler, Markus Heilig, Vijay A. Ramchandani

W98. Clinical and Genetic Predictors of Length of Sobriety in Alcoholics Treated with Acamprosate

> Joanna Biernacka, Jennifer Geske, Gregory Jenkins, Mark A. Frye, Doo-Sup Choi, Daniel Hall-Flavin, Terry Schneekloth, Falk Kiefer, Karl F. Mann, Victor Karpyak

W99. Alterations of BDNF Signaling and Splicing in Violent Suicide

<u>Giovanna Punzi</u>, Gianluca Ursini, Kristen Maynard, Joo Heon Shin, Bin Xie, Yuan Gao, Joel E. Kleinman, Thomas M. Hyde, Keri Martinowich, Daniel R. Weinberger

W100. Potential Methylation in the 5HT System: Analysis in Suicidal Behavior

<u>Vincenzo de Luca</u>, Ali Bani-Fatemi, Jiali Song, Aaron Howe, Nuwan Hettige, Ahmed Hassan

W101. Discovery and Validation of Blood Biomarkers for Suicidality

<u>Alexander B. Niculescu</u>, Helen Le-Niculescu, Daniel Levey, Mikias Ayalew, Nitika Jain, Evan Winiger, Ganesh Shankar, Mark Radel, Elizabeth Belanger, Hilary Duckworth, Robert Schweitzer, Michael Yard, George Sandusky, Anantha Shekhar, Nicholas Schork, Daniel Salomon

W102. Growth Factors as Biomarkers of Major Depressive Disorder and Potential Predictors of Antidepressant Drug Response

> <u>Angelos Halaris</u>, Anne Clark-Raymond, Edwin Meresh, Aparna Sharma, Robin Kang, Brandon Hage, Kathryn Morrissey, Jawed Fareed, Ghanshyam Pandey

W103. Task-related Brain Activation in Subjects with Chronic, Stable Schizophrenia and the Effects of a Single-dose α-7 Nicotinic Acetylcholine Receptor Agonist (AQW051): A Placebo-controlled, Double-blind, Randomized Study

<u>Deanna M. Barch</u>, Stephen R. Marder, Michael P. Harms, Lars F. Jarskog, Will Cronenwett, Li-Shiun Chen, Markus Weiss, Ralph P. Maguire, Nicole Pezous, Dominik Feuerbach, Cristina Lopez-Lopez, Rhett B. Behrje, Baltazar Gomez-Mancilla, Robert W. Buchanan

W104. Opioid Antagonism Decreases Hedonic Responses to Social Stimuli in Healthy Adults

Margaret C. Wardle, Anya K. Bershad, Matt Pulaski, Harriet de Wit

W105. Inpatient Resource Utilization and Cost-related Benefits of Long-acting Injectable Antipsychotics Across Different Age Groups of Medicaidinsured Schizophrenia Patients

<u>Craig Karson</u>, Steve Offord, Ross A. Baker, Anna Eramo, Jay Lin, Siddhesh Kamat

W106. A Trial of D-cycloserine to Treat the Social Deficit in Older Adolescents and Young Adults with Autism Spectrum Disorders

Maria R. Urbano, Leonore Okwara, Paul Manser, Kathrin Hartmann, Stephen Deutsch

W107. Selective Effects of the 5-HT2C Receptor Agonist Metachlorophenylpiperazine (mCPP) on Intake of a Palatable Snack Food in Healthy Female Volunteers: Correlation with Regional Brain Activations Measured by BOPLD fMRI

Colin T. Dourish, Jason M. Thomas, Suzanne Higgs

W108. Measurement of Immune Activation via Blood Gene Expression Early and Accross Treatment of Major Depressive Disorder

> <u>Marisa Toups</u>, Thomas Carmody, Cobi Heijnen, Robert Dantzer, Madhukar Trivedi

W109. Pregnenolone for Depression in Outpatients with Bipolar Disorder

<u>E. Sherwood Brown</u>, John Park, Christine E. Marx, Linda Hynan, Domingo Davila, Alyson Nakamura, Prabha Sunderajan, Alexander Lo, Traci Holmes

W110. Marijuana Withdrawal and Relapse in the Human Laboratory: Effect of Zolpidem Alone and in Combination with Nabilone

Margaret Haney, Ziva Cooper, Gillinder Bedi, Stephanie Collins Reed, Divya Ramesh, Richard W. Foltin

W111. Relationship between Tobacco Consumption and Lifetime Cannabis Use Status in Outpatients with Schizophrenia

Tony P. George, Rachel A. Rabin

W112. Intravenous Methamphetamine Self-administration by Humans in a Modified Progressive-ratio Paradigm

<u>Rajkumar J. Sevak</u>, Carmen Freire-Cobo, Eric Wagreich, Edythe D. London

W113. Topiramate Treatment of Heavy Drinkers: Moderation by a GRIK1 Polymorphism

> <u>Henry Kranzler</u>, Jonathan Covault, Richard Feinn, Stephen Armeli, Howard Tennen, Albert Arias, Joel Gelernter, Timothy Pond, Cheryl Oncken, Kyle Kampman

W114. Onset of Efficacy of Long-acting Injectable Paliperidone Palmitate for Negative Symptoms and Anxiety/Depression in Subjects with Schizophrenia

Dong-Jing Fu, Cynthia A. Bossie, Jennifer Kern Sliwa, Yi-Wen Ma, Larry Alphs

W115. Effects of Varenicline on Neural Correlates of Motivation for Alcohol in Heavy Drinkers: The Alcohol-food Incentive Delay Task

> Vatsalya Vatsalya, Reza Momenan, Melanie L. Schwandt, Marion Coe, Selena Bartlett, Daniel W. Hommer, Markus Heilig, <u>Vijay A.</u> <u>Ramchandani</u>

W116. Treatment with Paroxetine Increases Levels of Nociceptine in Cerebrospinal Fluid in Females with Fibromyalgia

Lars H. Tanum, Morten Vinje, Gunnar Ordeberg, Fred Nyberg

W117. Efficacy of Lurasidone in the Treatment of Schizophrenia with Prominent Negative Symptoms: A Post-hoc Analysis of Short-term Trials

> <u>Nina R. Schooler</u>, Andrei Pikalov, Jay Hsu, Josephine Cucchiaro, Robert Goldman, Antony Loebel

#### Poster Session III—Wednesday

W118. Critical Testing of the Alcohol Incentive-sensitization Model in Young Heavy Binge Drinkers Developing Symptoms of Alcohol Use Disorder

Andrea King, Patrick McNamara, Dingcai Cao

W119. Is Quality of Life Related to Cognitive Performance or Negative Symptoms in Patients with Schizophrenia? Results from a Double-blind, Active-controlled, Lurasidone Extension Study

> <u>Philip D. Harvey</u>, Antony Loebel, Josephine Cucchiaro, Debra Phillips, Cynthia Siu

W120. Abuse Potential of Intranasal Buprenorphine versus Buprenorphine/ Naloxone in Buprenorphine-maintained Heroin Users

Jermaine D. Jones, Maria A. Sullivan, Jeanne M. Manubay, Shanthi Mogali, Verena Metz, Sandra D. Comer

W121. Positive Symptoms Respond to Add-on Aspirin in Schizophrenia Patients with High Sera CRP Levels: A Post-hoc Analysis of an RCT

> Mark Weiser, Shimon Burshtein, Liana Fodoreanu, Roxana Chiriță, Ghiorghe Talău, Diana Cirjaliu, Naama Fund, Robert Yolken, John M. Davis, M.D, <u>Michael Davidson</u>

W122. Pharmacogenetics of CYP2C19 and Response to Escitalopram in Autism Spectrum Disorders (ASD)

<u>Jeffrey R. Bishop</u>, Fedra Najjar, Thomas Owley, Guter Stephen, Edwin H. Cook

W123. Implementation of Metabolic Monitoring Guidelines for Patients Receiving Antipsychotic Medications in a Large Outpatient Psychiatry Clinic: Interventions and Outcomes

Jayesh Kamath, Rana Singh, Xuesong Chen, Helen Wu

W124. Which Schizophrenia Subjects Relapse Despite Adherence to Longacting Antipsychotic Therapy?

> <u>Henry Nasrallah</u>, Ibrahim Turkoz, Cynthia A. Bossie, Dong-Jing Fu, Srihari Gopal, Larry Alphs, David Hough

W125. Plasma Oxytocin Concentrations Following MDMA or Intranasal Oxytocin in Humans

<u>Matthew G. Kirkpatrick</u>, Sunday M. Francis, Suma Jacob, Royce J. Lee, Harriet de Wit

W126. Effects of MDMA and THC on Social Subjective States and Social Processing in Humans

<u>Gillinder Bedi</u>, Daniel Burghart, Jenny Porter, Nicholas T. Van Dam, Kevin N. Ochsner, Margaret Haney

W127. Pro-attentional Effects of Amphetamine in Healthy Adults Are Predicted by Levels of Sensorimotor Gating

<u>Neal R. Swerdlow</u>, Savita G. Bhakta, Jo A. Talledo, Sarah N. Lamb, Bryan Balvaneda, Justin Kei, Hsun-Hua Chou

W128. Cariprazine Demonstrates High Dopamine D3 and D2 Receptor Occupancy in Patients with Schizophrenia: A Clinical PET Study with [11C]-(+)-PHNO

<u>Mark Slifstein</u>, Anissa Abi-Dargham, Deepak C. D'Souza, Richard E. Carson, István Laszlovszky, Suresh Durgam, Nika Adham, Béla Kiss, István Gyertyan, Margit Kapás, Yih Lee

W129. Lack of Subjective Abuse-related Effects of Intranasal Eluxadoline: A Novel Mu-delta Opiate Modulator for Oral Use in IBS-d

> Naama Levy-Cooperman, Gail McIntyre, Laura Bonifacio, Mike Davenport, Paul Covington, Scott Dove, June Almenoff, Bijan Chakraborty, Kerri A. Schoedel, Michael McDonnell, <u>Edward M.</u> <u>Sellers</u>

W130. The Effects of 5HT-7 Antagonism on Sleep in Humans: A Placebocontrolled Cross-over Study of Lurasidone

Andrew Krystal, Gary Zammit, Andrei Pikalov, Antony Loebel

W131. Trajectories of Response to Repeat Dose of Intravenous Subanesthetic Ketamine in Treatment Resistant Depression

Paulo R. Shiroma, Brian Johns, Michael Kuskowski, Paul Thuras, Kelvin O. Lim

- W132. Cognitive Training with Pharmacological Enhancement in Schizophrenia Ana D. Stan, Debra Bushong, Binu Thomas
- W133. Adverse Childhood Experiences Predict Heavier Drinking and Greater Alcohol Intake during Intravenous (IV) Alcohol Self-administration in Non-dependent Drinkers

<u>Bethany L. Stangl</u>, Melanie L. Schwandt, Laura E. Kwako, Jia Yan, Molly Zametkin, Vijay A. Ramchandani

W134. Δ<sup>9</sup>-THC Attenuates and d-amphetamine Potentiates Responses to a Psychosocial Stressor

Emma L. Childs, Harriet de Wit

W135. Meta-analysis: Response Curve to SSRIs in OCD

Michael H. Bloch, Yasmin Issari, Ewgeni Jakubovski

W136. A Pilot Study of a Dopamine β-hydroxylase Inhibitor, Nepicastat, in the Treatment of PTSD with Genotype Outcome Analysis

Lori Davis, David P. Graham, Hamner Mark, David Nielson, Thomas Kosten, Iouri Makotkine, Rachel Yehuda

W137. Preliminary Investigation of EEG Predictors in an Open-label, Flexibledose, Repeated Infusions of Ketamine as Augmentation in Treatment Resistant Depression

> <u>Cristina Cusin</u>, Matthias Eikermann, Sebastian Zaremba, Kara Pavone, Kelley Durham, Trina Chang, Paolo Cassano, Christina Dording, David Soskin, David Mischoulon, Maurizio Fava

W138. Intranasal Methamphetamine Self-administration in Humans during D-amphetamine Maintenance

<u>Paul Glaser</u>, Erika Pike, Lon Hays, William W. Stoops, Craig R. Rush

W139. Lurasidone in the Treatment of Early-stage Schizophrenia: A Post-hoc Analysis of Three Pooled Acute Treatment Studies

> <u>Jeffrey Lieberman</u>, Andrei Pikalov, Jay Hsu, Josephine Cucchiaro, Fred Grossman, Antony Loebel

W140. Perceptions of Obsessive Compulsive Disorder and Potential Impact on Treatment Outcome

<u>Michael Van Ameringen</u>, William Simpson, Beth Patterson, Jasmine Turna

W141. Do We Know Why There Are Regional Differences in Signal Detection in Global Neuroscience Clinical Trials?

<u>Amir Kalali</u>

W142. Trajectory of Neurocognition in First-episode Schizophrenia

Joey W. Trampush, Delbert G. Robinson, Todd Lencz, John Kane, Anil Malhotra, Terry E. Goldberg, Danielle Beech

- W143. Withdrawn
- W144. Chronic High Dose Adjunctive Intranasal Oxytocin in Schizophrenia Patients

David Feifel, Kai MacDonald, Cobb Patrice, Rebecca McKinney

W145. The Impact of Cocaine Use Patterns, Demographic and Mood Variables, and Addiction Severity on Neurocognitive Functioning in Individuals with Cocaine Use Disorders

> James Mahoney, Ari Kalechstein, Christopher D. Verrico, Tabish Iqbal, Thomas Newton, Richard De La Garza

W146. Efficacy and Safety of Treatment with Lurasidone Adjunctive to Lithium or Valproate in Bipolar I Depression: Results of Two 6-week Studies

Joseph R. Calabrese, Trisha Suppes, Kaushik Sarma, Robert Silva, Hans Kroger, Josephine Cucchiaro, Andrei Pikalov, Antony Loebel

W147. Lurasidone Adjunctive Therapy with Lithium or Valproate for the Treatment of Bipolar I Depression: A Randomized, Double-blind, Placebo-controlled Study (PREVAIL 3)

<u>Trisha Suppes</u>, Joseph R. Calabrese, Robert Silva, Hans Kroger, Josephine Cucchiaro, Andrei Pikalov, Antony Loebel

W148. Lurasidone in Bipolar I Depression: A 24 Week, Open-label Extension Study

<u>Terence A. Ketter</u>, Kaushik Sarma, Robert Silva, Jane Xu, Josephine Cucchiaro, Antony Loebel

W149. Short- and Longer-term Treatment with Lurasidone in Patients with Bipolar I Depression: Effect on Metabolic Syndrome

> <u>Susan McElroy</u>, Andrei Pikalov, Josephine Cucchiaro, Jay Hsu, Hans Kroger, Debra Phillips, Antony Loebel

W150. Early Improvement Predicts Endpoint Response to Lurasidone in Schizophrenia: Pooled Analysis of Five Double-blind Trials

> <u>Christoph U. Correll</u>, Andrei Pikalov, Jay Hsu, Josephine Cucchiaro, Robert Goldman, Antony Loebel

W151. Neurocognitive Impairments as Putative Predictors of Neurolepticinduced Movement Disorders in People with Schizophrenia

Anthony Ahmed

W152. Loss of Neural Signals Related to Cognitive Flexibility in the Rostral Caudate Following Short-term Cocaine Self-administration

Brianna Sleezer, Benjamin Hayden

W153. Long-term Reduction of Cocaine Seeking in Rats and Monkeys by Viral Vector-delivered Cocaine Hydrolase (CocH)

Marilyn E. Carroll, Natalie E. Zlebnik, Yang Gao, Stephen Brimijoin, Ph.D.

W154. The Neurokinin-1 Receptor Mediates Stress-induced Reinstatement to Alcohol and Cocaine Seeking

> Jesse R. Schank, Courtney King, Kejun Cheng, Kenner C. Rice, David Weinshenker, Jason P. Schroeder, Markus Heilig

W155. Chronic Nicotine Treatment Differentially Alters the Discriminative Stimulus Effects of Nicotine and Varenicline in Rhesus Monkeys

Colin S. Cunningham, Lance McMahon

W156. Antidepressant-like Effects of Buprenorphine Are Primarily Mediated through the Kappa Opioid Receptor

Edgardo Falcon, Irwin Lucki

W157. Antidepressant and Anti-inflammatory Properties in the Action of Agomelatine

Raffaella Molteni, Flavia Macchi, Andrea Carlo Rossetti, Elisa Colombo, Mario Dell'Agli, Marco A. Riva, <u>Giorgio Racagni</u>

W158. Agomelatine Treatment Induces Early and Time-dependent Modulation of Rat Hippocampal MiRNome

Daniela Tardito, Mara Seguini, Alessandra Mallei, Maurizio Popoli, Giorgio Racagni

W159. Lurasidone Exerts Antidepressant Properties in the Chronic Mild Stress Model through the Regulation of Synaptic and Neuroplastic Mechanisms in the Prefrontal Cortex

> <u>Marco A. Riva</u>, Flavia Macchi, Mariusz Papp, Giorgio Racagni, Raffaella Molteni

W160. Selective Orexin-2 Receptor Antagonism as Adjunctive Therapy for Insomnia in Depression

<u>Timothy Lovenberg</u>, Jonathan Shelton, SuJin Yun, Pascal Bonaventure, Brock Shireman, Christine Dugovic

W161. Selective Blockade of 2-arachidonoylglycerol Hydrolysis Affects Learning and Memory Performance While Slowing Down Epileptogenesis in Rodents

> <u>Guy Griebel</u>, Philippe Pichat, Sandra Beeské, Bruno Biton, Dominique Françon, Richard Alonso, Dmitri Wiederschain, Heike Arlt, Bingzhi Zhang, Patrick Avenet, George F. Koob, Johanna Escoubet

W162. Behavioral Effects of the Cannabinoid CB1 Receptor Negative Modulator ORG27569 in Rats

Yuanyuan Ding, Yanyan Qiu, Yanan Zhang, Jun-Xu Li

W163. Discovery and Characterization of a G Protein-biased Agonist that Inhibits β-arrestin Recruitment to the D2 Dopamine Receptor

> David R. Sibley, R. Benjamin Free, Lani Chun, Amy Moritz, Brittney Miller, Trevor Doyle, Jennie Conroy, Adrian Padron, Julie Meade, Jingbo Xiao, Yang Han, Lihua Duan, Marc Ferrer, Jonathan Javitch, Noel Southall, Juan Marugan

W164. Morphine-induced Conditioned Place Preference and Effects of Morphine Pre-exposure in Adolescent and Adult Mice

Wouter Koek

W165. Therapeutic Potential of Selective Orexin-1 Receptor Antagonists

<u>Pascal Bonaventure</u>, Diane Nepomuceno, Brian Lord, Leah Aluisio, Ian Fraser, James R. Shoblock, Tamara Berdyyeva, Brock Shireman, Christine Dugovic, SuJin Yun, Jonathan Shelton, Nicholas Carruthers, Timothy Lovenberg

### Poster Session III—Wednesday

W166. Improved Neuritogenesis and Mitochondrial Dynamics by Levetiracetam Might Explain Cognitive Improvement in Brain Aging and Animal Models of Alzheimer's Disease

Walter E. Mueller, Davide Miano, Carola Schiller, Kristina Leuner

W167. Fluoxetine Exposure during Adolescence Alters Responses to Aversive Circumstances in Adulthood

> <u>Sergio Iñiguez</u>, Vincent Vialou, Brandon L. Warren, Lace Riggs, Mary Kay Lobo, Raisa Ahmed, Bryan Cruz, Eric Nestler, Carlos A. Bolanos-Guzman

W168. A Role for Innate Immune Signaling in Microglia in Behavioral Changes Induced by Repeated Social Defeat Stress in Mice

> Xiang Nie, Shiho Kitaoka, Kohei Tanaka, Eri Segi-Nishida, Yuki Imoto, Atsubumi Ogawa, Shuh Narumiya, <u>Tomoyuki Furuyashiki</u>

W169. Effects of Ethanol and Antidepressant on the Platelet BDNF Release Function in the Peripheral Blood: Implication in the Pathogenesis of Psychiatric Disease

> <u>Toshikazu Saito</u>, Eri Hashimoto, Wataru Ukai, Takao Ishii, Yoshiyasu Kigawa, Kengo Furuse, Hanako Tsujino

W170. The M1 Muscarinic Receptor Subtype Regulates the Antidepressant-like Effects of the Rapidly-acting Antidepressant Scopolamine

> <u>Jeffrey M. Witkin</u>, Carl Overshiner, John Catlow, Douglas Schober, Beverly Heinz, Alexander Nikolayev, Tolstikov Vladimir, Wesley Anderson, Richard Higgs, Kuo Ming-Shang, Christian Felder

W171. Adolescent Cannabis Exposure Differentially Affects Heroin Reinforcement and Accumbens Dopamine Transmission in Lewis and Fisher344 Rats

Gaetano Di Chiara, Cristina Cadoni, Daniele Lecca, Sandro Fenu

W172. Therapeutic Effects of TrkB Ligands on Depression-like Behaviors and Dendritic Changes in the Hippocampus and Nucleus Accumbens after Inflammation

> <u>Kenji Hashimoto</u>, Ji-Chun Zhang, Jin Wu, Qian Ren, Suxia Li, Yukihiko Shirayama

W173. Chronic Methamphetamine-induced Recognition Memory Deficits Are Associated with Impaired Long-term Depression and Decreased glun2b Surface Expression in the Perirhinal Cortex

> Michael D. Scofield, Heather Trantham-Davidson, Marek Schwendt, Ronald E. See, <u>Carmela M. Reichel</u>

W174. Methylphenidate Enhancement of Early-stage Sensory Signal Processing

Barry Waterhouse, Rachel Navarra, Gerard Zitnik, Brian Clark

W175. Optogenetic Control of Central Serotonergic Neurons Affects Anxiety and Impulsivity

<u>Yu Ohmura</u>, Kenji Tanaka, Iku Tsutsui-Kimura, Akihiro Yamanaka, Tomomi Tsunematsu, Mitsuhiro Yoshioka

W176. Ketamine Is a Potent Antidepressant in Two Rodent Models of Depression

<u>Aleksander Mathé</u>, Vasco Sousa, Christina Weide Fischer, Tiberiu Loredan Stan, Gregers Wegener, Andreas Lennartsson, Per Svenningsson

W177. What's Serotonin Got to Do with It? Studies on the Actions of SSRIs and Cocaine in SERT M172 Mice

Linda D. Simmler, Alexander G. Nackenoff, Sonja J. Stutz, Noelle Anastasio, Kathryn Cunningham, Randy D. Blakely

W178. Endocannabinoid Elevation Reverses Social Withdrawal and Normalizes Neuronal Activation Patterns in the PCP Rat Model of Schizophrenia Julien Matricon, Alexandre Seillier, Andrea Giuffrida

- W179. Inhibition of Select Bromodomain Proteins Attenuates Cocaine Reward Gregory C. Sartor, Shaun P. Brothers, Claes Wahlestedt
- W180. Suppression of Drug-evoked Nucleus Accumbens Dopamine by Somatic Hyperpolarization

James E. McCutcheon, Samantha M. Fortin, Jackson J. Cone, Christopher G. Sinon, Ilana B. Witten, Karl Deisseroth, Garret D. Stuber, Mitchell F. Roitman

W181. Glucagon-like Peptide-1 Receptor Activation Reduces Cocaine Reward

<u>Gregg Stanwood</u>, Devon Graham, India Reddy, Lynette Daws, Aurelio Galli

W182. Lithium Ameliorates Rotenone-induced Methylation and Hydroxymethylation of DNA in Cortical Primary Neurons

Gustavo Scola, L. Trevor Young, Helena Kim, Mirian Salvador, <u>Ana</u><u>Andreazza</u>

W183. A Microarray and Proteomics Study of Lithium-treated Mice and Knockout Mice with Lithium-like Behavior Reveals a Common Effect on Mitochondrial Function and Autophagy

> <u>Galila Agam</u>, Lilach Toker, Yuly Bersudsky, R.H. Belmaker, Inbar Plaschkes, Vered Chalifa-Caspi, Dieder Moechars, Roberto Buccafusca, Gerard T. Berry

W184. ORM-12741: Receptor Pharmacology of a Novel Alpha2C-adrenergic Receptor Subtype Selective Antagonist with Multi-therapeutic Potential

> Jukka Sallinen, Juha Rouru, Jyrki Lehtimaki, Päivi Marjamäki, Merja Haaparanta-Solin, Eveliina Arponen, Semi Helin, Olof Solin, Frank Tarazi, <u>Mohammed Shahid</u>

W185. Differential Effects of Vilazodone vs. Citalopram and Paroxetine on Serotonin Transporters and Receptors

Yong Kee Choi, Ronald Oosting, Pradeep Banerjee, Frank Tarazi

W186. A Progressive Ratio Determination of the Relative Reinforcing Effect of Methylphenidate versus Cocaine by Intravenous Self-administration Testing in Rats

David J. Heal, Niki Buckley, Emma L. Johnson, Jane Gosden, Sharon L. Smith

W187. Neonatal SSRI Exposure Alters Neurodevelopment and Risk for Depression in Model Rats

<u>Sarah M. Clinton</u>, Matthew E. Glover, Phyllis C. Pugh, Joshua Cohen, Huda Akil

W188. Effects of Buprenorphine and ALKS 33, Alone and in Combination, on Monoamine Release within the Nucleus Accumbens Shell and Medial Prefrontal Cortex of Male Wistar Rats

Daniel R. Deaver, Jacobi I. Cunningham, Reginald L. Dean, Mark Todtenkopf, David J. Eyerman

W189. Investigating the Interaction between Lisdexamfetamine and S-citalopram on Monoamine Neurochemistry by Dual-probe Microdialysis in Freely Moving Rats: Evidence for Synergistic Augmentation of Serotonin and Dopamine Efflux

Pete Hutson, Helen Rowley, Rajiv Kulkarni, David J. Heal

W190. GLYX-13, a NMDA Receptor Glycine-site Functional Partial Agonist, Produces Long Lasting Antidepressant-like Effects through Modulation of Long-term Synaptic Plasticity

> <u>Jeffrey Burgdorf</u>, Xiao-lei Zhang, Amanda Gross, Roger Kroes, Patric Stanton, J. David Leander, Ronald M. Burch, Joseph Moskal

W191. Levomilnacipran Inhibits Both Norepinephrine and Serotonin Reuptake Across the Clinical Dose Range

Joann O'Connor, Laishun Chen, Carl Gommoll, Stephen R. Zukin

### Poster Session III—Wednesday

W192. Human Neuronal Precursors: Melatonin Abolishes Cytoskeletal Alterations and Promotes Neuronal Development in Olfactory Neuroepithelial Cells Obtained from Schizophrenic Subject

> <u>Gloria Benítez-King</u>, Tania Galván-Arrieta, Carlos Berlanga, Horacio Zamudio-Meza

W193. The Effects of Gene Knockout of the Vesicular Monoamine Transporter 2 (VMAT2; SLC18A2) and the Dopamine Transporter (DAT; SLC3A6) on Ethanol Consumption and Escalation in Mice

Frank S. Hall, Alexandra Houston-Ludlam, Zhicheng Lin, George Uhl

W194. THC Elicits Temporary or Persistent Changes in Expression of Genes Implicated in Neurodevelopment in Adolescent Rat Brain Regions

> <u>Bertha K. Madras</u>, Gregory M. Miller, Lisa Ogawa, Josh Zimmer, Eric Vallender, Yasmin Hurd, Susan Westmoreland

W195. The Role of Efficacy on the Interaction between Mu Opioid Receptor Agonists and Cannabinoid Receptor Agonists

David R. Maguire, Charles P. France

W196. Dissecting Nucleus Accumbens Dynorphin Neurons in Aversion and Reward

Ream Al-Hasani, Jordan G. McCall, Nicole Capik, Blessan Sebastian, Daniel Hong, Audra Foshage, Michael Krashes, Bradford Lowell, Thomas Kash, <u>Michael R. Bruchas</u>

W197. Rapamycin, an Inhibitor of mTORC1 Signaling Activity, Improved Measures of Sociability in the BTBR T+ Itpr3tf/J Mouse Model of Autism Spectrum Disorder

Jessica Burket, Andrew Benson, Amy Tang, Stephen Deutsch

W198. MDPV has Potent and Atypical Effects on Dopamine Release in Adolescent and Adult Rats

Cynthia M. Kuhn, Sabrina Ergun, Elizabeth Sears, Quentin Walker

W199. Chronic Lithium Treatment Diminishes the Amplitude of Electrically Evoked Dopamine Concentration Transients in the Nucleus Accumbens Core

Adem Can, Roger Cachope, Douglas Frost, Joseph Cheer, Todd D. Gould

W200. Effects of Monoamine Releasers with Varying Selectivity to Release Dopamine vs. Norepinephrine in Assays of Cocaine Discrimination and Cocaine vs. Food Choice

<u>Matthew L. Banks</u>, Clayton Bauer, Bruce Blough, Richard B. Rothman, John Partilla, Steve Negus

W201. NS1738, a Positive Allosteric Modulator of Alpha7 Nicotinic Receptors, as Adjunctive Treatment in Schizophrenia: An Experimental Study

> <u>Monica M. Marcus</u>, Åsa Konradsson-Geuken, Kristin Feltmann, Vladimir Ivanov, Björn Schilström, Kent Jardemark, Torgny H. Svensson

W202. Acute Vilazodone Administration Induces Hypothermia in Mice through a 5-HT1A Mechanism

Alvaro Garcia-Garcia, Pradeep Banerjee, E. David. Leonardo

W203. Discovery of Metabotropic Glutamate Receptor Subtype 5 PAMs that Display Stimulus Bias Reveals that In Vivo Efficacy in Animal Models Can be Achieved without Direct Potentiation of NMDAR Currents

> Jerri M. Rook, Paige N. Vinson, Thomas M. Bridges, Shaun R. Stauffer, Ayan Ghoshal, J. Scott. Daniels, Colleen M. Niswender, Hilde Lavreysen, Claire Mackie, Jose Manuel. Bartolome, Gregor J. Macdonald, Thomas Steckler, Carrie K. Jones, Craig W. Lindsley, <u>P. Jeffrey Conn</u>

W204. Want It, Need It, and Can't Control It: The Dynamic Relationship between Impulsivity and the Propensity to Binge Eat

Noelle Anastasio, Kathryn Cunningham

W205. Withdrawn

W206. Electrophysiological Investigation of the Effects of a Subanesthetic Dose of Ketamine on Monoamine Systems

<u>Pierre Blier</u>, Karim El Iskandarani, Chris Oosterhof, Mostafa El Mansari

W207. The Effects of Methylphenidate and Atomoxetine on Glutamate in the Prefrontal Cortex of the Awake Spontaneously Hypertensive Rat Model of ADHD

Paul Glaser, Erin Miller, Greg Gerhardt

W208. Characterization of the Novel M4 Muscarinic Acetylcholine Receptor Positive Allosteric Modulator Vu0467154 in Animal Models of Antipsychotic-like Activity, Cognitive Enhancement and Changes in Sleep-wake Architecture

> <u>Carrie K. Jones</u>, Thomas M. Bridges, Michael Bubser, Robert W. Gould, Ditte Dencker Thorbek, Michael D. Grannan, J. Scott. Daniels, Meredith J. Noetzel, Colleen M. Niswender, Mark E. Duggan, Nicholas J. Brandon, John Dunlop, Michael W. Wood, Craig W. Lindsley, P. Jeffrey. Conn

W209. Hypnotic and Anxiolytic Properties of the Selective Melatonin MT2 Receptor Partial Agonist UCM765

> <u>Stefano Comai</u>, Rafael Ochoa-Sanchez, Quentin Rainer, Gabriella Gobbi

W210. Forebrain-specific CRF Overexpression during Early Life Increases Vulnerability for PTSD-like Symptoms in Adulthood

<u>Mate Toth</u>, Maya Gross, Isabelle Mansuy, Emilio Merlo-Pich, Victoria Risbrough

W211. Transdermal Cannabidiol: Long-lasting Beneficial Actions in Animal Models of Drug Seeking, Anxiety, and Impulsivity

> <u>Friedbert Weiss</u>, Remi Martin-Fardon, Dana Hammell, Stan Banks, Rajita Sinha, Audra Stinchcomb, Gustavo Gonzalez-Cuevas

#### Poster Session III—Wednesday

W212. Intranasal Delivery of an Interfering Peptide with Antidepressant-like Effect

Fang Liu, Virginia Brown

W213. Adenosine Receptor Involvement in Methamphetamine Conditioned Place Preference, Self-administration and Reinstatement

Ryan Bachtell, Kevin Kavanagh, Sophia Levis, Casey O'Neill

W214. Evaluating a Novel Brain-penetrant HDAC Inhibitor in Rat Behavioral Models in Relation to Target Occupancy Assessed by Pet Imaging

<u>Frederick A. Schroeder</u>, Changning Wang, Misha M. Riley, Surya Reis, Yan-Ling Zhang, Stephen J. Haggarty, Jacob M. Hooker

W215. β-arrestin Dependence of the Putative Antipsychotics M100907 and LY379268, in Animal Models of Psychosis

<u>Caitlin E. McOmish</u>, James Hanks, Elizabeth LaMarca, Molly Belkin, Elena Y. Demireva, Jay A. Gingrich

W216. Development of a Novel Class of Antipsychotics: Multifunctional PAT Compounds (5HT2A Antagonists / 5HT2C Agonists) Ameliorate the Positive and Cognitive Disrupting Symptoms Associated with Psychosis

> Drake Morgan, Clinton Canal, Krishnakanth Kondabolu, Myong Kim, Kimberly Robertson, Neil E. Rowland, Glen M. Sizemore, Raymond G. Booth

W217. Chronic Ethanol Increases Excitability in the Ventral Bed Nucleus of Stria Terminalis via Postsynaptic Serotonin2c Receptor Signaling

Catherine Marcinkiewcz, Cayce Dorrier, Thomas L. Kash

W218. Role of Central Amygdala PACAP in the Stress Response

<u>Valentina Sabino</u>, Attilio Iemolo, Riccardo Dore, Xiaofan Wang, Pietro Cottone

### Poster Session III—Wednesday

W219. NMDA Receptors in the Nucleus Accumbens Shell Mediate Compulsive Eating of Palatable Food

> <u>Pietro Cottone</u>, Karen Smith, Rahul Rao, Marta Valenza, Clara Velazquez-Sanchez, Valentina Sabino

W220. Panic Disorder and Agoraphobia: Novel Glutamate Mechanisms and Therapeutic Approaches from Preclinical Model

<u>Anantha Shekhar</u>, Philip L. Johnson, Andrei Molosh, Stephanie D. Fitz, Amy Dietrich, William Truitt, Cris Barnaby, Luc Ver Donck

- W221. Subgrouping Central Serotonin Neurons by Their Networks Yue Ping Guo, <u>Kathryn Commons</u>
- W222. Kappa Opioid Receptors Inhibit Glutamatergic Transmission to the Extended Amygdala in an Input Specific Manner

Thomas L. Kash, Nicole Capik, Michael R. Bruchas

W223. Suicidal Ideation in Depressed New Mothers: Relationship with Childhood Trauma and Sleep Disturbance

Dorothy Sit, James Luther, Jesse Dills, Heather Eng, Dan Buysse, Michele Okun, Stephen Wisniewski, Katherine L. Wisner

W224. Gonadal Hormone Regulation of Stress Circuitry Activity in Healthy Women Is Disrupted in Major Depressive Disorder

> <u>Emily G. Jacobs</u>, Jill M. Goldstein, Laura M. Holsen, Katrina Lancaster, Anne Remington, Stephen Buka, Susan Whitfield-Gabrieli, Anne A. Klibanski

W225. Linked Sex Differences in Cognition and Functional Connectivity in Youth

<u>Theodore D. Satterthwaite</u>, Daniel Wolf, David Roalf, Kosha Ruparel, Guray Erus, Simon Vandekar, Efstathios Gennatas, Mark Elliott, Alex Smith, Hakon Hakonarson, Ragini Verma, Christos Davatzikos, Raquel E. Gur, Ruben C. Gur

#### Poster Session III—Wednesday

W226. Sex Differences in Marijuana's Positive Subjective Effects in Daily Marijuana Smokers

Ziva D. Cooper, Margaret Haney

W227. Sex-specific Behavioral and Neuroanatomical Markers of Susceptibility to Failed Fear Suppression

Tina Gruene, Elian Roberts, Rebecca Shansky

W228. Estrogen Influences C-fos Expression in the Fear Extinction Network in Female Rats

Kara K. Cover, Lisa Maeng, Aaron Landau, Daria Turner, Mohammed R. Milad, <u>Kelimer Lebron-Milad</u>

W229. Response to Yohimbine and Cocaine Cues in Cocaine-dependent Individuals

> <u>Megan Moran-Santa Maria</u>, Aimee McRae-Clark, Nate Baker, Viswanathan Ramakrishnan, Kathleen T. Brady

W230. Sex Differences in Progesterone, Allopregnanolone, and ACTH Responses to Metyrapone in Men and Women with PTSD

> <u>Sabra Inslicht</u>, Erin Madden, Anne Richards, Evelyn Rucker, Aoife O'Donovan, Madhu Rao, Lisa Talbot, Thomas Metzler, Richard Hauger, Thomas Neylan

W231. Diet-induced Obesity Alters Drug Reward Differentially in Males and Females

Sari Izenwasser

W232. Reduced Motivation to Self-administer Methamphetamine by Oxytocin in a Behavioral-economics Paradigm Predicts Reinstatement of Methamphetamine Seeking

Brittney M. Cox, Brandon Bentzley, Carmela M. Reichel, Ronald E. See, Gary Aston-Jones
# Poster Session III—Wednesday

W233. Sex Differences in Corticotropin Release Factor-evoked Anxiety-related Behavior

> <u>Debra Bangasser</u>, Hannah Simko, Adam Hawkins, Brittany Wicks, Rob Cole, Jeremy Schmidt, Michelle Lerner

W234. Contributions of Estrogen and Oral Contraceptive Use to Sex Differences in Functional Responding to Conditioned Cues during Fear Conditioning

Moon Jung Hwang, Huijin Song, Rachel Zsido, Edward F. Pace-Schott, Karen Klahr K. Miller, <u>Mohammed R. Milad</u>

W235. Sex Differences in the Neural Processing of Emotions within the Theoretical Framework of the Circumplex Model of Affect

<u>Jarod Peterson</u>, James Russell, Yuankai Huo, Angela Tseng, Bradley S. Peterson, Zhishun Wang

W236. Adolescent Sex Differences in Fronto-limbic Activity during Selective Attention and Emotion Processing

Crystal E. Schiller, Joshua Bizzell, Sarah Hart, Ayse Belger

W237. Is Alzheimer Disease a Different Disease in Men and Women? Observations from Autopsied Brains and Transgenic Studies

Bradley Chaharyn, Paul Pennington, Kelsey Fehr, Zelan Wei, Jennifer Chlan, <u>Darrell Mousseau</u>

| A     | CNP 52nd Annual Meeting | Final Program |
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| Notes |                         |               |
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## **ACNP 2013 Presenter Disclosures (continued)**

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**Davis, Kenneth**: *Part 1:* My wife, Bonnie M. 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 M. 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 M. 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 M. 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. She receives royalty income from this license, *Part 3:* My wife, Bonnie M. 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. She receives royalty income from this license.

de Wit, Harriet: Part 1: I conducted a research study funded by Unilever in 2011.

**Dourish, Colin**: *Part 1*: Employee, Director and shareholder of P1vital, *Part 2*: Employee, Director and shareholder of P1vital, *Part 3*: Employee, Director and shareholder of P1vital, *Part 5*: P1vital

Dubocovich, Margarita: Part 1: Takeda Pharmaceuticals North America, Inc.

**Duman, Ronald**: *Part 1:* Lilly, Forest, Bristol Myers Sqibb, Taisho, Johnson & Johnson, Pfizer, Lundbeck, *Part 4:* Lilly, Forest, Lundbeck, Johnson & Johnson

Duncan, Erica: Part 4: Grant support from Brain Plasticity, Inc.

**Elliott, Mark**: *Part 4:* The study reported here was sponsored and funded by AstraZeneca Pharmaceuticals.

**Evins, A. Eden**: *Part 1*: Pfizer: Supplemental research support for the NIDA funded trial: R01 DA021245 Extended Duration Varenicline for Prevention of Smoking in Schizophrenia, Envivo Pharmaceuticals: Supplemental research support for the NIDA funded R01 DA030992 Proof of Concept Trial of an Alpha-7 Nicotinic Agonist for Nicotine Dependence, GSK: Supplemental research support for NIDA funded U01 DA019378 Cooperative Drug Discovery Group for Nicotine Dependence, *Part 4*: Pfizer: Supplemental research support for the NIDA funded trial: R01 DA021245 Extended Duration Varenicline for Prevention of Smoking in Schizophrenia, Envivo Pharmaceuticals: Supplemental research support for the NIDA funded trial: R01 DA021245 Extended Duration Varenicline for Prevention of Smoking in Schizophrenia, Envivo Pharmaceuticals: Supplemental research support for the NIDA funded R01 DA030992 Proof of Concept Trial of an Alpha-7 Nicotinic Agonist for Nicotine Dependence, GSK: Supplemental research support for NIDA funded U01 DA019378 Cooperative Drug Discovery Group for Nicotine Dependence, GSK: Supplemental research support for Nicotine Dependence Drug Discovery Group for Nicotine Dependence

Fan, Xiaoduo: *Part 1*: Eli Lilly - advisory board, *Part 4*: Eli Lilly - investigator initiated clinical trial grant

Fava, Maurizio: Part 1: Advisory/Consulting: Abbott Laboratories; Affectis Pharmaceuticals AG; Alkermes, Inc.; Amarin Pharma Inc.; Aspect Medical Systems; AstraZeneca; Auspex Pharmaceuticals; Bayer AG; Best Practice Project Management, Inc.; BioMarin Pharmaceuticals, Inc.; Biovail Corporation; BrainCells Inc; Bristol-Myers Squibb; CeNeRx BioPharma; Cephalon, Inc.; CNS Response, Inc.; Compellis Pharmaceuticals; Cypress Pharmaceutical, Inc.; DiagnoSearch Life Sciences (P) Ltd.; Dinippon Sumitomo Pharma Co. Inc.; Dov Pharmaceuticals, Inc.; Edgemont Pharmaceuticals, Inc.; Elisai Inc.; Eli Lilly and Company; EnVivo Pharmaceuticals, Inc.; ePharmaSolutions; EPIX Pharmaceuticals, Inc.; Euthymics Bioscience, Inc.; Fabre-Kramer Pharmaceuticals, Inc.; Forest Pharmaceuticals, Inc.: GenOmind, LLC: GlaxoSmithKline: Grunenthal GmbH: i3 Innovus/Ingenis: Janssen Pharmaceutica; Jazz Pharmaceuticals, Inc.; Johnson & Johnson Pharmaceutical Research & Development, LLC; Knoll Pharmaceuticals Corp.; Labopharm Inc.; Lorex Pharmaceuticals; Lundbeck Inc.; MedAvante, Inc.; Merck & Co., Inc.; MSI Methylation Sciences, Inc.; Naurex, Inc.; Neuralstem, Inc.; Neuronetics, Inc.; NextWave Pharmaceuticals; Novartis AG; NuPathe; Nutrition 21; Orexigen Therapeutics, Inc.; Organon Pharmaceuticals; Otsuka Pharmaceuticals; Pamlab, LLC.; Pfizer Inc.; PharmaStar; Pharmavite® LLC.; PharmoRx Therapeutics; Precision Human Biolaboratory; Prexa Pharmaceuticals, Inc.; Puretech Ventures; PsychoGenics; Psylin Neurosciences, Inc.; Rexahn Pharmaceuticals, Inc.; Ridge Diagnostics, Inc.; Roche; Sanofi-Aventis US LLC.; Sepracor Inc.; Servier Laboratories; Schering-Plough Corporation; Solvay Pharmaceuticals, Inc.; Somaxon Pharmaceuticals, Inc.; Somerset Pharmaceuticals, Inc.; Sunovion Pharmaceuticals; Supernus Pharmaceuticals, Inc.; Synthelabo; Takeda Pharmaceutical Company Limited; Tal Medical, Inc.; Tetragenex Pharmaceuticals, Inc.; Teva; TransForm Pharmaceuticals, Inc.; Transcept Pharmaceuticals, Inc.; Vanda Pharmaceuticals, Inc.; Speaking/Publishing: Adamed, Co; Advanced Meeting Partners; American Psychiatric Association; American Society of Clinical Psychopharmacology; AstraZeneca; Belvoir Media Group; Boehringer Ingelheim GmbH; Bristol-Myers Squibb; Cephalon, Inc.; CME Institute/Physicians Postgraduate Press, Inc.; Eli Lilly and Company; Forest Pharmaceuticals, Inc.; GlaxoSmithKline; Imedex, LLC; MGH Psychiatry Academy/ Primedia; MGH Psychiatry Academy/Reed Elsevier; Novartis AG; Organon Pharmaceuticals; Pfizer Inc.; PharmaStar; United BioSource, Corp.; Wyeth-Ayerst Laboratories; Equity Holdings: Compellis; PsyBrain, Inc.; Royalty/patent, other income: Patent for Sequential Parallel Comparison Design (SPCD), which are licensed by MGH to RCT Logic, LLC; and patent application for a combination of azapirones and bupropion in Major Depressive Disorder (MDD).; Copyright for the MGH Cognitive & Physical Functioning Questionnaire (CPFQ), Sexual Functioning Inventory (SFI), Antidepressant Treatment Response Questionnaire (ATRQ), Discontinuation-Emergent Signs & Symptoms (DESS), and SAFER; Lippincott, Williams & Wilkins; Wolkers Kluwer; World Scientific Publishing Co. Pte.Ltd., Part 2: Belvoir Media Group for editing a newsletter: 2011-\$12,000., Part 4: Abbott Laboratories; Alkermes, Inc.; Aspect Medical Systems; AstraZeneca; BioResearch; BrainCells Inc.; Bristol-Myers Squibb; CeNeRx BioPharma; Cephalon, Inc.; Clintara, LLC; Covance; Covidien; Eli Lilly and Company; EnVivo Pharmaceuticals, Inc.; Euthymics Bioscience, Inc.; Forest Pharmaceuticals, Inc.; Ganeden Biotech, Inc.; GlaxoSmithKline; Icon Clinical Research; i3 Innovus/Ingenix; Johnson & Johnson Pharmaceutical Research & Development; Lichtwer Pharma GmbH; Lorex Pharmaceuticals; NARSAD; NCCAM; NIDA; NIMH; Novartis AG; Organon Pharmaceuticals; PamLab, LLC.; Pfizer Inc.; Pharmavite® LLC; Photothera; Roche; RCT Logic, LLC (formerly Clinical Trials Solutions, LLC); Sanofi-Aventis US LLC; Shire; Solvay Pharmaceuticals, Inc.; Synthelabo; Wyeth-Ayerst Laboratories

## **ACNP 2013 Presenter Disclosures (continued)**

Fleischhacker, W. Wolfgang : *Part 1:* Amgen, Lundbeck, Roche, Bristol-Myers Squibb, Otsuka, Janssen, MedAvante, Merck, Vanda, Endo, Takeda, Pfizer, Reckitt-Benckiser, *Part 3:* Janssen, Otsuka, Reckitt-Benckiser

Frail, Donald: *Part 1:* I am an employee of AstraZeneca. , *Part 2:* I am an employee of AstraZeneca. , *Part 3:* I am an employee of AstraZeneca and was an employee of Pfizer. , *Part 5:* I am an employee of AstraZeneca.

**Gainetdinov, Raul**: *Part 4:* I have research grants from F. Hoffmann La-Roche (Basel, Switzerland) on the topic of this presentation.

George, Tony: *Part 1:* Grant support from Pfizer, Speakers Bureau Pfizer, 2011 and 2012, Data Monitoring Committee, Novartis, 2011-present, *Part 4:* Pfizer - for studies of varenicline in smokers with mental illness, including schizophrenia

Geyer, Mark: *Part 1:* Consulting compensation from Abbott, Acadia, Addex, Cerca, Lundbeck, Merck, Neurocrine, Omeros, San Diego Instruments, Takeda, DART and Teva, *Part 2:* Equity interest in San Diego Instruments, *Part 3:* Equity interest in San Diego Instruments, *Part 4:* Research grant support from Intracellular Therapeutics, Johnson & Johnson, NIDA, NIMH, and the U.S. Veteran's Administration VISN 22 Mental Illness Research, Education, and Clinical Center.

Glatt, Stephen: Part 1: I serve as a scientific consultant to SynapDx Corp.

Gobbi, Gabriella: Part 1: Lilly, Astra-Zeneca, Lundbeck, Cosmas Therapeutics

**Goff, Donald**: *Part 1*: DSMB member: Otsuka Pharmaceuticals, *Part 4*: PamLab, Pfizer, Novartis, GSK

**Gold, Lisa**: *Part 1:* Full-time employee of Merck and Co, Inc, *Part 2:* Full-time employee of Merck and Co, Inc, *Part 3:* Full-time employee of Merck and Co, Inc, *Part 5:* Full-time employee of Merck and Co, Inc

**Gopal, Srihari**: *Part 2:* Shareholder Johnson & Johnson (JNJ), Shareholder Merck (MRK), *Part 5:* Full time employee Johnson & Johnson (JNJ)

Gordon, Joshua: Part 1: Speaker at Pfizer Basic Research Division Neuroscience Symposium, honorarium, 2011

**Gründer, Gerhard**: *Part 1*: Dr. Gründer has served as a consultant for Bristol-Myers Squibb (New York, NY), Cheplapharm (Greifswald, Germany), Eli Lilly (Indianapolis, Ind), Forest Laboratories (New York, NY, USA), Lundbeck (Copenhagen, Denmark), Otsuka (Rockville, Md.), Roche (Basel, Switzerland), and Servier (Paris, France). He has served on the speakers' bureau of Bristol-Myers Squibb, Eli Lilly, Gedeon Richter (Budapest, Hungary), Otsuka, Roche, and Servier. He has received grant support from Alkermes, Eli Lilly, and Roche. He is co-founder of Pharma-Image – Molecular Imaging Technologies GmbH, Düsseldorf. , *Part 2*: 2011: Eli Lilly, and Roche. He is co-founder of Pharma-Seli Lilly, and Roche. He is co-founder of Pharma-Image – Molecular Imaging Technologies GmbH, Düsseldorf. Technologies GmbH, Düsseldorf.

## **ACNP 2013 Presenter Disclosures (continued)**

**Grace, Anthony** : *Part 1*: Johnson & Johnson, Lundbeck, Pfizer, GSK, Puretech Ventures, Merck, Takeda, Dainippon Sumitomo, Otsuka, Lilly, Roche, Asubio, *Part 4*: Lilly, Lundbeck

**Graham, Danielle**: *Part 5*: I am a full time employee of EMD Serono Research & Development Institute.

Greenberg, Benjamin: *Part 1:* Meeting Travel Expense, Medtronic Inc, *Part 4:* Research Grant Support, Hoffman-LaRoche, Inc.

**Greist, John** : *Part 1:* eResearch Technology, Healthcare Technology Systems, Pfizer, *Part 2:* eResearch Technology, Healthcare Technology Systems, Possibly Pfizer, though I expect something less than \$10,000., If I own any stock in any pharmaceutical or device company it would be in retirement accounts such as the State of Wisconsin Retirement Plan over which I have no control. I have never purchased or held any pharmaceutical stocks in my personal investment account, nor has my spouse., *Part 3:* Please see #2 above., *Part 4:* AstraZeneca, eResearch Technology, Forest, Lilly, Novo Nordisk, Otsuka, Pfizer, Takeda, Transcept, UCB, *Part 5:*Healthcare Technology Systems is a medical assessment company. Though it is not technically a pharmaceutical/biotech/medical device company, I list it here to avoid any possible misunderstanding.

**Gur, Raquel**: *Part 4*: The study reported here was sponsored and funded by AstraZeneca Pharmaceuticals.

**Gur, Ruben**: *Part 4*: The study reported here was sponsored and funded by AstraZeneca Pharmaceuticals.

**Gurney, Mark**: *Part 1*: Dr. Gurney is an employee of Tetra Discovery Partners., *Part 2*: Dr. Gurney is an employee of Tetra Discovery Partners., *Part 3*: Dr. Gurney is an employee of Tetra Discovery Partners., *Part 5*: Tetra Discovery Partners.

Haber, Suzanne: *Part 1:* Dr. Haber has received consultation fees from Medtronic, Inc and Pfizer, Inc.

Harrison, Paul : *Part 1:* Advisory board, Sunovion (2013), Honorarium for educational talks, Otsuka (2013), *Part 2:* Employment: University of Oxford, Expert witness work for Pinsent Masons, London, Deputy Editor Honorarium, Biological Psychiatry, *Part 4:* Unrestricted educational grant from Takeda (Cambridge) 2012-13

Hasler, Gregor : *Part 1:* Servier (Suisse) SA, Lundbeck (Schweiz) AG, Schweizerische Gesellschaft für Bipolare Störungen, AstraZeneca, Eli Lilly (Suisse) SA, *Part 4:* Novartis Switzerland

Hen, Rene: Part 1: Serve on Scientific Advisory Boards for Roche Pharmaceuticals, Lundbeck and Servier

## **ACNP 2013 Presenter Disclosures (continued)**

Heres, Stephan: *Part 1:* I have received honoraria from Janssen-Cilag, Eli Lilly, Sanofi-Aventis and Johnson & Johnson. I have accepted travel or hospitality payment from Janssen-Cilag, Sanofi-Aventis, Johnson & Johnson, Pfizer, Bristol-Myers-Squibb, AstraZeneca, Lundbeck, Novartis and Eli Lilly. I have participated in clinical trials sponsored or supported by Eli Lilly, Janssen Cilag, Johnson & Johnson, Bristol-Myers-Squibb, AstraZeneca, Lundbeck, Novartis, Servier, Pierre Fabre, Pfizer, Organon, Roche and Merck. I have participated in advisory activities and boards for Janssen, Johnson & Johnson, Eli Lilly, Lundbeck and Roche.

**Hermanson, Daniel** : *Part 1*: DJH, LJM and SP have submitted a patent application entitled "Compositions and Methods for Substrate-Selective Inhibition of Endocannabinoid Oxygenation," which includes the compound LM-4131.

Hickie, Ian: Part 1: Servier, Astrazeneca, Pfizer, Janseen, Eli Lilly, Part 4: Servier Laboratories

**Higgs, Suzanne**: *Part 1*: Academic Supervisor of PhD studentship part funded by P1vital, *Part 4*: Academic Supervisor of PhD studentship part funded by P1vital

**Hollander, Eric**: *Part 1:* research grants: Simons Foundation, Roche, Transcept, Forest, Coronado Biosciences, consultant: Roche, Coronado Biosciences, *Part 4:* research grants: Simons Foundation, Roche, Transcept, Forest, Coronado Biosciences,

**Hyman, Steven**: *Part 1:* Member - Novartis Science Board, Consultant - AstraZeneca, iMed, Scientific Advisory Board - Fidelity Biosciences, *Part 2:* Novartis Science Board, Scientific Advisory Board - Fidelity Biosciences

**Innis, Robert**: *Part 1*: Eli Lilly has provide funds to NIMH to support my research, *Part 4*: Eli Lilly has provide funds to NIMH to support my research.

**Iosifescu, Dan**: *Part 1:* In the previous 36 months, Dr. Iosifescu has received research funding through Mount Sinai School of Medicine from AstraZeneca, Brainsway, Euthymics, Neosync, and Roche; he has received consulting fees from CNS Response, Otsuka, Servier and Sunovion., *Part 4:* In the previous 36 months, Dr. Iosifescu has received research funding through Mount Sinai School of Medicine from AstraZeneca, Brainsway, Euthymics, Neosync, and Roche; he has received consulting fees from CNS Response, Otsuka, Servier and Sunovion.

Jarskog, Lars: *Part 1*: Research grants from Sunovion, Amgen, Roche. DSMB board member - Janssen.

**Javitt, Daniel**: *Part 1:* Honoraria from Sunovion, BMS, Eli Lilly, Takeda, Omeros, Otsuka, Consensus Medical Communications, Guidepoint global, American Capital, Clearpoint communications, Vindico Medical Communication, and Clearview Healthcare. Research support from Pfizer and Roche; equity in, Glytech, Inc. and AASI; intellectual property rights for use of glycine, D-serine and glycine transport inhibitors in schizophrenia, and serves, on the advisory board of Promentis, *Part 2:* Columbia, NYS OMH, Glytech, *Part 4:* Pfizer, Roche

Kalin, Ned: Part 1: Honorariums: CME Outfitters, Elsevier, Letters & Sciences; Scientific Advisory Board, Corcept Therapeutics, Neuronetics, CeNeRx BioPharma, Neurocrine Biosciences, Neuronetics, LLC, Medivation, Janssen; Stockholder, Equity Options, Corcept Therapeutics, CeNeRx BioPharma; Owner, Promoter Neurosciences, LLC; Patents, Promoter sequences for corticotropin-releasing factor CRF2alpha and method of identifying agents that alter the activity of the promoter sequences: U.S. Patent issued on 07-04-06; patent #7071323, U.S. Patent issued on 05-12-09; patent #7,531,356, Promoter sequences for urocortin II and the use thereof: U.S. Patent issued on 08-08-06; patent #7087385, Promoter sequences for corticotropin-releasing factor binding protein and use thereof: U.S. Patent issued on 10-17-06; patent #7122650, Part 2: Elsevier, Part 4: Project Investigator: Neurobehavioral Bases of Emotion Regulation and Dysregulation in Adolescence. P50 MH84051, National Institute of Mental Health/National Institute of Health, \$234,757,00 2008-2013, Principal Investigator: Developmental Mechanisms Underlying the Risk to Develop Anxiety and Depression. Subproject on a Silvio O. Conte Center for Interdisciplinary Research on Brain, Behavior & Mental Health, National Institute of Mental Health, \$1,855,782, 2008-2013, Principal Investigator: Development and regulation of emotion in primates. R01 MH046729, National Institute of Mental Health/National Institute of Health, \$425,841.00, 2012-2017, Principal Investigator: Brain mechanisms underlying childhood anxiety. R21MH092581, National Institute of Mental Health/National Institute of Health, \$125,000, 2012-2014, Principal Investigator: Brain Mechanisms Mediating Genetic Risk Factors for Anxiety and Depression. DHHS, PHS R01 MH081884. National Institute of Mental Health, \$4,472,284, 2008-2012, Principal Investigator: Defining corticotropin-releasing factor (CRF) system changes in amygdala and medial temporal cortex in association with depression and suicide. The Stanley Medical Research Institute, \$150,000, 2009-2011, Principal Investigator: Development and Regulation of Emotion in Primates. R01 MH046729, National Institute of Mental Health, \$2,743,637, 2005-2011, Principle Investigator: Combining mouse and monkey models to understand human risk for psychopathology. MH091550. National Institute of Mental Health, \$275,000, 2010-2012, Co-Principal Investigator: Neural Substrates of Affective Style and Emotion Regulation. R01, MH043454, National Institute of Mental Health, \$150,000 Supplement, 2008-2011, APIRE/Janssen Resident Psychiatric Mentor Grant,

Kantrowitz, Joshua: *Part 1*: Dr. Kantrowitz reports having received consulting payments within the last 2 years from Otsuka Pharmaceuticals, Quadrant Health, RTI Health solutions, the Healthcare Advisory Board, Vindico Medical Education, Health Advances, LLC, Strategic Edge Communications. He owns a small number of shares of common stock in GlaxoSmithKline., *Part 2*: NYS OMH, Columbia, RFMH, St Luke's-Roosevelt, *Part 4*: He has conducted clinical research supported by the NIMH, the Stanley Foundation, Roche-Genetech, EnVivo, Psychogenics, Sunovion, Novartis, Pfizer, Lilly and GlaxoSmithKline.

Kenny, Paul : Part 1: Consultant to Pfizer, Inc., Co-founder of Eolas Therapeutics.

**Kinon, Bruce**: *Part 1*: Employee of Eli Lilly and Company, Shareholder of Eli Lilly and Company, *Part 2*: Employee of Eli Lilly and Company, Shareholder of Eli Lilly and Company, *Part 3*: Employee of Eli Lilly and Company, *Part 5*: Employee of Eli Lilly and Company

## **ACNP 2013 Presenter Disclosures (continued)**

Klann, Eric: *Part 1:* I am a consultant for Takeda Pharmaceuticals. My wife is employed by Takda Pharmaceuticals, *Part 2:* Consultant for Takeda Pharmaceuticals., *Part 3:* Consultant for Takeda Pharmaceuticals during sabbatical of 2012-13 academic year.

Knight, Scott: Part 5: Sigma Aldrich

**Kohler, Christian**: *Part 4*: The study reported here was sponsored and funded by AstraZeneca Pharmaceuticals.

Krystal, John: *Part 1:* Abbott, Amgen, AstraZeneca, BMS, Eisei, Estellas, Forest, Johnson and Johnson, Lilly, Lohocla, Mnemosyne, Naurex, Novartis, Pfizer, Shire, Sunovion, Takedam, Teva, *Part 4:* AstraZeneca, Pfizer

Lanz, Thomas: Part 1: Employee of Pfizer, Part 3: Employee of Pfizer, Part 5: Pfizer

Lapidus, Kyle: *Part 1*: Dr. Lapidus has received support for this project from the Brain and Behavior Research Foundation's Young Investigator Award and Apire Janssen Psychiatric Resident Research Scholars Award and serves as scientific advisor for Halo Neuro, Inc. He also participated in an interview on the future of antidepressants with LCN Consulting, Inc., *Part 4*: Dr. Lapidus has received support for this project from the Brain and Behavior Research Foundation's Young Investigator Award and Apire Janssen Psychiatric Resident Research Scholars Award and Apire Janssen Psychiatric Resident Research Scholars Award.

Lavretsky, Helen: *Part 1:* Rsearch grants from Forest Research Institute and Alzheimer's Research and Prevention foundation, *Part 4:* Forest Research Institute- research grants

**Lisanby, Sarah**: *Part 4*: Research Grants to my institution from Brainsway, NeoSync, ANS/St. Jude. Equipment support from Magstim, Magventures.

Malhotra, Anil: Part 1: Genomind

Mann, J. John: *Part 2:* Royalties from Research Foundation for Mental Health for C-SSRS, *Part 4:* Unrelated past grants from GSK and Novartis

**March, Mary**: *Part 4:* The study reported here was sponsored and funded by AstraZeneca Pharmaceuticals.

Markou, Athina: Part 4: Bristol-Myers-Squibb.

**Marnett, Lawrence**: *Part 1*: DJH, LJM and SP have submitted a patent application entitled "Compositions and Methods for Substrate-Selective Inhibition of Endocannabinoid Oxygenation," which includes the compound LM-4131.

**Marx, Christine**: *Part 1*: Applicant or co-applicant, pending patents on the use of neurosteroids and derivatives in CNS disorders and for lowering cholesterol (no patents issued, no licensing in place). Unpaid scientific advisor, Sage Therapeutics.

## **ACNP 2013 Presenter Disclosures (continued)**

Mathalon, Daniel: Part 1: Consultant for Amgen, Consultant for Bristol Myers Squibb

**Matsumoto, Mickey**: *Part 1:* I am an employee of Astellas Research Institute of America LLC, a subsidiary of Astellas Pharma Inc., *Part 2:* Astellas Research Institute of America LLC., *Part 3:* Astellas Research Institute of America LLC., *Part 5:* Astellas Research Institute of America LLC.

**McCracken, James**: *Part 1:* Research Contracts: Roche, Seaside Therapeutics, Otsuka, Consultant Income: Roche, BioMarin, PharmaNet, Speaker Honoraria: Tourette Syndrome Association, *Part 2:* Research Contracts: Roche, Seaside Therapeutics

McCullumsmith, Cheryl: Part 1: Jansenn Pharmaceuticals : Suicide Advisory Board

**McGorry, Patrick**: *Part 1*: I have received honoraria for educational and consultancy activities and/or travel support to attend such consultancy meetings from Janssen Cilag, Servier and Roche and unrestricted research grant support from Janssen Cilag and Astra Zeneca, *Part 4*: Unrestricted research grant support from Janssen Cilag and Astra Zeneca

**Meyer-Lindenberg, Andreas**: *Part 1:* Speaker and Advisory boards: AstraZeneca, J+J, Lundbeck, Servier, Lilly

Miller, Andrew: Part 1: Abbott Laboratories, AstraZeneca, Centocor Inc., GlaxoSmithKline, Lundbeck Research USA, F. Hoffmann-La Roche Ltd., Schering-Plough Research Institute and Wyeth/Pfizer Inc., Part 4: Centocor Inc., GlaxoSmithKline, and Schering-Plough Research Institute

Miyakawa, Tsuyoshi: Part 1: Advisor/Consultant for Astellas Pharma Inc.

**Murrough, James**: *Part 1*: Dr. Murrough has received research support from Janssen Pharmaceuticals and Avanir Pharmaceuticals, *Part 4*: Dr. Murrough has received research support from Janssen Pharmaceuticals and Avanir Pharmaceuticals.

Nemeroff, Charles: *Part 1:* Skyland Trail, Cenerx, Novadel Pharma, Takeda, Revaax Pharma, Xhale, Allergan, Lilly, Roche, Shire, SK Pharma, PharmaNeuroboost, *Part 2:* Cenerx, Novadel Pharma, PharmaNeuroboost, Xhale, *Part 3:* Xhale, PharmaNeuroboost, Cenerx, Novadel Pharma

Nuechterlein, Keith: Part 1: Investigator-Initiated Research Grant from Janssen Scientific Affairs, LLC, Research grant from Brain Plasticity, Inc., Consultant and research grant, Genentech, Consultant, Otsuka, Part 4: Investigator-Initiated Research Grant from Janssen Scientific Affairs, LLC, Research grant from Brain Plasticity, Inc., Research grant from Genentech

Nye, Jeffrey: *Part 1:* Employee of Janssen/J&J, *Part 2:* Employee of Janssen/J&J, *Part 3:* Employee of Janssen/J&J, *Part 4:* Employee of Janssen/J&J, *Part 5:* Employee of Janssen/J&J

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**O'Malley, Stephanie**: *Part 1:* ACTIVE workgroup supported be Eli Lilly, Lundbeck, Alkermes, Pfizer, Johnson and Johnson, GSK, Abbott, Hazelden Foundation, Applied behavior research, Pfizer, *Part 4:* Eli Lilly contract, Pfizer medication supplies

**Patel, Maxine**: *Part 1*: Consultancy: Janssen; Endo; Amgen; Lundbeck, Principal or Chief Investigator for clinical studies: Amgen; Lundbeck, *Part 4*: Principal or Chief Investigator for clinical studies: Amgen; Lundbeck,

**Patel, Sachin**: *Part 1*: DJH, LJM and SP have submitted a patent application entitled "Compositions and Methods for Substrate-Selective Inhibition of Endocannabinoid Oxygenation," which includes the compound LM-4131.

Paul, Steven: Part 1: Alnylam Pharmaceuticals (Board of Directors), Constellation Pharmaceuticals (Board of Directors), Eli Lilly (Stockholder), Karuna Pharmaceuticals (Board of Directors), Sage Therapeutics (Founder and shareholder), Sigma Aldrich Company (Board of Directors), Tal Medical Pharmaceuticals (Scientific Advisory Board and Board of Directors), Third Rock Ventures (Venture Partner), Part 2: Alnylam Pharmaceuticals (Board of Directors), Constellation Pharmaceuticals (Board of Directors), Eli Lilly (Stockholder), Karuna Pharmaceuticals (Board of Directors), Sage Therapeutics (Founder and shareholder), Sigma Aldrich Company (Board of Directors), Tal Medical Pharmaceuticals (Scientific Advisory Board and Board of Directors), Third Rock Ventures (Venture Partner), Part 3: Alnylam Pharmaceuticals (Board of Directors), Eli Lilly (Stockholder), Sage Therapeutics (Founder and shareholder), Sigma Aldrich Company (Board of Directors), Third Rock Ventures (Venture Partner), Part 4: Alzheimer's Drug Discovery Foundation, AstraZeneca Pharmaceuticals, Johnson & Johnson Pharmaceuticals, Alzheimer's Drug Discovery Foundation, NIH R01 grant.

Pearlson, Godfrey: Part 1: Consultant BMS 2012

**Pelham, William**: *Part 1*: Gave a talk at a conference in Japan that was sponsored by Janssen Pharmaceuticals

Perkins, Diana: Part 1: Consultant: Genentech/Roche, Sunovion, Otsuka

**Peterchev, Angel**: *Part 1*: Dr. Peterchev is inventor on patents and patent applications on TMS technology assigned to Columbia University and Duke University, including technology licensed to Rogue Research; was Principal Investigator on a research grant to Duke from Rogue Research and equipment donations to Columbia and Duke by Magstim, MagVenture, and ANS/St. Jude Medical; has received patent royalties from Rogue Research through Columbia for TMS technology; and has received travel support from Rogue Research through Duke, *Part 3*: Dr. Peterchev has received patent royalties from Rogue Research through Columbia University for TMS technology that he invented, *Part 4*: Dr. Peterchev was Principal Investigator on a research grant to Duke from Rogue Research and of equipment donations to Columbia and Duke by Magstim and MagVenture.

**Phillips, Mary**: *Part 2*: I have been a consultant for Cardiff University, Department of Psychological Medicine, UK. This relationship is due to end in 2013.

Pletcher, Mathew: Part 1: Employee of Pfizer, Part 5: Pfizer

## **ACNP 2013 Presenter Disclosures (continued)**

**Posner, Kelly**: *Part 1*: Dr. Posner is the director of the Center for Suicide Risk Assessment. The Center, as part of an effort to help execute the FDA suicidality classification mandates, has received support from the following pharmaceutical companies: Abbott, Aerial Biopharma, Albany Molecular Research, Alder Biopharma, Alfresa, Alkermes, Amgen, Astellas Pharm, Astra Zeneca, Biogen, Biomarin Pharmaceutical, Biovail Technologies, Boehringer Ingelheim, Bracket, Bristol Myers Squibb, Cato Research, Celerion, Cephalon, Cetero Research, Chiesi Pharmaceuticals, Covance, CRI Worldwide, Daiichi Sankyo Company, Depomed, Douglas Pharmaceuticals/VersaPharm, EISAI, Elan, EnVivo, Epiomed, Forest, Gilead, GlaxoSmithKline, Grunenthal, GW Pharma Limited, Human Genome Sciences, i3 International, i3 Research, i3 Pharmaceutical Services, ICON Development Solutions, Impax Laboratories, INC Research, Ingenix, IntelGenx Corp, IntraCellular Therapies, Ironwood, IRIS, Isis, Ivax, Janssen, Jazz, Johnson & Johnson, Lilly USA, Lotus, Lundbeck, MedAvante, MedImmune, Merck, Mochida, Neurocrine Biosciences, Neuronex, Neurosearch, NextWave Pharma, Novartis, Noven, NovoNordisk, Omeros, Orexigen Therapeutics, Orion, Otsuka, Pamlab, Parexel, Pfizer, PGx Health, Pharmaceutical Research Associates, Pharmanet i3, Pierrel Research, PPD, Prana Biotechnology, ProPhase, Psyadon, QED Pharmaceuticals, Quintiles, Receptos, Reckitt Benckiser, Rho, Rhythm, Roche, Sanofi-Aventis, Schering-Plough, Schwarz Biosciences, SCOPE International, Sepracor, Shionogi, Shire, Siena Biotech, SK Life Science, Sunovion, Supernus Pharmaceuticals, Synosia Therapeutics, Takeda Global Research & Development Center, Takeda Pharmaceuticals, TauRx Therapeutics, Theravance, UCB Biosciences, UCB Korea, UCB Pharma, United BioSource Corp, Upsher-Smith Laboratories, Vaccinex, Valeant Pharmaceuticals, Vernalis, Vivus, WorldWide Clinical Trials, Wyeth Ayerst, Wyeth Pharmaceuticals, Wyeth Research, Xenoport and Zalicus. Dr. Posner receives royalty payments from the e-CSSRS, which are distributed to her by her employer, the Research Foundation for Mental Hygiene.

Potenza, Marc: *Part 1:* Consulting to Boehringer-Ingelheim and Lundbeck; financial interests in Somaxon. Grant from Psyadon, *Part 4:* Grant from Psyadon.

**Rauch, Scott**: *Part 1*: 2011 NIMH RDoC - honorarium, 2011 & 2012 - Oxford University Press - royalty, 2011 & 2012 - APPA - royalty, *Part 2*: None other than my primary employer: McLean Hospital/Partners Healthcare, *Part 3*: None other than my primary employer: McLean Hospital/Partners Healthcare, *Part 4*: Cyberonics, Medtronic

Ray, Lara: Part 1: I am a paid consultant for GSK.

**Reynolds, Charles**: *Part 2*: I receive an honorarium from the American Association for Geriatric Psychiatry, for service as associate editor of the American Journal of Geriatric Psychiatry, *Part 4*: During the past three years I have received pharmaceutical supplies for my NIH sponsored clinical trials from Pfizer (venlafaxine, aripiprazole, donepezil) and Eli Lilly (duloxetine), *Part 5*: not applicable; primary employer = University of Pittsburgh and UPMC

**Risbrough, Victoria**: *Part 4*: Research grants awarded from Johnson and Johnson, Omeros Pharmaceuticals, Sunovion Pharmaceuticals, and Johnson and Johnson.

## **ACNP 2013 Presenter Disclosures (continued)**

**Robbins, Trevor**: *Part 1:* Consultancy: Cambridge Cognition, Lilly, Merck, GlaxoSmithKline, Lundbeck, Teva, Shire Pharmaceuticals, ChemPartners, Royalties: Cambridge Cognition (CANTAB), Editorial Honoraria: Springer Verlag (Psychopharmacology), *Part 2:* Cambridge Cognition, *Part 3:* Cambridge Cognition, *Part 4:* Lilly, Lundbeck, GlaxoSmithKline

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**Ruparel, Kosha**: *Part 4*: The study reported here was sponsored and funded by AstraZeneca Pharmaceuticals.

**Russo, Scott**: *Part 4*: Dr Russo receives laboratory support from Johnson and Johnson to study IL-6 and depression.

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**Satterthwaite, Theodore**: *Part 4*: The study reported here was sponsored and funded by AstraZeneca Pharmaceuticals.

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Schadt, Eric: *Part 1*: SAB for Pacific Biosciences and SAB for Berg, *Part 2*: SAB for Pacific Biosciences and SAB for Berg.

**Schork, Nicholas**: *Part 1*: Dr. Schork is a founder of CypherGenomics (http://www.cyphergenomics.com/) and on the board of MD Revolution (http://mdrevolution.com/) and has stock as a result.

## **ACNP 2013 Presenter Disclosures (continued)**

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**Swanson, James**: *Part 1*: I have been a consultant with Noven Pharmaceuticals and BLK Pharma, and I recieved indirect support from pharmaceutucal companies from a professional organzation, the European Network of Hyperkinetic Disorders (EUNETHYDIS), to make presentations at annual meetings.

Sweeney, John: Part 1: Consultant to Lilly, Takeda, Roche and BMS

## **ACNP 2013 Presenter Disclosures (continued)**

**Swift, Robert**: *Part 1:* Consultant to Transcept Pharmaceuticals - received fee, Scientific Advisory Board for D&A Pharma - received honorarium and travel expenses to meeting, Consultant to Pharmaceutico CT - received fee and travel expense reimbursement, *Part 2:* Consultant to Pharmaceutico CT - fee and travel expense reimbursement

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**Turetsky, Bruce**: *Part 4:* The study reported here was sponsored and funded by AstraZeneca Pharmaceuticals.

Uchida, Hiroyuki: *Part 1*: Dr. Uchida has received speaker's honoraria from Otsuka Pharmaceutical, Janssen Pharmaceutical, Novartis Pharma, Eli Lilly, Shionogi, GlaxoSmithKline, Yoshitomi Yakuhin, Dainippon-Sumitomo Pharma, and Janssen Pharmaceutical within the past two years. *Part 4*: Dr. Uchida has received grants from Pfizer, Astellas Pharmaceutical, Eisai, Otsuka Pharmaceutical, GlaxoSmithKline, Shionogi, and Dainippon-Sumitomo Pharma, Eli Lilly, Mochida Pharmaceutical, Meiji-Seika Pharma, Janssen Pharmaceutical, and Yoshitomi Yakuhin within the past two years.

**Uher, Rudolf**: *Part 1*: Dr Uher co-chairs a steering board of a research project initiated and funded by Bristol Myers Squibb and collaborates with Pfizer, Glaxo-Smith Kline and Roche as part of the European Union Innovative Medicine Initiative funded NEWMEDS project. Dr Uher has received no personal income from any pharmaceutical or biotech industry and holds no equity in companies active in medicine, pharmaceuticals or biotechnology.

Van Zeeland, Ashley: *Part 1:* Dr. Van Zeeland is a Co-founder and CEO of CypherGenomics (http://www.cyphergenomics.com/) and has stock as a result, *Part 5:* Cypher Genomics

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Wahlestedt, Claes: Part 1: Pfizer, OPKO

**Walker, Brendan**: *Part 4*: Completed a cooperative research agreement on 9/24/2011 with H. Lundbeck A/S, Copenhagen on the kappa-opoid mechanisms of nalmefene.

## **ACNP 2013 Presenter Disclosures (continued)**

**Warden, Melissa**: *Part 1*: Stanford University has filed for patent protection on technology invented by Dr. Melissa R. Warden and Dr. Karl Deisseroth.

**Waxmonsky, James**: *Part 1*: Research Contract Noven Pharmaceuticals, Research Contract Janssen, Research Contract Shire Inc., Advisory Board Noven Pharmaceuticals (<\$5,000), *Part 4*: Research Contract Noven Pharmaceuticals, Research Contract Janssen, Research Contract Shire Inc.

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**Wolf, Daniel**: *Part 4*: The study reported here was sponsored and funded by AstraZeneca Pharmaceuticals.

**Wolf, Marina**: *Part 1*: I have 50,000 shares (~\$50,000) in a non-publicly traded entity: Grace Laboratories LLC, 1755 Logans Knoll NE, Atlanta GA 30329. I do not receive any income at this time. There is no linkage to my research or to ACNP., I have 50,000 shares (~\$50,000) in a non-publicly traded entity: CIS Blotech Inc, 2701 North Decatur Rd, Decatur, GA 30033. I do not receive any income at this time. There is no linkage to my research or to ACNP., *Part 2*: I have 50,000 shares (~\$50,000) in a non-publicly traded entity: Grace Laboratories LLC, 1755 Logans Knoll NE, Atlanta GA 30329. I do not receive any income at this time. There is no linkage to my research or to ACNP., *Part 2*: I have 50,000 shares (~\$50,000) in a non-publicly traded entity: Grace Laboratories LLC, 1755 Logans Knoll NE, Atlanta GA 30329. I do not receive any income at this time. There is no linkage to my research or to ACNP., I have 50,000 shares (~\$50,000) in a non-publicly traded entity: CIS Blotech Inc, 2701 North Decatur Rd, Decatur, GA 30033. I do not receive any income at this time. There is no linkage to my research or to ACNP.

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Woods, Scott: Part 1: Investigator-initiated grant from Pfizer, Part 4: Investigator-initiated grant from Pfizer

Yates III, John R.: Part 1: ThermoFisher, Part 2: ThermoFisher, Part 4: Roche

**Youngstrom, Eric**: *Part 1*: Eric Youngstrom has consulted with Lundbeck and received past travel support from Bristol-Myers Squibb. He has consulted with Penn State about analyses for a grant funded by Pfizer, and received grant funding from NIMH.

**Zarate, Carlos**: *Part 1*: Dr. Zarate is listed as a co-inventor on a patent application for the use of ketamine and its metabolites in major depression. Dr. Zarate has assigned his rights in the patent to the U.S. government but will share a percentage of any royalties that may be received by the government.

## Zhang, H. Steve: Part 5: Sangamo Biosciences

Zorumski, Charles: Part 1: I serve on the Scientific Advisory Board of Sage Therapeutics.

**Zukin, Stephen**: *Part 5:* Former employee of AstraZeneca Pharmaceuticals in Wilmington DE, the company that funded/sponsored the study reported here., Current employee of Forest Research Institute in Jersey City, New Jersey.

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