International Behavioral
Neuroscience Society

Annual Meeting
Program and Abstracts

Steamboat Springs, Colorado, USA
May 24-29, 2011

Abstracts of the International Behavioral Neuroscience Society, Volume 20, May 2011

TABLE OF CONTENTS

IBNS CENTRAL OFFICE

Marianne Van Wagner, Executive Coordinator

International Behavioral Neuroscience Society
8181 Tezel Road #10269
San Antonio, Texas 78250 USA

(830) 796-9393 tel.
(830) 796-9394 fax
(866) 377-4416 (toll-free from within the US)
ibns@ibnshomepage.org
http://www.ibnshomepage.org

PRESIDENTIAL WELCOME

Dear Conference Participants, Colleagues, and Friends,

It is my pleasure to welcome you to the commemorative 20th annual meeting of the International Behavioral Neuroscience Society. What a wonderful two decades of behavioral neuroscience! This year's meeting in tranquil Steamboat Springs offers one of the most innovative programs in the history of the Society. I'm thrilled that three of our discipline's most accomplished scientists have agreed to be our Keynote Speakers this year: Janice Kiecolt-Glaser will inform us about the toxicity of stress on immune functions; Kerry Ressler will address the regulation of fear in mice and men; and Stephen Suomi will examine the gene-environment interplay in risk and resilience in primates. To celebrate our 20th Anniversary Meeting, three of our esteemed past-presidents have agreed to bring us up to date in their respective research areas. Paul Sanberg (IBNS President 1993) will speak about the importance of translational neuroscience, Bob Blanchard (IBNS President 2003) will remind us how important it is to bring nature into our sometimes too sterile laboratories and Sue Carter (IBNS President 2004) will address a topic—the healing power of love—that is near and dear to all of our hearts (or make that near and dear to our oxytocin neurocircuits!).

As exciting behavioral neuroscience findings continue to be reported in traditional scientific journals, it is becoming increasingly important to report this information in responsible and informative ways to broader audiences. A special session entitled *Science and the Media* will address this public relations challenge. Three accomplished science journalists have generously agreed to step away from their busy careers to speak at this year's meeting: Sandra Blakeslee, author and frequent contributor to the *New York Times* will inform us about why scientists and journalists are sometimes perceived to be from different planets; Mariette DiChristina, Editor-in-Chief of *Scientific American* and *Scientific American Mind* will discuss the critical role of scientists in public outreach; and Paul Raeburn, author, journalism professor and frequent guest-host of NPR's Talk of the Nation, will update us on the emerging role of science blogs.

As the infomercials often tell us---THERE'S MORE! Don Stein has agreed to be the first speaker in the new Professional Journeys series, a series proposed to remind our young scientists that resilience is key to a successful career in science. I won't attempt to paraphrase Don's title—it's a classic: *Progesterone and Brain Injury: From Bench to Bench to Bench to Bench to Bench to Bedside*. Yes, even the most accomplished scientists in our discipline have had to recover from multiple frustrations and disappointments, but the end-result can be worth all the heartache!

In typical IBNS fashion, the members have proposed several fascinating sessions on topics ranging from pheromones to autism; additionally, there are workshops, several student events, oral sessions, and two exciting poster sessions. A special thanks is extended to the Program Committee Wim Crusio, Chair, Stephen Kent, Co-Chair, K.-P. Ossenkopp, Nancy Ostrowski, Leonie de Visser, John Bruno, Byron Jones, Mikhail Pletnikov and Hee-Sup Shin for developing such a stellar program. The Local Organizing Committee Robert Spencer, Chair, Jodi Lukkes, and Karen Stevens has once again been helpful informing us about local resources. We also appreciate our valued corporate sponsor Elsevier for their continued support. Finally, our most heartfelt thanks are extended to IBNS Executive Coordinator Marianne Van Wagner who, year after year, choreographs interesting meetings in engaging settings.

Enjoy the conference!

Kelly Lambert
President, International Behavioral Neuroscience Society

OFFICERS

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We are pleased to announce the recipients of the IBNS Travel Awards for the 2011 meeting in Steamboat Springs, Colorado, USA. These awards will be presented at the Awards Banquet on Saturday evening. Award winners will receive a cash award, certificate, and waiver of registration and banquet fees. Congratulations to all.

TRAVEL AWARDS

(listed alphabetically)

Presentations given by the Travel Awardees are indicated in the program by the symbol †.

Postdoctoral Travel Awards

Dr. Laurence Daniele Coutellier, NIMH, National Institute of Health, Bethesda MD USA
Dr. Eimeira Padilla, University of Texas at Austin, Austin, TX USA
Dr. Lissandra C. Baldan Ramsey, Yale University, New Haven, CT USA

Graduate Student Travel Awards

Ms. Amanda A. Braun, University of Cincinnati, Cincinnati, OH USA
Mr. Andrew Robert Burke, University of South Dakota, Vermillion, SD, USA
Mr. Richard Chu, Drexel University College of Medicine, Philadelphia, PA, USA
Mrs. Amy E. Clipperton Allen, University of Guelph, Guelph, Ontario, Canada
Ms. Geetha Kannan, Johns Hopkins University, Baltimore, MD USA

Undergraduate Travel Awards

Ms. Manoela Viar Fogaça, School of Medicine of Ribeirão Preto, Ribeirão Preto, São Paulo, Brazil
Mr. Fernando Midea Cuccovia Vasconcelos Reis, Univ. of Sao Paulo, Ribeirão Preto, São Paulo, Brazil
Ms. Kerisa Lea Shelton, Northern Arizona University, Flagstaff, AZ USA
Ms. Kalpana Subedi, Morgan State University, Baltimore, MD USA

Travel awardees are presenting orally in the Travel Award Blitz and will also have their research presented in a poster session.

The IBNS would like to express our gratitude to the following organizations that have given financial support to the 20th International Behavioral Neuroscience Society Conference. This financial support enabled many students to attend the conference and also allowed recruitment of excellent speakers.

SPONSORS

National Institute of Mental Health

Grant Number: 2R13MH065244-06

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**These companies will be onsite during the meeting. Please take time to stop by and visit the booths located in the Meeting Foyer.*

The Society would like to extend our deep appreciation to the following committees that are responsible for the success of this meeting:

Program Committee

John P Bruno, The Ohio State University, Columbus, OH, USA
Wim E. Crusio, Centre National De La Recherche Scientifique, Talence, France (**Chair**)
Leonie De Visser, Rudolf Magnus Institute of Neuroscience, Utrecht, The Netherlands
Byron C. Jones, Pennsylvania State University, University Park, PA, USA
Stephen Kent, La Trobe University, Bundoora, Victoria, Australia (**Co-Chair**)
Klaus-Peter Ossenkopp, University of Western Ontario, London, Ontario, Canada
Nancy L. Ostrowski, Eli Lilly & Company, Indianapolis, IN, USA
Mikhail Pletnikov, John Hopkins Univ. Sch. of Medicine, Baltimore, MD, USA
Hee-Sup Shin, Korea Institute of Science and Technology, Seoul, Republic of Korea

Education and Training Committee

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Anna Phan, University of Guelph, Guelph, Ontario, Canada
Katerina V. Savelieva, External Pharma, Inc., The Woodlands, TX USA
Peter Shiromani, Harvard Medical School, West Roxbury, MA, USA
Matthew Skelton, Cincinnati Children's Hospital Medical Center, Cincinnati, OH, USA

Local Organizing Committee

Robert L. Spencer, University of Colorado, Boulder, CO USA (**Chair**)
Jodi Lukkes, University of Boulder, Boulder, CO, USA
Karen Stevens, University of Colorado, Sch. of Medicine, Auroa, CO, USA

Any IBNS member who would like to become more involved in the Society may volunteer to serve on an IBNS committee. Committee details can be found on our website at http://www.ibnshomepage.org/committees.htm.

KEYNOTE SPEAKERS

Janice Kiecolt-Glaser, Ohio State University
How stress kills: Perspectives from psychoneuroimmunology

Kerry Ressler, Emory University
Examining fear and its regulation in mice and men

Stephen Suomi, NIH/NICHD
Risk, resilience, and gene-environment interplay in primates

PRESIDENTIAL ADDRESS

Kelly Lambert, Randolph-Macon College
The dynamic parental brain: More than a mom and pop operation

MEDIA AND SCIENCE SESSION

Sandra Blakeslee, Science Writer/Author, *New York Times*
Scientists are from Mars; Journalists are from Venus

Mariette DiChristina, Editor-in-Chief, *Scientific American*
The scientist's role in public outreach

Paul Raeburn, Journalism Professor/Author,
frequent guest host of NPR's Talk of the Nation
Open science: How the rise of science blogs is changing the conduct of research

PROFESSIONAL JOURNEYS SERIES

Donald G. Stein, Emory University School of Medicine
Progesterone and brain injury: From bench to bench to bench to bench to bench to bedside

PAST PRESIDENTS' SYMPOSIUM

Paul R. Sanberg, University of South Florida, IBNS President 1993
Translating laboratory discovery to the clinic: From antidepressant to stem cell therapy

Robert Blanchard, University of Hawaii, IBNS President 2003
Bringing natural behaviors into the laboratory: Novel models of social behavior in the mouse

C. Sue Carter, University of Illinois at Chicago, IBNS President 2004
The healing power of love: An oxytocin hypothesis

SPECIAL SYMPOSIA

Sex, fear and pheromones: biology of semiochemicals in rodents.
Co-Chairs: **Ajai Vyas and Iain McGregor**

Orexin/hypocretin's role in mobilizing adaptive and pathological panic and anxiety responses.
Co-Chairs: **Anantha Shekhar and Philip L. Johnson**

Examining the genetic and neural components of cognitive flexibility using mice.
Co-Chairs: **Jared W. Young and Jonathan L. Brigman**

The use of animal models to understand mechanisms underlying environmental impact on brain development.
Co-Chairs: **F. Scott Hall and Susan L. Andersen**

Autism-relevant behaviors of mouse models of ASD.
Chairperson: **Robert J. Blanchard**

Organizer: **Corina Bondi**, IBNS Student Representative to Council

Wednesday, May 25

2:00-3:00 **Workshop:** *Grants (Rainbow Room)*

This workshop will focus on funding opportunities from government and industry (e.g., collaborations and consulting), the current outlook for research grants, types of grants available from different funding institutions, and strategies to ensure successful applications.

Panel participants:

- Christine Hohmann (Morgan State Univ)
- F. Scott Hall (NIDA)
- Nancy Ostrowski (Eli Lilly)

8:00-9:30 PM **Student/Postdoc Social.** *(Villas Gallery)*

Come meet and interact with your peers from across the world--a sure pathway to future friendships and collaborations. Food and drinks will be provided.

Friday, May 27

2:00-3:00 **Workshop:** *Science Jobs on the Global Market (Rainbow Room)*

A diverse, international panel will share their knowledge and impressions of current funding issues and grant and career opportunities in their areas. This portion of the workshop will be especially relevant to those applying for postdocs and other jobs around the world.

Panel participants:

- Anders Agmo (Norway)
- C. Sue Carter (USA)
- Wim Crusio (France)
- Martin Kavaliers (Canada)
- Abbe MacBeth (Noldus)

Saturday, May 28

1:15-3:15 **Meet the Professionals** – Student/Postdoc event *(Skyline/Sunset Room)*

It is not often that so many successful professionals from diverse institutions are all in one place and willing to share their knowledge and experience in an informal setting! Similar events at IBNS meetings in the past have been extremely well-received, and this event will certainly be an experience to remember. Friendly faculty members will participate in a "negotiating exercise" with students and postdocs, helping to enhance their skills in asking the right questions and focusing on the most appropriate issues when applying for jobs in the current competitive job market. Lunch will be provided for participants.

NOTE: During each of these professional development events, new and kindly donated science-relevant and professional development books will be given away via raffle drawings. Although all participants will be winners by enhancing their knowledge by the end of these events, some will also win a highly useful book!

IBNS 2011 - CALL FOR SYMPOSIA and SATELLITE PROPOSALS

The Program Committee is now soliciting proposals for symposia and satellites for the 2012 Annual Meeting of the International Behavioral Neuroscience Society.

A typical symposium includes four (4) speakers and is scheduled for two (2) hours. The time and date of symposia are set by the Program Committee. Satellites are structured and financed by the organizers. Satellite meetings and may be held either prior to or after the IBNS meeting dates.

All proposals should include a title, the name of the chairperson(s), a substantive description of the topic and proposed talks, the list of speakers, their affiliations, and tentative titles of their talks. Satellite proposals should also include the anticipated location and plans for financing.

All proposals are reviewed by the Program Committee and then submitted to the IBNS Council for consideration.

The deadline for priority consideration of symposium proposals is September 1, 2011. Please send your proposal to the Program Committee Chair, Dr. Stephen Kent at *s.kent@latrobe.edu.au* and COPY the IBNS Central Office at *ibns@ibnshomepage.org*. Please use subject line: Symposia/Satellite Proposal 2012.

PROGRAM NOTES:

- Presenting authors are indicated in the program by **bold** type.
- † Indicates Travel Award recipient.
- Unless otherwise indicated in program, events will be held in the Storm Peak/Mt. Werner Ballroom.

Tuesday, May 24, 2011

9:00-12:00 **Council Meeting.** *(Aspen Boardroom)*

4:00-6:30 **Registration**. *(Registration Booth, next to Rainbow Room)* For late arrivals only, the registration desk will be open on Wednesday morning at 8:00 a.m.

7:00-8:30 **Cocktail Reception.** *(Pool Tent)*

Wednesday, May 25, 2011

7:30-8:30 **Continental Breakfast.** *(Twilight Room)*

8:15-8:30 **Welcome**: IBNS President, **Kelly Lambert**. *(Storm Peak/Mt. Werner)*

8:30-10:45 **Symposium 1:** SEX, FEAR AND PHEROMONES: BIOLOGY OF SEMIO-CHEMICALS IN RODENTS. **Co-Chairs: Ajai Vyas and Iain McGregor.**

8:30 WHEN A RAT SMELLS A CAT: BEHAVIORAL AND NEURAL RESPONSES TO PREDATOR ODORS IN RODENTS. **McGregor, I.S.**; Bowen, M.T.; Kevin, R.; May, M.; Kendig, M.; Hunt, G.E.

9:00 SPECIALIZED ODORS THAT INITIATE INNATE DEFENSIVE BEHAVIOR IN THE MOUSE. **Stowers, L.**

9:30 ODORS PARASITES AND MATE RESPONSES. **Kavaliers, M.**; Choleris, E.

10:00 SEX, FEAR AND PARASITES: PARASITIC MANIPULATION OF SEMIOCHEMICALS IN BROWN RAT. **Vyas, A.**

10:30 Discussant: **Caroline Blanchard**

10:45-11:15 **Break & Exhibit Viewing** *(Meeting Foyer)*

11:15-12:15 **Presidential Lecture: Kelly Lambert**, Randolph-Macon College
The Dynamic Parental Brain: More than a Mom and Pop Operation.

12:15-2:00 **Break**

2:00-3:00 **Workshop:** *Grants (Rainbow Room)*

3:00-5:15 **Symposium 2:** OREXIN/HYPOCRETIN'S ROLE IN MOBILIZING ADAPTIVE AND PATHOLOGICAL PANIC AND ANXIETY RESPONSES. **Co-Chairs: Anantha Shekhar and Philip L. Johnson**

3:00 ROLE OF OREXIN IN MOBILIZING ADAPTIVE BEHAVIORAL AND PHYSIOLOGICAL RESPONSES. **Shekhar, A.**; Truitt, W.; Samuels, B.; Fitz, S.D.; Lowry, C.A.; Johnson, P.L.

3:30 EVIDENCE THAT OREXIN IS A CRITICAL SUBSTRATE UNDERLYING PATHOLOGICAL ANXIETY AND PANIC RESPONSES IN RATS AND HUMANS. **Johnson, P.L.**; Truitt, W.; Fitz, S.D.; Minick, P., Dietrich, A.; Sanghani, S.; Trskman-Bendz, L.; Goddard, A.W.; Brundin, L.; Shekhar, A.

4:00 NEURAL CIRCUITS AND MECHANISMS INVOLVED IN OREXIN A-INDUCED ANXIETY-LIKE BEHAVIOR. **Truitt, W.A.**; Johnson, P.L.; Molosh, A.; Lungwitz, E.; Harvey, B.; Dietrich, A.D.; Minick, P.E.; Shekhar, A.

4:30 THE ROLE OF OREXIN AND ITS RECEPTORS IN BEHAVIORAL AND PHYSIOLOGIC RESPONSES EVOKED BY AMPHETAMINES. **Rusyniak, D.**

5:00 Discussant: **Adrian Dunn**

5:30-8:00 **Past Presidents' Symposium**

5:30 Introduction: **Kelly Lambert**

6:00 BRINGING NATURAL BEHAVIORS INTO THE LAB: MODELING SOCIAL AND DEFENSIVE BEHAVIORS IN RODENTS. **Blanchard, R.J.**

6:40 THE HEALING POWER OF LOVE: AN OXYTOCIN HYPOTHESIS. **Carter, S.**

7:20 TRANSLATING LABORATORY DISCOVERY TO THE CLINIC: FROM ANTIDEPRESSANT TO STEM CELL THERAPY. **Sanberg, P.R.**

8:00-9:30 **Student/Postdoc Social.** *(Villas Gallery)*
(Note: This function is for students/postdocs only.)

Thursday, May 26, 2011

7:30-8:30 **Continental Breakfast.** *(Twilight Room)*

8:30-10:45 **Symposium 3**: EXAMINING THE GENETIC AND NEURAL COMPONENTS OF COGNITIVE FLEXIBILITY USING MICE. Co-Chairs: **Jared W. Young and Jonathan L. Brigman**

8:30 BALANCING FLEXIBILTY AND EFFICIENT ACTION: CORTICOSTRIATAL NETWORKS IN THE MOUSE. **Brigman, J.L.**; Wright; T., Davis; M.I.; Saksida; L.M., Bussey; T.J., Jiang; Z., Lovinger; D.M.; Nakazawa, K; Holmes, A;

9:00 SYSTEMS ANALYSIS OF CEREBELLAR MODULATION OF EXECUTIVE FUNCTION. Dickson, P.E.; Martin L.A.; Rogers, T.D.; Blaha, C.D.; Goldowitz, D.; **Mittleman, G.**

9:30 FAST SPIKING INTERNEURONS ASSIST CORTICAL TUNING. **Powell, E.M.**; Xu, J.; Bissonette, G.B.;

10:00 DELAYING THE EUREKA MOMENT BY REMOVING THE ALPHA 7 NICOTINIC ACETYLCHOLINE RECEPTOR. **Young, J.W.**; Meves, J.M.; Tarantino, I.S.; Caldwell, S.; Geyer, M.A.

10:30 Discussant: **Joram Feldon**

10:45-11:15 **Break & Exhibit Viewing** *(Meeting Foyer)*

11:15-12:15 **Keynote Speaker: Janice Kiecolt-Glaser**, Ohio State University
How Stress Kills: Perspectives from psychoneuroimmunology

12:15-2:00 **Break**

2:00-4:00 **Media and Science Session**

2:00 **Sandra Blakeslee**, Science Writer/Author, *New York Times*
Scientists are from Mars; Journalists are from Venus

2:40 **Mariette DiChristina**, Editor-in-Chief, *Scientific American*
The Scientist's Role in Public Outreach

3:20 **Paul Raeburn**, Journalism Professor/Author,
frequent guest host of NPR's Talk of the Nation
Open Science: How the rise of science blogs is changing the conduct of research

4:00-5:30 **Oral Session 1:** Chairperson: **Nancy Ostrowski**

4:00 UNUSUAL IMMUNE REGULATION IN THE BTBR T + tf/J MOUSE: POSSIBLE MECHANISM FOR THE AUTISTIC LIKE PHENOTYPE OBSERVED IN THIS STRAIN. **Benno, R.**; Schanz, N.; Pettit, L.; Vassiliou, E.

4:15 LEUKEMIA IN MICE INDUCES STRESS-LIKE BEHAVIORAL, NEUROENDOCRINE AND NEUROCHEMICAL CHANGES. **Dunn, A.J.**; Newman, R.A.; Swiergiel, A.H.

4:30 AUTOPHAGY ENHANCEMENT: A POSSIBLE NEW DRUG TARGET FOR AFFECTIVE DISORDERS. **Einat, H.;** Anderson, G.W.

4:45 ANTIDEPRESSANT-LIKE ACTIONS OF DIETARY CHOLINE SUPPLEMENTATION IN A RAT MODEL. **Glenn, M.J.**; Adams, R.; Saporta, A.N.; Cameron, S.; Gillies, S.; McClurg, L.M.

5:00 XAMOTEROL RESCUES MEMORY DEFICIT IN MOUSE MODEL OF DOWN SYNDROME BY ACTIVATION OF BETA-1 ADRENERGIC RECEPTOR. Faizi, M.; Mobley, W.C.; Coutellier, L.; **Shamloo, M.**

5:15 DOMINANCE AND SUBMISSIVENESS IN THE ETIOLOGY AND TREATMENT OF AFFECTIVE DISORDERS. **Pinhasov, A.**

6:30-8:30 **Poster Session 1:** *(Sunshine Peak)*

Anxiety and Stress

1. THE EFFECT OF ANXIOGENICS ON PREFRONTAL CORTICAL SINGLE UNIT ACTIVITY, LOCAL FIELD POTENTIAL OSCILLATIONS AND BEHAVIORAL FLEXIBILITY IN FREELY-MOVING RATS. **Bondi, C.O.**; Burkowski, A.J.; Del Arco, A.; Wood, J.; Moghaddam, B.

2. THE ANXIOLYTIC EFFECTS OF CANNABIDIOL IN A POST-TRAUMATIC STRESS MODEL ARE MEDIATED BY 5HT1A RECEPTORS. **Campos, A.C.**; Ferreira, F.R.; Guimaraes, F.S.

3. HIGH INTENSITY ACUTE STRESS INDUCES UNIQUE PATTERNS OF ASSOCIATIVE AND NON-ASSOCIATIVE FEAR MEMORY BEHAVIOR IN AN ANIMAL MODEL OF POSTTTRAUMATIC STRESS DISORDER. **Corley, M.**; Takahashi, L.

4. INTRA-AMYGDALA INFUSION OF A GROUP I AGONIST MODULATES PASSIVE AND ACTIVE DEFENSIVE BEHAVIORS IN A SEX SPECIFIC MANNER. **De Jesus-Burgos, M.**; Cruz-Santana, Y.; and Perez-Acevedo, N.L.

5. EFFECTS OF AN OREXIN 1 RECEPTOR ANTAGONIST ON INVERSE BENZODIAZEPINE AGONIST-INDUCED PANICOGENIC-RELATED RESPONSES AND CELLULAR RESPONSES IN THE BRAIN**. Federici, L.**; Fitz, S.D; Hammes, N.; Early, M.; Dietrich, A.; Lowry, C.A.; Samuels, B.C.; Shekhar, A.; Johnson, P.L.

6. THE PROTECTIVE EFFECTS OF EXERCISE ON CHRONIC STRESS-INDUCED NEUROTOXICITY. **Gerecke, K.M.;** Kolobova, A.; Allen, S.

7. TOXOPLASMA INDUCED OVERLAP IN DEFENSIVE AND REPRODUCTIVE NETWORKS IN THE MEDIAL AMYGDALA. Hari Dass, S.;**Vyas, A.**

8. THE PARADOX EFFECT OF NOMIFENSINE ON ACTIVE AVOIDANCE LEARNING IN AN ANIMAL MODEL OF BEHAVIORAL INHIBITION. **Jiao, X.**; Pang, K.; Beck, K.; Servatius, R.

9. OREXIN-A INJECTIONS INTO THE BNST INDUCES ANXIETY-LIKE BEHAVIOR VIA INTERACTIONS WITH GLUTAMATERGIC RECEPTORS IN THE RAT. **Lungwitz, E.**; Johnson, P.; Harvey, B.; Deal, R.; Dietrich, A.; Minick, P.; Shekhar, A.; Truitt, W.

10. AN INBRED MOUSE MODEL OF IMPAIRED FEAR EXTINCTION: CORTICO-AMYGDALA DENDRITIC DYSMORPHOLOGY. EFFECTS OF MEMORY REACTIVATION. **Martin, K.**; Lederle, L.; Holmes, A.

11. EVIDENCE FOR A LACK OF PHASIC INHIBITORY PROPERTIES OF HABITUATED STRESSORS ON HPA AXIS RESPONSES IN RATS. **Masini, C.V.**; Day, H.E.W.; Gray, T.; Crema, L.M.; Nyhuis, T.J.; Babb, J.A.; Campeau, S.

12. THE COMBINED EFFECTS OF STRESS AND ENRICHMENT ON POST-PARTUM BEHAVIOUR IN THE RAT. **Pichette, N.**; Falicki, A.; Mileva, G.; Rees, S.; Bielajew, C.

13. EFFECT OF STRESSOR CHALLENGE DURATION ON THE BEHAVIORAL AND NEUROENDOCRINE EXPRESSION OF STRESS RESPONSE HABITUATION. **Ramsey, R.E.**; Spencer, R.L.

14. IMPACT OF UNPREDICTABLE CHRONIC SOCIAL DEFEAT ON PALATABLE FEEDING AND BEHAVIOURAL MARKERS OF DEPRESSION AND ANXIETY. **MacKay, J.C.**; Patterson, Z.R.; James, J.; Kent, P.; Abizaid, A.; Merali, Z.

15. THE IMPACT OF JUVENILE STRESS ON PALATABLE FEEDING AND BEHAVIOURAL MARKERS OF DEPRESSION AND ANXIETY IN JUVENILIY. **MacKay, J.C.**; James, J.; Cayer, C.; Kent, P.; Merali, Z.

16. ANXIOGENIC-LIKE EFFECT OF CANNABIDIOL INJECTED INTO DE RAT PRELIMBIC PREFRONTAL CORTEX. †**Fogaca, M.V.**; Campos, A.C.; Guimaraes, F.S.

17. INCREASED LONG-TERM FEAR MEMORY IN MICE LACKING TIP39 SIGNALING: A NEW MODEL FOR THE STUDY OF FEAR-RELATED PSYCHOPATHOLOGY? †**Coutellier, L.**; Usdin, T.B.

18. NOVELTY-EVOKED ACTIVITY IN THE OPEN FIELD PREDICTS SUSCEPTIBILITY TO HELPLESS BEHAVIOR. †**Padilla, E.**; Shumake, J.; Barrett, D.; Holmes, G.; Sheridan, E.; Auchter, A.; Rothardt, A.; Gonzalez-Lima, F.

19. CORTICOSTERONE MEDIATES RISK ASSESSMENT BEHAVIORS IN THE ANTERIOR CINGULATE CORTEX OF RATS. †**Reis, F.M.C.V.**; Albrechet-Souza, L.; Franci, C. R.; Brandao, M. L.

20. ABUSE LIABILITY ASSESSMENT OF CINNAMOMUM CASSIA. †**Shelton, S.**; Pope, S.; Birkett, M.

21. EFFECTS OF CINNAMON ON PHYSIOLOGICAL RESPONSE TO A COGNITIVE EMOTIONAL STRESSOR. **Tebbe, D.**; Shelstad, T.; Gilbert, A.; Birkett, M.

22. SUBJECTIVE EFFECTS OF CINNAMON ON RESPONSE TO A LABORATORY STRESSOR. **Kontnik, M.**; Marcus, D.; Birkett, M.

23. EXPERIMENTAL EVIDENCE THAT DEACTIVATION OF SECURITY-MOTIVATION AFTER EXPOSURE TO POTENTIAL THREAT IS DYSFUNCTIONAL IN OBSESSIVE-COMPULSIVE DISORDER (OCD). **Hinds, A.**; Woody, E.; Schmidt, L.; Van Ameringen, M.; Szechtman, H.

24. COMPULSIVE CHECKING BEHAVIOR IN AN ANIMAL MODEL OF OBSESSIVE COMPULSIVE DISORDER. **Thompson, B.S.**; Greene-Collozi, E.; Andersen, S.L.

25. THE NEUROBIOLOGY OF BENZODIAZEPINE ABUSE AND THE ROLE OF SOCIAL DEFEAT. **Doss, L.**; van der Kooy, D

Development

26. LOW DIETARY PHYTOESTROGEN ELICITS ANXIOGENIC BEHAVIOR IN RAT PUPS MODERATED BY DIETARY PERIOD AND MATERNAL INFLUENCE. **Sanstrum, B.J.**; Totton, R.R.; Becker, L.A.

27. PRE- AND POST-NATAL ENVIRONMENTAL ENRICHMENT HAS IMMEDIATE BUT NOT LASTING CONSEQUENCES ON OFFSPRING SOCIAL BEHAVIOUR**. Mileva, G.**; Pichette, N.; Sparling, J.; Baker, S.; Bielajew, C.

28. NEONATAL STRESS ALTERS ULTRASONIC VOCALIZATIONS IN BALB/CBYJ MOUSE PUPS. **Miller, O.**; Akintola, T.; Hodges, A.; Hohmann, C.F.

29. BEHAVIOURAL AND NEURAL CHARACTERISTICS OF ACUTE AND CHRONIC MEPHEDRONE (4-METHYLMETHCATHINONE, MEOW) TREATMENT IN ADOLESCENT RATS. **Motbey, C.**; Hunt, G.E.; Bowen, M.; Artiss, S.; McGregor, I.S.

30. INCREASED IMMOBILITY OF NEONATALLY STRESSED MALE Balb/CbyJ MICE IN A MODIFIED FORCED SWIM TEST A POTENTIAL MODEL OF DEPRESSION? **Naidu, L.**; Miller, O.; Hohmann, C.

31. ALTERATIONS IN THE HIPPOCAMPAL EPIGENOME AS A RESULT OF PERINATAL EXPOSURE TO ETHANOL IN THE RAT. **Perkins, A.**; Lehmann, C.; Lawrence, R.; Kelly, S.

32. SELECTIVE ROLE OF NEUROPEPTIDE Y RECEPTOR SUBTYPE Y2 IN THE ANABOLIC STEROID INDUCED SEXUAL BEHAVIOR. **Ramos-Pratts, K.**; Huertas, A.; Parrilla, J.; Roig-Lpez, J.; Barreto-Estrada, J.

33. ENVIRONMENTAL ENRICHMENT ATTENUATES DEVELOPMENTAL LEAD EXPOSURE-INDUCED DEFICITS ON EGOCENTRIC AND ALLOCENTRIC SPATIAL NAVIGATION IN RATS. Goodwill, H.S.; McLean, M.C.; Wheeler, A.P.; **Schroeder, J.A.**

34. DEVELOPMENTAL EXPOSURE TO CHRONIC STRESS AND MANGANESE IN RATS, BUT NOT LOW LEVELS OF LEAD, AFFECTS ANXIETY RESPONSES AND EGOCENTRIC LEARNING. **Williams, M.T.**; Graham, D.L.; Amos-Kroohs, R.M.; Braun, A.A.; Grace, C.E.; Schaefer, T.L.; Skelton, M.R.; Vorhees, C.V.

35. EFFECTS OF NEONATAL STRESS ON SOCIAL BEHAVIOR AND CORTICAL AND HIPPOCAMPAL BDNF LEVELS IN Balb/CByJ MICE. †**Subedi, K.**; Naidu, L.; Azhagiri, A.; Pardo, C.A.; Koban, M.; Hohmann, C.F.

36. BEHAVIORAL ANALYSIS OF HDC-KO MICE IN THE BEHAVIORS RELEVANT FOR TOURETTE SYNDROME. †**Baldan Ramsey, L.C.**; Ohtsu, H.; de Araujo, I.; Pittenger, C.

Friday, May 27, 2011

7:30-8:30 **Continental Breakfast.** *(Twilight Room)*

8:30-10:45 **Symposium 4:** AUTISM-RELEVANT BEHAVIORS OF MOUSE MODELS OF ASD. Chairperson: **Robert J. Blanchard**

8:30 AUTISTIC FEATURES AND THEIR POSSIBLE TREATMENT IN THE FMR1-KNOCK OUT MOUSE. Pietropaolo, S.; **Crusio, W.E.**

9:00 ENHANCED SOCIABILITY, HYPER-DEFENSIVENESS AND DECREASED COCAINE-INDUCED BEHAVIORAL REACTIVITY IN MALE MICE WITH MECP2-308 MUTATION. **Pearson, B.L.**; Meyza, K.Z.; Defensor, E.B.; Pobbe, R.L.H.; Bolivar, V.J.; Blanchard, D.C.; Blanchard, R.J.

9:30 SOCIAL AND ENVIRONMENTAL FACTORS RELEVANT TO THE DEVELOPMENT OF SOCIABILITY IN INBRED MICE. **Yang, M.**; Crawley, J.N.

10:00 BTBR MICE SHOW AUTISM-LIKE BEHAVIOR CHANGES ON ETHO-LOGICALLY-RELEVANT TASKS RELATED TO SOCIALITY. **Blanchard, C.**

10:30 Discussant: **Iain S. McGregor**

10:45-11:15 **Break & Exhibit Viewing** *(Meeting Foyer)*

11:15-12:15 **Keynote Speaker: Kerry Ressler**, Emory University
Examining fear and its regulation in mice and men

12:15-2:00 **Break**

2:00-3:00 **Workshop:** *Science Jobs (Rainbow Room)*

3:00-5:30 **Travel Award Slide Blitz.** Chairperson: **Anders Agmo** *(Storm Peak/Mt. Werner)*

3:05 INCREASED LONG-TERM FEAR MEMORY IN MICE LACKING TIP39 SIGNALING: A NEW MODEL FOR THE STUDY OF FEAR-RELATED PSYCHOPATHOLOGY? †**Coutellier, L.**; Usdin, T.B.

3:16 NOVELTY-EVOKED ACTIVITY IN THE OPEN FIELD PREDICTS SUSCEPTIBILITY TO HELPLESS BEHAVIOR. †**Padilla, E.**; Shumake, J.; Barrett, D.; Holmes, G.; Sheridan, E.; Auchter, A.; Rothardt, A.; Gonzalez-Lima, F.

3:27 BEHAVIORAL ANALYSIS OF HDC-KO MICE IN THE BEHAVIORS RELEVANT FOR TOURETTE SYNDROME. †**Baldan Ramsey, L.C.**; Ohtsu, H.; de Araujo, I.; Pittenger, C.

3:38 DORSAL STRIATAL DOPAMINE DEPLETION IMPAIRS BOTH ALLOCENTRIC AND EGOCENTRIC NAVIGATION. †**Braun, A.A.**; Graham, D.L.; Schaefer, T.L.; Vorhees, C.V.; Williams, M.T.

3:49 ADULT MEDIAL PREFRONTAL CORTEX AND NUCLEUS ACCUMBENS AMPHETAMINE-INDUCED DOPAMINE RELEASE FOLLOWING ADOLESCENT SOCIAL DEFEAT. †**Burke, A**.; Forster, G.; Novick, A.; Roberts, C.; Watt, M.

4:00 IMPACT OF METHYLPHENIDATE ON A RODENT MODEL OF SUSTAINED ATTENTION AND LOCOMOTION: DIFFERENTIAL EFFECTS ON HIGH VERSUS LOW PERFORMERS. †**Chu, R**.; Nicholson, S.; Shumsky, J.S.; Waterhouse, B.D.

4:11 EFFECTS OF CHRONIC ERβ AGONIST DPN ON A SOCIALLY TRANSMITTED FOOD PREFERENCE IN OVARIECTOMIZED CD1 MICE. †**Clipperton Allen, A.E.**; Mikloska, K.V.; Roussel, V.R.; Ying, H.L.; Choleris, E.

4:22 TOXOPLASMA GONDII MOUSE MODEL OF SCHIZOPHRENIA-LIKE NEUROBEHAVIORAL ABNORMALITIES IN MICE: GENDER-RELATED EFFECTS AND MOLECULAR CORRELATES. †**Kannan, G.**; Xiao, J-C; Krasnova, I.N.; Cadet, J.; Yolken, R.; Jones-Brando, L.; Pletnikov, M.V.

4:33 ANXIOGENIC-LIKE EFFECT OF CANNABIDIOL INJECTED INTO DE RAT PRELIMBIC PREFRONTAL CORTEX. †**Fogaca, M.V.**; Campos, A.C.; Guimaraes, F.S.

4:44 CORTICOSTERONE MEDIATES RISK ASSESSMENT BEHAVIORS IN THE ANTERIOR CINGULATE CORTEX OF RATS. †**Reis, F.M.C.V.**; Albrechet-Souza, L.; Franci, C. R.; Brandao, M. L.

4:55 ABUSE LIABILITY ASSESSMENT OF CINNAMOMUM CASSIA. †**Shelton, S.**; Pope, S.; Birkett, M.

5:06 EFFECTS OF NEONATAL STRESS ON SOCIAL BEHAVIOR AND CORTICAL AND HIPPOCAMPAL BDNF LEVELS IN Balb/CByJ MICE. †**Subedi, K.**; Naidu, L.; Azhagiri, A.; Pardo, C.A.; Koban, M.; Hohmann, C.F.

6:30-8:30 **Poster Session 2:** *(Sunshine Peak)*

Cognition

37. ANDROSTENEDIONE IS ASSOCIATED WITH SPATIAL REFERENCE AND WORKING MEMORY IMPAIRMENT IN TRANSITIONAL AND SURGICALLY MENOPAUSAL MIDDLE-AGED RATS. **Acosta, J.I.**; Mennenga, S.M.; Camp, B.W.; Gerson, J.E.; Villa, S.R.; Bimonte-Nelson, H.A.

38. POTENTIAL ENHANCEMENT IN THE TRANSFER OF SYMBOLIC LEARNING IN RAT MOTHERS COMPARED TO VIRGIN FEMALES. **Bilinski, T.**; Au, A.; Meyer, E.; Kinsley, C.H.

39. ENDOGENEOUS HUNGER SUBSTANCE OREXIN A MODULATES SPATIAL PLASTICITY. **Oomura, Y.**; Aou, S.; Fukunaga, K.; Sasaki, K.

40. RAPID EFFECTS OF INTRAHIPPOCAMPAL DELIVERY OF 17β-ESTRADIOL ON OBJECT PLACEMENT LEARNING IN FEMALE MICE. Phan, A.; Molinaro, L.P.; MacLusky, N.J.; **Choleris, E.**

41. POTENTIAL EVIDENCE FOR PROSPECTIVE MEMORY IN PAROUS RATS. Franssen, R.A.; **Rafferty K.A.**; McDaniel, E.M.; Byce S.J.; Kinsley C.H.

42. MEDIAL SEPTAL GABAERGIC CONTROL OF HIPPOCAMPAL ACETYLCHOLINE RELEASE AND SHORT-TERM MEMORY. **Roland, J.J.**; Janke, K.L.; Savage, L.M.; Servatius, R.J.; Pang, K.C.H.

43. WORKING MEMORY IMPAIRMENTS WITH PROLONGED HIGH ALTITUDE RESIDENCE: AN FMRI STUDY. **Yan, X.**; Zhang, J.; Gong, Q.; Weng, X.

44. C-FOS EXPRESSION IN THE PERIAQUEDUCTAL GRAY VARIES RELATIVE TO THE METHOD OF CONDITIONED TASTE AVERSION EXTINCTION EMPLOYED. **Mickley, G.A.**; Wilson, G.N.; Remus, J.; Ramos, L.; Ketchesin, K.; Biesan, O.; Luchsinger, J.; Prodan, S.

45. AN L-TYPE CA++ CHANNEL BLOCKER ENHANCES THE ACTION OF DONEPEZIL IN OBJECT RECOGNITION MEMORY IN RATS. **Rose, G.M.**; Trippodi-Murphy, C.

46. IMPACT OF METHYLPHENIDATE ON A RODENT MODEL OF SUSTAINED ATTENTION AND LOCOMOTION: DIFFERENTIAL EFFECTS ON HIGH VERSUS LOW PERFORMERS. †**Chu, R**.; Nicholson, S.; Shumsky, J.S.; Waterhouse, B.D.

47. EFFECTS OF CHRONIC INTERMITTENT ETHANOL EXPOSURE ON CORTICO-STRIATAL-MEDIATED DISCRIMINATION AND REVERSAL LEARNING. **DeBrouse, L**.; Plitt, A.; Hurd, B.; Saksida, L.; Bussey, T.; Camp, M.; Holmes, A.

48. DORSAL STRIATAL DOPAMINE DEPLETION IMPAIRS BOTH ALLOCENTRIC AND EGOCENTRIC NAVIGATION. †**Braun, A.A.**; Graham, D.L.; Schaefer, T.L.; Vorhees, C.V.; Williams, M.T.

Reward and Addiction

49. ANTAGONISM OF CARBACHOL-INDUCED 22 kHz VOCALIZATION BY AMPHETAMINE IN THE RAT HYPOTHALAMIC-PREOPTIC AREA. Silkstone, M.; **Brudzynski, S.M.**

50. THE EFFECT OF PRENATAL METHAMPHETAMINE EXPOSURE ON DRUG-SEEKING BEHAVIOR OF ADULT MALE RATS. **Slamberov, R.**; Schutov, B.; Hrub, L.; Pometlov, M.

51. THE PUTATIVE ROLE OF THE NUCLEUS INCERTUS IN FEEDING BEHAVIOUR OF RATS. **Rajkumar, R.**; Suri, S.; Lee, L.C.; Dawe, G.S.

52. ADULT MEDIAL PREFRONTAL CORTEX AND NUCLEUS ACCUMBENS AMPHETAMINE-INDUCED DOPAMINE RELEASE FOLLOWING ADOLESCENT SOCIAL DEFEAT. †**Burke, A**.; Forster, G.; Novick, A.; Roberts, C.; Watt, M.

Social Behavior

53. HIPPOCAMPAL GENE EXPRESSION DURING ESCAPE FROM SOCIAL AGGRESSION IN HAMSTERS. **Arendt, D.H.**; Smith, J.P.; Bastida, C.C.; Prasad, M.; Rasmussen, T.L.; Summers, T.R.; Delville, Y.; Summers, C.H.

54. INTRA-VTA PERTUSSIS TOXIN INFUSIONS STIMULATE MATERNAL BEHAVIOR IN ADULT, NULLIPAROUS FEMALE RATS. **Bridges, R.S.**; Schoen, M.K.; Carini, L.M.; Gleason, E.D.; Lovelock, D.F.; Byrnes, E.M.; Byrnes, J.J.

55. SOCIAL DECISION MAKING DRIVES BEHAVIORAL AND NEURAL PLASTICITY DURING LEARNED ESCAPE. **Rasmussen,T.L.**; Summers, T.R.; Carpenter, R.E.; Smith, J.P.; Arendt, D.H.; Summers, C.H.

56. ANXIETY IS ALLEVIATED BY ESCAPE FROM SOCIAL AGGRESSION IN HAMSTERS. **Smith, J.P.**; Arendt, D.H.; Bastida, C.C.; Rasmussen, T.L.; Summers, T.R.; Delville, Y.; Summers, C.H.

57. A NOVEL ANIMAL MODEL FOR STUDIES IN AGGRESSION AND PATERNAL STRESS. **Ten Eyck, G.R.**

58. PAIR BONDING IN THE BLACK-PENCILLED MARMOSET (CALLITHRIX PENICILLATA): BEHAVIORAL CHARACTERISTICS. **Birnie, A.K.**; Smith, A.S.; French, J.A.; Agmo, A.

59. EFFECTS OF CHRONIC ERβ AGONIST DPN ON A SOCIALLY TRANSMITTED FOOD PREFERENCE IN OVARIECTOMIZED CD1 MICE. †**Clipperton Allen, A.E.**; Mikloska, K.V.; Roussel, V.R.; Ying, H.L.; Choleris, E.

60. NEONATALLY SEROTONIN DEPLETED MICE SHOW EARLY DEFICITS IN SOCIAL BEHAVIOR. **Ayorinde, M.;** Blue, M.E.; Hohmann, C.F.

61. IDENTIFYING THE ROLE OF SEROTONIN IN AUTISM-LIKE BEHAVIOR IN JUVENILE MICE. **Lewter, L.**; Hohmann, C.F.; Blue, M.E.

62. NEONATAL BLOCKADE OF GASTRIN RELEASING PEPTIDE RECEPTORS AS AN ANIMAL MODEL OF AUTISM. Johnstone, J.; **Mackay, J.C.**; Du, L.; Kent, P; Merali, Z.

63. ATYPICAL ULTRASONIC VOCALIZATIONS IN A MOUSE MODEL OF DOWN SYNDROME. **Pearson, J.**; Fernandez, F.; Costa, A.

Locomotion and Exploration

64. THE 5-HT1A RECEPTOR CONTRIBUTES SUBSTANTIALLY TO THE EFFECTS OF INDOLEALKYLAMINE HALLUCINOGENS ON LOCOMOTOR ACTIVITY AND INVESTIGATORY BEHAVIOR IN MICE. **Halberstadt, A.L.**; Geyer, M.A.

65. TOXOPLASMA GONDII MOUSE MODEL OF SCHIZOPHRENIA-LIKE NEUROBEHAVIORAL ABNORMALITIES IN MICE: GENDER-RELATED EFFECTS AND MOLECULAR CORRELATES. †**Kannan, G.**; Xiao, J-C; Krasnova, I.N.; Cadet, J.; Yolken, R.; Jones-Brando, L.; Pletnikov, M.V.

Saturday, May 28, 2011

7:30-8:30 **Continental Breakfast.** *(Twilight Room)*

8:30-10:45 **Symposium 5:** THE USE OF ANIMAL MODELS TO UNDERSTAND MECHANISMS UNDERLYING ENVIRONMENTAL IMPACT ON BRAIN DEVELOPMENT. Co-Chairs: **F. Scott Hall and Susan L. Andersen**

8:30 THE EFFECTS OF ISOLATION-REARING AND AMITRIPTYLINE ON GENE EXPRESSION IN THE HIPPOCAMPUS: CAN GENE EXPRESSION STUDIES HELP REVEAL THE UNDERLYING MECHANISMS OF GENE-ENVIRONMENT INTERACTIONS? **Hall, F.S.**; Cole, S.W.; Andrews, A.M; Knutson, B.

9:00 RILUZOLE AND FLUOXETINE MODULATE THE EFFECTS OF MATERNAL SEPARATION ON DEPRESSIVE BEHAVIOR IN A SEX-DEPENDENT MANNER. **Andersen, S.L.**; Vaccaro, K.; Thompson, B.S.; Freund, N.

9:30 THE EFFECTS OF POST-WEANING SOCIAL ISOLATION ON SEROTONERGIC SYSTEMS AND BEHAVIOR. **Lukkes, J.L.**; Lowry, C.A.

10:00 SYNERGISTIC INTERACTIONS BETWEEN MILD PRENATAL IMMUNE CHALLENGE AND PERI-PUBERTAL STRESS IN THE DISRUPTION OF ADULT BEHAVIORAL FUNCTIONS RELEVANT TO SCHIZOPHRENIA. **Feldon, J.**; Giovanoli, S.; Meyer, U.

10:30 Discussant: **Stephen J. Suomi**

10:45-11:15 **Break & Exhibit Viewing** *(Meeting Foyer)*

11:15-12:15 ***Professional Journeys Series*** **Donald G. Stein**, Emory University School of Medicine. *Progesterone and Brain Injury: From Bench to Bench to Bench to Bench to Bench to Bedside*

12:15-1:15 **IBNS Business Meeting –** ALL IBNS members are invited to attend.

1:15-3:15 **Meet the Professionals** – Student/Postdoc event *(Skyline/Sunset Room)*

3:30-5:00 **Oral Session 2:** Chairperson: **Wim Crusio** *(Twilight Room)*

3:30 SKYSCRAPERS AND HAYLOFTS: AN EXPLORATION OF DIFFERENTIAL HOUSING IN LONG-EVANS RATS. Franssen, C.L.; Kaufman, C.; **Bardi, M.**; Lambert, K.G.

3:45 ANTERIOR OLFACTORY NUCLEUS SUPPRESSES IPSILATERAL AMYGDALA IN SOCIAL BUFFERING OF CONDITIONED FEAR RESPONSES. **Kiyokawa, Y.**; Takeuchi, Y.; Mori, Y.

4:00 NITRIC OXIDE PRODUCING NEURONS IN THE DORSAL RAPHE NUCLEUS ARE ACTIVATED BY RESTRAINT STRESS IN THE WAKING RAT. **Vasudeva, R**. K.; Waterhouse, B. D.

4:15 CHRONIC STRESS MODULATES MICROGLIAL-NEURONAL INTERACTIONS IN PREFRONTAL CORTEX: IMPLICATIONS FOR DEVELOPMENT OF DEPRESSION. Walker, F.R.; Tynan, R.; Ng, A.; Nalivaiko, E.; **Day, T.A.**

4:30 ALARM PHEROMONE SUPPRESSES SEXUAL BEHAVIOR IN MALE RATS. **Kobayashi, T.**; Kiyokawa, Y.; Takeuchi, Y.; Mori, Y.

6:00-7:00 Keynote Speaker: **Stephen Suomi**, NIH/NICHD *(Storm Peak/Mt. Werner)*
Risk, resilience, and gene-environment interplay in primates

7:00-7:30 **Cash Bar**

7:30-11:00 **Banquet**. Awards, buffet, dancing.

Wednesday, May 25, 2011

8:30-10:45 **Symposium 1:** SEX, FEAR AND PHEROMONES: BIOLOGY OF SEMIO-CHEMICALS IN RODENTS. **Co-Chairs: Ajai Vyas and Iain McGregor.**

WHEN A RAT SMELLS A CAT: BEHAVIORAL AND NEURAL RESPONSES TO PREDATOR ODORS IN RODENTS. McGregor, I.S.; Bowen, M.T.; Kevin, R.; May, M.; Kendig, M.; Hunt, G.E. School of Psychology, University of Sydney, NSW 2006, Australia. Prey species show persistent defensive behavior when exposed to odors from sympatric predators. For more than a decade our laboratory has examined the effect of cat fur-related odors on the behavior of laboratory rats. This work shows that cat fur odor is most likely a kairomone, a chemical or a mixture of chemicals produced by a predator that increases the fitness of the prey at the expense of the predator. A variety of cat-related stimuli (collars, cloths rubbed on a cat, cat fur, fur extracts) are effective in provoking defensive responses in rats, and also in conditioning prolonged residual fear to associated cues and environments. Fos immunohistochemistry studies show fur-related stimuli activate accessory olfactory (pheromone-processing) brain regions in rats as well as a complex network that includes the medial amygdala, hypothalamic defensive zone, periaqueductal grey and cuneiform nucleus. Rats exposed to cat fur while in a group show pronounced huddling behavior and such group exposure to predatory threat elicits different patterns of brain activation than is seen with rats exposed to cat fur when alone. Some rats in a fur-exposed group are far more courageous in the face of predatory threat and ongoing research is focusing on the characteristics of these commando rats. We have also shown that repeated fur exposure to adolescent rats can actually promote a more robust and resilient behavioural repertoire in later adulthood, showing that living under early predatory threat may have some developmental advantages. The exact chemical constituent(s) in cat fur that give rise to defensive behaviors in rats is not clear at present, with our recent work suggesting that it is a non-volatile semiochemical that may vary subtly across individual cats. Overall, work with predatory odors in the laboratory can be very informative and rewarding for the researcher, and can provide naturalistic models that are of great utility in pharmacological, neurobiological and behavioral investigations. Supported by an Australian Research Council grant to ISM

SPECIALIZED ODORS THAT INITIATE INNATE DEFENSIVE BEHAVIOR IN THE MOUSE. Stowers, L. Department of Cell Biology. The Scripps Research Institute. La Jolla, CA 92037 USA. We are studying how subsets of neurons specify behavior. Pheromone ligands activate dedicated subsets of neurons to generate instinctive behavior which provides a powerful experimental system to study neural function. Our approach is unique in that we are quantifying the mouses natural behavior in order to identify the underlying sensory mechanisms. As biochemical assays are used to map and elucidate metabolic pathways, we use innate behavior as a functional assay to identify the corresponding ligand cues and cognate neurons that generate behavior. We are directing our experiments to identify underlying neural mechanisms that encode innate fear behavior. We have found that mice display innate fear-like behavior to diverse predator odors. Biochemical analysis of predator odors revealed that Mup proteins purified from cats and rats are sufficient to evoke this behavior. In the mouse, vomeronasal sensory neurons are necessary for both detection and response to these cues. Comparison of predator Mups and mouse Mup pheromones shows that they activate different subsets of neurons. These experiments provide insight to the organization of the neural code that imitates innate fear.

ODORS, PARASITES AND MATE RESPONSES. Martin Kavaliers1 and Elena Choleris2, 1Department of Psychology University of Western Ontario, London, Canada, 2Department of Psychology University of Guelph, Guelph Canada. Social behavior entails the processing of social information and recognition of individuals. Social recognition allows the establishment of group hierarchies and mediates the development of appropriate social preferences in relation to adaptive mate choices. The neural-hormonal systems implicated in the processing of olfactory linked social recognition include, the neuropetide oxytocin (OT) as well as estrogenic (ER) mechanisms. Male and female mice with deletion for the gene OT [OT knockout (OTKO)] or estrogen receptors alpha and beta(ERKO alpha or beta) are impaired in social recognition and memory, with ER alpha and E beta being differentially involved. A major cost of social behavior is the increased risk of exposure to parasites and infection. Animals utilize social information, including volatile and involatile chemical signals, to recognize and avoid infected conspecifics. Female and male mice distinguish between infected and uninfected conspecifics of the same or opposite sex by volatile and involatile urinary odors, displaying aversive response to, and avoidance of the odors of infected mice. This recognition and avoidance involves OT and ERs. OTKO and ERKO (alpha or beta) mice are specifically impaired in their recognition of, aversion to, and memory of the odors of infected individuals. The olfactory and mate choice responses of females can be further modulated by social factors such as previous experience and the mate choices of other females. Female mice use indirect social information from cues produced by individuals with similar interests and requirement. This mate copying, which can modulate and attenuate the aversive responses to infected individuals, also involves OT. Thus, estrogenic regulation of the OT system controls social recognition and adaptive social

behaviors such as the olfactory based avoidance of parasitized conspecifics, mate choices and mate copying. Supported by NSERC (MK and EC),

SEX, FEAR AND PARASITES: PARASITIC MANIPULATION OF SEMIOCHEMICALS IN BROWN RAT. Vyas, A. Nanyang Technological University, Singapore 637551. avyas@ntu.edu.sg To be successful, a male animal typically requires two traits: ability to attract mates and avoid predation. In case of brown rats, both of these abilities are guided by olfactory cues. Rats eavesdrop on odor cues left by predators like cats; and show innate fear to these scents. Similarly, male rats communicate their reproductive worth through urinary pheromones; while females detect and detest urinary marks of parasitized males. Both of these responses are strong and hard-wired, reflecting intense selection pressure for their maintenance. Yet, these responses are detrimental to parasites that require trophic transmission and/or sexual transmission. Protozoan parasite, Toxoplasma gondii, exactly fits this description. It obligatorily requires cat intestines to sexually reproduce; and it can be transmitted through sexual intercourse. Innate olfactory responses of rats are decidedly a bad news for this parasite. Toxoplasma circumvents these obstacles by manipulating response of host to odors. It induces attraction to cat odor and imparts male pheromones greater attractiveness. It is likely that same biological mediators underlie both of these behavioral manipulations.

11:15-12:15 **Presidential Lecture: Kelly Lambert**, Randolph-Macon College
The Dynamic Parental Brain: More than a Mom and Pop Operation.

THE DYNAMIC PARENTAL BRAIN: MORE THAT A MOM AND POP OPERATION. Lambert, K. G. Department of Psychology, Randolph-Macon College, Ashland VA 23005 USA. As mammals transition from virgin to parental status, multiple neurobiological modifications accompany the redirection of attention and resources from self-care to nurturing offspring. In the maternal rat, for example, more efficient foraging strategies, enhanced motor agility, mitigated stress/anxiety responses, and increased resilience following neural insult characterize the nature of effects observed in our laboratory. These seemingly adaptive behavioral modifications are accompanied by neuroplastic responses in brain areas, such as the hippocampus, typically not associated with maternal behavior. The convergence of these findings emphasizes the dramatic neurobiological alterations associated with the transition from the virgin to maternal brain, enabling the new mother to meet the many demands of motherhood. Alternatively, the male plays a less essential role in caring for offspring in most mammals; however, we have used the California Deer Mouse (Peromysucs califonricus) as a paternal model in our lab to investigate neurobiological changes accompanying parenthood in the absence of pregnancy, parturition and lactation. Compared to the non-paternal common deer mouse (Peromyscus maniculatus), the California mice exhibit decreased activation in fear circuits, along with increased involvement of vasopressin and oxytocin, as they exhibit nurturing responses such as retrieval and grooming of pups. In accordance with the maternal models we have investigated, the paternal model confirms the multifaceted nature of the emergence and maintenance of nurturing responses characterizing parental behavior-neurobiological changes that may be viewed as the basic neuroarchitecture of more general affiliative social behaviors.

3:00-5:15 **Symposium 2:** OREXIN/HYPOCRETIN'S ROLE IN MOBILIZING ADAPTIVE AND PATHOLOGICAL PANIC AND ANXIETY RESPONSES. **Co-Chairs: Anantha Shekhar and Philip L. Johnson**

ROLE OF OREXIN IN MOBILIZING ADAPTIVE BEHAVIORAL AND PHYSIOLOGICAL RESPONSES. Shekhar, A.1,2; Truitt, W.3; Samuels, B.4; Fitz, S.D.2; Lowry, C.A.5; Johnson, P.L.2. 1 Indiana Clinical and Translational Sciences Institute and Departments of 2Psychiatry, 3Anatomy and Cell Biology, 4STARK Neuroscience Research Institute, Indiana University School of Medicine, Indianapolis, IN, U.S.A; 5Department of Integrative Physiology, University of Colorado, Boulder, CO, USA A panic response is an adaptive response to deal with an imminent threat and consists of an integrated pattern of behavioral, metabolic, cardiorespiratory, thermoregulatory, and endocrine responses that are highly conserved across vertebrate species. In the 1920s and 1940s Philip Bard and Walter Hess respectively determined that the posterior regions of the hypothalamus are critical for a fight-or-flight reaction to deal with an imminent threat. These hypothalamic regions have more recently localized to be comprised of the perifornical (PeF) and dorsomedial hypothalamus (DMH) regions. In 1998 a novel wake-promoting neuropeptide called orexin/hypocretin (ORX) was discovered and determined to be exclusively synthesized in the DMH/PeF and adjacent lateral hypothalamus. Here we review data that, taken together, show ORXs ability to mobilize coordinated adaptive panic-associated behavioral and cardioexcitation in response to: 1) ethologically relevant stressor (predator odor); 2) direct local disinhibition with a GABAA receptor antagonist; 3) anxiogenic drugs (i.e., FG-7142, caffeine and yohimbine); 4) and a panicogenic challenge (i.e., 20%CO2 exposure). Acknowledgement: RO1 MH52619 to AS and RO1 MH065702 to AS and CAL.Indiana CTSI Project Development Award, NIH Student LRP, and National Alliance for Schizophrenia and Depression Young Investigators Award to PLJ.

EVIDENCE THAT OREXIN IS A CRITICAL SUBSTRATE UNDERLYING PATHOLOGICAL ANXIETY AND PANIC RESPONSES IN RATS AND HUMANS. Philip L. Johnson1; William Truitt1,2; Stephanie D. Fitz1; Pamela E. Minick1; Amy Dietrich1; Sonal Sanghani3; Lil Trskman-Bendz4; Andrew W. Goddard1; Lena Brundin4; and Anantha Shekhar1,5. Departments of 1Psychiatry, 2Anatomy and Cell Biology, and 3Biochemistry, Indiana University School of Medicine, Indianapolis, IN, U.S.A; 4Section of Psychiatry, Department of Clinical Sciences, Lund University Hospital, Lund, Sweden; 5 Indiana Clinical and Translational Sciences Institute, Indianapolis, IN, USA Panic disorder is a severe anxiety disorder with recurrent, debilitating panic attacks. In subjects with panic disorder there is evidence of decreased central GABAergic activity as well as marked increases in autonomic and respiratory responses following ordinarily mild interoceptive stressors [e.g., intravenous infusions of 0.5M sodium lactate (Goddard et al., 2004, Am.J.Psychiatry)]. Similarly, in an animal model of panic disorder, chronic inhibition of GABA synthesis in the dorsomedial/perifornical hypothalamus (DMH/PeF) of rats produces anxiety-like states and a similar vulnerability to sodium lactate-induced cardioexcitatory responses (Shekhar and Keim,1997; Johnson and Shekhar,2006, J.Neurosci.). Within the brain, orexin (ORX, also known as hypocretin)-containing neurons are exclusive to the DMH/PeF and adjacent lateral hypothalamus and play a critical role in arousal, vigilance and central autonomic mobilization, all of which are key components of panic (Sakurai, 2007, Nat.Rev.Neurosci.). In a recent publication (Johnson, et al.,2010,Nat.Med.), we demonstrate that activation of ORX neurons is necessary for developing a panic-prone state in the animal panic model, and either silencing the hypothalamic ORX gene product with RNA interference or systemic ORX1 antagonists blocks the panic responses. Moreover, we show that humans with panic anxiety have elevated levels of ORX in the cerebrospinal fluid compared to humans without panic anxiety. Taken together our results suggest that the ORX system may be involved in the pathophysiology of panic anxiety, and that ORX antagonists constitute a potential novel treatment strategy for panic disorder. Acknowledgement: Indiana CTSI Project Development Award, NIH Student LRP, and National Alliance for Schizophrenia and Depression Young Investigators Award to PLJ; RO1 MH52619 and RO1 MH065702 to AS.

NEURAL CIRCUITS AND MECHANISMS INVOLVED IN OREXIN A-INDUCED ANXIETY-LIKE BEHAVIOR. Truitt, W.A.1; Johnson, P.L.2; Molosh, A.2; Lungwitz, E.3; Harvey, B.4; Dietrich, A.D.1; Minick, P.E.1 and Shekhar5, A. Departments of 1Anatomy and Cellular Biology, 2Psychiatry, and 3Medical Neurobiology program, Indiana University School of Medicine; 4Project SEED; 5 Indiana Clinical and Translational Sciences Institute, Indianapolis, IN, USA. Recently we reported that the orexin (ORX) system is pivotally involved in anxiety and panic-like responses. Specifically, ORX receptor antagonist and silencing the ORX gene block anxiety and panic-like responses to sodium lactate panicogenic challenges in rats. Here we present data demonstrating that the bed nucleus of the stria terminalis (BNST) is a putative site of action for ORXs involvement in anxiety. Systemic ORX 1 receptor antagonists block anxiety-like and BNST induced cFos responses to systemic injections of N-methyl-beta-carboline-3-carboxamide (FG-7142). ORX 1 receptor antagonists placed directly into the BNST also blocks lactate-induced anxiety. Conversely, ORX A injections directly into the BNST induces anxiety-like responses. In ORX neuron terminals, ORX co-localize and is co-released with glutamate. In mesencephalic regions, ORX potentiates ionotropic glutamate receptor action and ORXs postsynaptic action appears to be dependent on glutamate signaling. To determine if the anxiety-inducing effects of ORX A in the BNST also is dependent on glutamate, rats received BNST injections of NMDA or AMPA receptor antagonists prior to ORX A BNST injections. Anxiety-like responses to ORX A were completely blocked by NMDA antagonist and partially blocked with AMPA receptor antagonists. ORX A in the BNST also increased phosphorylation of NMDA receptors. Supported by RO1s MH52619 and MH065702.

THE ROLE OF OREXIN AND ITS RECEPTORS IN BEHAVIORAL AND PHYSIOLOGIC RESPONSES EVOKED BY AMPHETAMINES Rusyniak, D.E. Dept. of Emergency Medicine, Pharmacology and Toxicology, Indiana University School of Medicine, Indianapolis, IN 46202 USA Amphetamine abuse and its complications are global epidemics with the number of world-wide users surpassing that of cocaine and heroin. An increase in the number of users has resulted in a concomitant increase in the number of medical complications: Myocardial infarction, ischemic and hemorrhagic stroke, rhabdomyolysis and renal failure, and in severe cases fatal hyperthermia. In addition to increasing mortality, hyperthermia evoked by amphetamines is associated with the development of dopaminergic and serotonergic neurotoxicity. One of the difficulties in developing specific therapies for amphetamine-related hyperthermia has been a lack of knowledge of the central pathways involved. Work from our laboratory has shown that a key brain region involved in mediating hyperthermia and behavioral responses to amphetamines is the dorsomedial hypothalamus (DHM). In the past decade, the DMH has emerged as a key hypothalamic effector region whose activation plays an important role in generating fever and stress responses. Inhibiting neural activity in the DMH region prevents hyperthermia and locomotor responses to amphetamines. Within and just lateral to the DMH are neurons that synthesize preproorexin which is then cleaved into the neuropeptides orexin-A and orexin-B. The orexin neurons and their receptors are involved in the regulation of feeding, wakefulness, heart rate, locomotion, and body temperature. Amphetamines affect similar systems and have been shown to increase the expression of c-Fos (a marker of neuronal activity) in orexin neurons. Therefore, orexin neurons and their receptors make ideal targets for investigating the central mechanisms behind amphetamine-evoked responses. Orexin receptors appear to mediate the responses to amphetamines and our laboratory has recently shown that blocking orexin-1 receptors significantly inhibits the hyperthermia brought on by methamphetamine.

5:30-8:00 **Past Presidents' Symposium**

BRINGING NATURAL BEHAVIORS INTO THE LAB: MODELING SOCIAL AND DEFENSIVE BEHAVIORS IN RODENTS. Blanchard, R.J. Department of Psychology, University of Hawaii. Bringing natural behaviors into the lab has involved a two-part process. First, semi-natural situations such as the Visible Burrow System provide rodents with features such as a social group, tunnels and burrows, space to facilitate social avoidance, etc. that are typically found in the natural environment and may support and facilitate successful outcomes of specific behaviors. These, used in conjunction with exposure to a predator or other manipulations, provide an overview of the range of behaviors relevant to sociality or defensiveness. A second component of this strategy is to specifically manipulate relevant features of this environment in a more focused and restricted situation, in order to confirm (or disconfirm!) the relationship between supporting or facilitating environmental stimuli, and behavioral responses. A defense-relevant example is the Mouse Defense Test Battery, in which particular defensive responses to approach and contact by a predator are strongly determined by features such as availability of escape, predator-prey distance, and predator ambiguity. For social behaviors, when an environment allows an animal to avoid aversive conspecific contact, this is typically the prepotent response. However, when such avoidance is precluded by environmental manipulations, antisocial motivations may be expressed by more specific behavior patterns, additionally permitting an enhanced analysis of the animals motivational state. Such specificity may provide a more detailed and secure basis for the translation of rodent behaviors to normal and abnormal human response patterns, facilitating the use of animal models for investigation of genetic and neural correlates of psychopathology.

THE HEALING POWER OF LOVE: AN OXYTOCIN HYPOTHESIS. Carter, C. S., The Brain Body Center, Department of Psychiatry, University of Illinois at Chicago. Loving relationships may be protective or restorative in the face of challenge or illness, possibly through actions that involve the mammalian neuropeptide, oxytocin. Oxytocin, was originally believed to be only a female hormone, involved primarily in birth and lactation. However, it is now clear that, in both sexes, oxytocin plays an essential role in the selective sociality that characterizes love. Oxytocin also may regulate endocrine and autonomic reactivity to stressors, reduce fear, alter the detection of subtle emotional signals, and increase trust. Drawing primarily on research in humans and the socially monogamous prairie vole, this presentation will examine mechanisms capable of regulating the release of endogenous oxytocin, especially in the face of major life challenges and across the mammalian lifespan. We also will examine novel neurobiological mechanisms and properties through which oxytocin may help to explain the health benefits of love.

TRANSLATING LABORATORY DISCOVERY TO THE CLINIC: FROM ANTIDEPRESSANT TO STEM CELL THERAPY Dr. Paul R. Sanberg Senior Associate Vice President, Office of Research and Innovation Distinguished University Professor Executive Director, Center of Excellence for Aging and Brain Repair Vice-Chair, Department of Neurosurgery and Brain Repair University of South Florida 12901 Bruce B. Downs Blvd, MDC 78, Tampa, Florida, USA The continued successful development of the antidepressant drug TC-5214, a very long journey in discovery, began with the behavioral neuroscience of nicotine in rats to a search for an effective treatment for children with Tourette syndrome. This project led to the discovery of a decades-old blood pressure medicine, mecamylamine. Which in clinical trials demonstrated beneficial effects in depression. Eventually a unique version of the drug was created, patented, and is now the subject of one of the largest pharmaceutical licensing agreements in the U.S., between Targacept, USF and Astra Zeneca. Stem cell therapy is a promising approach to the treatment of a variety of human degenerative diseases and injury that has been shown to be useful in many animal models and in early clinical trials. The use of embryonic stem cells has limited their acceptability and wide spread application in certain countries. The last few years have witnessed an expansion in stem cell research and the potential for therapy following the revolutionary experiments in models of numerous human disorders. In addition to controversial embryonic stem cell research, adult stem cell sources like hematopoietic stem cells, mesenchymal stem cells, epidermal stem cells, pancreatic stem cells, and several other organ stem cells are currently identified and characterized in laboratories all over the world. We have focused primarily on hematopoietic derived cells in bone marrow and umbilical cord blood, showing potential therapeutic applications in stroke, Alzheimer's and ALS animal models. The future of stem cells and regenerative medicine is open-ended with science, politics, ethics and commercial interests all playing a significant role. With the ongoing discovery of novel cell therapies, the applications for brain repair become increasingly promising. With every discipline of science, this will require continuous well-thought-out research, years of study, and dedicated investigators. This talk will focus on the process of translating laboratory discovery to the clinic. By building a foundation of excellence in basic and clinical research, while focusing on translating innovative ideas into industrial partnerships, educational and clinical services; addressing key needs of the community and those suffering from brain injury/disease and depression. (PRS is a consultant of Astra Zeneca and Saneron CCEL Therapeutics, Inc.)

8:30-10:45 **Symposium 3**: EXAMINING THE GENETIC AND NEURAL COMPONENTS OF COGNITIVE FLEXIBILITY USING MICE. Co-Chairs: **Jared W. Young and Jonathan L. Brigman**

BALANCING FLEXIBILTY AND EFFICIENT ACTION: CORTICOSTRIATAL NETWORKS IN THE MOUSE Brigman1, J.L.; Wright1; T., Davis2; M.I.; Saksida3; L.M., Bussey3; T.J., Jiang4; Z., Lovinger2; D.M.; Nakazawa4, K; and Holmes1, A; 1Section on Behavioral Science and Genetics, NIAAA,, NIH; 2Section on Synaptic Physiology, NIAAA, NIH; 3Department of Experimental Psychology, University of Cambridge and Medical Research Council and Wellcome Trust Behavioral and Clinical Neuroscience Institute, UK; 4 Unit on Genetics of Cognition and Behavior, NIMH, NIH Imbalance between behavioral flexibility and habit is theorized to contribute to a wide range of neuropsychiatric disorders as well as drug addiction and the glutamatergic system has been implicated in these processes. We utilized the pharmacological and genetic tools to investigate the contribution of corticostriatal networks and NMDARs to cognitive flexibility and habit-like behavior in a touchscreen-based visual discrimination and reversal task. To establish the neural circuits underlying the behaviors, patterns of cortical and striatal neuronal activation associated with performance were mapped using the immediate early gene c-Fos. Next, ex vivo electrophysiological analysis of glutamate-mediated neuronal transmission and plasticity in dorsolateral striatal (DLS) slices was performed in mice that had been trained to the same performance stages. The contribution of GluN2B-containing NMDARs to these behaviors was examined by testing the performance of mutant mice either lacking GluN2B in cortex (and CA1 hippocampus) or cortex and striatum. To further elucidate the role of the GluN2B subunit, non-mutant mice injected with GluN2B antagonist directly into DLS or lateral orbitofrontal cortex (lOFC) were tested on early or late stage reversal learning. To examine the in vivo patterns of neuronal activation that accompanies task behavior, electrophysiological recordings were made in the DLS of mice performing at different stages of proficiency. Results showed that early (perseverative) reversal performance was associated with activation of prefrontal regions, while late (well-learned/habit-like) discrimination performance was associated with high DLS activation. Synaptic plasticity was greatest when performance was intermediate and choice behavior was at chance. Loss of GluN2B in cortex was sufficient to impair reversal, while GluN2B in cortex and striatum impaired discrimination. GluN2B antagonism in lOFC impaired early reversal performance by increasing perseveration but not late stage learning while antagonism in DLS significantly impaired late, but not early learning. Results from in vivo recording showed learning related changes in striatal firing patterns. Collectively, these findings help demonstrate a major role for GluN2B in regulating corticostriatal control of flexibility and habit. Research supported by the National Institute on Alcohol Abuse and Alcoholism Intramural Research Program.

SYSTEMS ANALYSIS OF CEREBELLAR MODULATION OF EXECUTIVE FUNCTION. Dickson, P.E. 1; Martin L.A. 2; Rogers, T.D. 1; Blaha, C.D. 1; Goldowitz, D. 3; Mittleman, G. 1 1. Dept of Psychology, University of Memphis, Memphis, TN 38152. 2. Azusa Pacific University, Azusa, CA 91702. 3. Dept Medical Genetics, University of British Columbia, Vancouver, BC V5Z 4H4. Neuroimaging studies showing cerebellar activation during cognitive tasks and research demonstrating cerebellar pathophysiology coupled with deficits in executive functions (e.g., autism) provide evidence for a cerebellar role in cognition. We investigated the relationship between developmental loss of cerebellar Purkinje cells (the sole outflow of cerebellar cortex) and cognitive dysfunction through a series of studies utilizing aggregation chimeras made from wildtype and lurcher mouse embryos. Lurcher mice lose 100% of their cerebellar Purkinje cells while individual wildtypelurcher chimeras have between 0 and 100% of normal Purkinje cell numbers. We assessed the performance of these chimeras in operant tasks designed to measure spatial working memory, sustained attention, perseverative lever pressing, and serial reversal learning of a conditional visual discrimination. The strategy in all experiments was to determine if Purkinje cell numbers correlated with behavioral measures in order to determine what cognitive abilities were or were not affected by Purkinje cell loss. We found that Purkinje cell numbers were significantly correlated with serial reversal learning and perseverative lever pressing in that lower numbers were associated with more learning errors and enhanced perseveration. A second set of experiments used in vivo electrochemistry to examine possible mechanisms by which efferent projections from the cerebellum could modulate prefrontal cortex (PFC) function. Lurcher mice showed altered dopamine function in the medial PFC which could account for the observed altered performance on behavioral measures of executive function. These experiments suggest (1) a causal relationship between cerebellar pathophysiology and executive function deficits and (2) a possible mechanism for these observed effects.

FAST SPIKING INTERNEURONS ASSIST CORTICAL TUNING DURING FRONTAL MEDIATED TASKS. Powell, E.M.; Xu., J.; Bissonette, G.B., Dept of Anatomy and Neurobiology, University of Maryland School of Medicine, Baltimore, MD 21201, USA In a constantly changing environment, the ability to shift from one learned behavioral strategy to another more adaptive strategy is imperative. Research suggests there may be common underlying alterations to common neural circuits for the similar cognitive etiologies which are observed in multiple psychiatric disorders. One of these common anatomical manifestations involves deficits to the GABAergic system in the cerebral cortex, specifically to the fast spiking parvalbumin expressing interneurons. When these interneurons are disrupted in the mouse orbital frontal cortex (OFC),

behavioral deficits in ability to optimally modify learned associations and update decision making behaviors become apparent. In vitro research has implicated PV+ interneurons are key to generation of high frequency oscillations, coordinating large ensembles of neurons. Postmortem anatomical studies of human patients diagnosed with schizophrenia show altered PV+ cells in the frontal cortical areas. To better understand the functional changes elicited by such a deficit, we have developed a mouse task in which we can record in vivo single unit activity in awake behaving animals to evaluate murine OFC function during reversal learning. Further, we have studied the role of developmental alterations to cortical GABAergic tone in decision making. Using a transgenic animal model to produce a specific frontal cortical GABAergic deficit in adult mice, we have assessed cortical function during decision making through behavioral and in vivo physiological techniques, using single cell and local field potential recordings. Our research works to illuminate a common neural substrate for learning and prefrontal decision related processes between mouse neuroanatomical circuitry and behavior with human cortical function in psychiatric disease states. Support: This work is supported by NARSAD (EMP), R01 DA018826 (EMP), and R01 MH57689 (EMP).

DELAYING THE EUREKA MOMENT BY REMOVING THE ALPHA 7 NICOTINIC ACETYLCHOLINE RECEPTOR. Young, J.W.; Meves, J.M.; Tarantino, I.S.; Caldwell, S., and Geyer, M.A. Department of Psychiatry, University of California San Diego, 9500 Gilman Drive MC 0804, La Jolla, CA 92093-0804 The a7 nicotinic acetylcholine receptor (nAChR) has long been a procognitive therapeutic target to treat schizophrenia. Evidence for the role of this receptor in cognition has been lacking however, in part due to the limited availability of suitable ligands. Behavior of a7 nAChR knockout (KO) mice has been examined but cognitive assessment using tests with cross-species translatability have been limited. We assessed the performance of a7 nAChR KO and wildtype (WT) littermate mice in 1) the attentional set-shifting task (ASST) of executive functioning, 2) the radial arm maze test (RAM) of spatial working memory span capacity, and 3) the novel object recognition test of short-term memory. Motivation was assessed using 4) the progressive ratio breakpoint study (PRBS), with 5) the Behavioral Pattern Monitor used to assess exploration, and 6) prepulse inhibition used to assess sensorimotor gating. a7 nAChR KO mice exhibited comparable set-shifting, spatial span capacity, short-term memory, motivation, exploration, and sensorimotor gating to WT littermates. Impaired conceptual learning (rule acquisition) across multiple paradigms, stimuli, and modalities (ASST-digging in bowls, RAM-arm entries, PRBS-holepoking) was observed in the KO mice however. The data presented here support the notion that this receptor is important for the eureka moment, when patterns in the environment become clear and a rule is learned. This finding along with the impaired attention observed previously in these mice suggest agonist treatment studies should examine effects on attention and conceptual learning clinically, perhaps in combination with cognitive behavioral therapy.

11:15-12:15 **Keynote Speaker: Janice Kiecolt-Glaser**, Ohio State University
How Stress Kills: Perspectives from psychoneuroimmunology

HOW STRESS KILLS: PERSPECTIVES FROM PSYCHONEUROIMMUNOLOGY. Kiecolt-Glaser, Janice K. Institute for Behavioral Medicine Research, The Ohio State University College of Medicine, Columbus, OH 43210 Inflammation can be substantially enhanced by stress and depression. Inflammation influences the onset and course of a spectrum of conditions associated with aging including cardiovascular disease, type II diabetes, osteoporosis, Alzheimer's disease, and frailty and functional decline. Furthermore, stress and depression also contribute to greater risk for infection, prolonged infectious episodes, and delayed wound healing, all processes that indirectly fuel sustained proinflammatory cytokine production. Compounding the risks, health behaviors such as poor sleep are commonplace correlates of stress and depression that further enhance proinflammatory cytokine production. In addition to these pathways, stress and depression can permanently alter the responsiveness of the immune system; stressors can effectively prime the inflammatory response, promoting larger proinflammatory cytokine increases in response to subsequent stressors and/or minor infectious challenges. Through these pathways stress and depression may influence the incidence and progression of age-related diseases.

2:00-4:00 **Media and Science Session**

SCIENTISTS FROM MARS; JOURNALISTS ARE FROM VENUS. Sandra Blakeslee, Science Writer/Author, *New York Times.* As the number of full-time science journalists writing for main stream media shrinks, more and more scientists realize that they need to take a larger role in publicizing their research. But how? What lessons can scientists learn from journalists? How difficult is the transition from laboratory bench to published writer? Sandra Blakeslee is co-director of the annual Santa Fe Science Writing Workshop which draws scientists from around the world who are interested in the nuts and bolts of science reporting. http://sciwrite.org

THE SCIENTIST'S ROLE IN PUBLIC OUTREACH. **Mariette DiChristina**, Editor-in-Chief, *Scientific American*

OPEN SCIENCE: HOW THE RISE OF SCIENCE BLOGS IS CHANGING RESEARCH. Raeburn, P. In December, 2010, NASA held a press conference to announce the discovery of arsenic-based life in Californias Mono Lake. The authors of the study, which appeared in Science, suggested that the existence of this life form on Earth made it more likely than ever that life could exist on other planets in seemingly hostile environments. But the paper was attacked by scientist-bloggers within hours. Neither NASA nor Science has issued any substantive response and the papers findings now reside in some nightmarish world of uncertainty. The same thing could happen to any paper that is particularly controversial including much of what is published by behavioral neuroscientists.

4:00-5:30 **Oral Session 1:** Chairperson: **Nancy Ostrowski**

UNUSUAL IMMUNE REGULATION IN THE BTBR T + tf/J MOUSE: POSSIBLE MECHANISM FOR THE AUTISTIC LIKE PHENOTYPE OBSERVED IN THIS STRAIN. Benno, R.; Schanz, N.; Pettit, L.; Vassiliou, E. Biology Department, William Paterson University, Wayne, NJ 07470, USA and Department of Biological Sciences, Kean University, Union, NJ 07083, USA. Studies on the BTBR T+ tf/J (BTBR) mouse have shown that this strain may serve as a model system for Autism Spectrum Disorder (ASD). The BTBR mouse has proven to be valuable for these ASD studies since it meets all of the three defining symptoms of autism: poor social communication, minimal social interaction and repetitive behaviors that typically involve interaction with a limited number of objects. While the validity of the BTBR mouse as a model for ASDs has been firmly established, it is unclear as to the possible underlying mechanisms responsible for these aberrant behaviors. Several lines of evidence point to the fact that a differential immune regulation in the BTBR strain may be responsible for this autistic phenotype. For example, in a survey of 11 inbred strains, BTBR mice were shown to have significantly higher numbers of T-reg lymphocytes compared to all other strains. BTBRs have also been shown to have an unusual SNP for the enzyme KMO, which has been implicated in microglial initiated neuropathology. In addition, preliminary studies in our laboratory have shown that there are differences in the response of the BTBR mouse to in utero exposure to the viral mimic poly I:C during gestation. In this study, we investigated the immune response in terms of the pro and anti-inflammatory cytokines in the BTBR, the 129S and the C57BL/6J mouse strains. Adult female mice from these three strains were injected with poly I:C (10 mg/kg) and three hours later their plasma and brains were analyzed. Our results support our working hypothesis that there is a differential immune regulation in the autistic like BTBR mouse, which may be responsible for this abnormal phenotype relative to the 129S and C57 controls..

LEUKEMIA IN MICE INDUCES STRESS-LIKE CHANGES IN BEHAVIOR, HPA AXIS AND CATECHOLAMINES AND INDOLEAMINES. Dunn, A.J., LSUHSC-Shreveport and University of Hawaii, Swiergiel, A.H., LSUHSC, and Newman, R.A. M.D. Anderson Cancer Center, Houston, Texas. Cancer in humans is known to be associated with neurobehavioral disturbances, including depression and fatigue, but the underlying mechanisms have not been established. We have studied the responses of DBA2 mice inoculated intraperitoneally with L1210 mouse leukemia cells at doses of either 5,000 or 50,000 cells per mouse. At various subsequent times, the behavioral activity of the mice was assessed in the open field test, the tail-suspension test (TST), and the Porsolt forced swim test (FST). No consistent differences from controls were observed when the behavior of the inoculated animals was studied on Days 8 or 11 following inoculation. However, on Day 15, mice inoculated with either dose of leukemia cells exhibited statistically significant increases in immobility in the TST and the FST, considered to parallel depression-like behavior. In the open field, the mice injected with 50,000 L1210 cells showed statistically significant decreases in activity at this time, but those that received only 5,000 cells did not. This suggests that the depression-like behavior in the TST and FST was not attributable solely to a general reduction in activity. Plasma corticosterone was elevated in the inoculated mice at both doses, indicating activation of the hypothalamic-pituitary adrenocortical (HPA) axis. The mice also exhibited statistically significant increases in the metabolism of norepinephrine (NE), but not dopamine (DA), in the hypothalamus, as well as increases in tryptophan and serotonin (5 hydroxytryptamine, 5-HT) metabolism (indicated by increases in 5 hydroxyindoleacetic acid, 5-HIAA) in the cortex and hypothalamus. The HPA activation and that of brain noradrenergic and serotonergic systems are characteristic stress responses, and may underlie the behavioral responses. The behavioral data indicate the depression-like activity of tumor inoculation, and echo the depression frequently observed in cancer patients. These observations suggest that leukemic mice exhibit neurobehavioral disturbances that resemble the responses observed in humans. Thus the mouse model may be useful for assessing potential therapies for the neurobehavioral disturbances observed in human cancer patients.

AUTOPHAGY ENHANCEMENT: A POSSIBLE NEW DRUG TARGET FOR AFFECTIVE DISORDERS. Einat, H.; Anderson, G.W. Dept. of PPPS, College of Pharmacy, University of Minnesota, Duluth, MN 55812, USA. The pathophysiology of affective disorders and therapeutic basis of effective treatments are not clear. Recent data implicates cellular resilience in these mechanisms. Recent data also shows that mood stabilizers enhance autophagy in-vitro, a pathway for clearance of toxic and aggregate proteins. It is therefore suggested that enhancing autophagy can provide a target for novel treatments. To test this possibility, we evaluated the behavioral and biochemical effects of 3 dissimilar drugs that enhance autophagy in-vitro: rapamycin, nicardipine and trehalose. The results show that rapamycin induces antidepressant- but not antimanic-like effects in rodents. Nicardipine has marginal antidepressant- and antimanic-like effects whereas the effects of trehalose were ambiguous. Biochemical evaluation of autophagy induction in brain suggests that at least for rapamycin, there is an enhancement of autophagy after peripheral administration at the same dose and regimen needed to induce the behavioral effects. Whereas these results are not unequivocal, it is suggested that they support the hypothesis that autophagy enhancement might be a potential new drug target for affective disorders. It is important to note that the effects of the tested drugs might also be related to other mechanisms. Rapamycin has significant effects on the immune system and nicardipine is a Ca++ channel blocker and both mechanisms have been connected with affective disorders. Yet, the dissimilarity between the drugs and their mechanistic conversion on enhancing autophagy do offer support to the possibility that autophagy enhancement may, at least in part, be related to their behavioral effects.

ANTIDEPRESSANT-LIKE ACTIONS OF DIETARY CHOLINE SUPPLEMENTATION IN A RAT MODEL. Glenn, M.J.; Adams, R.; Saporta, A.N.; Cameron, S.; Gillies, S.; McClurg, L.M. Dept. of Psychology. Colby College, Waterville, ME 04901. Choline is an essential nutrient that serves a host of functions in the brain and body. Among these are its critical contributions to nerve impulse transmission as the precursor to the neurotransmitter acetylcholine and to gene transcription as a key component in signaling pathways and an important source of methyl groups. It is well known that levels of choline during pre- and postnatal development have important ramifications for adult neural form, function, and plasticity. Numerous studies also reveal that there is a profound impact on adult cognition and emerging data from our lab indicate that emotionality may also be affected. In the present study we explored the hypothesis that such changes by choline may buffer the brain from the negative and potentially pathological consequences of stress. To study this we administered a choline-supplemented synthetic diet (AIN76A with 5 g/kg choline chloride) or a choline-sufficient diet (also AIN76A but with 1 g/kg choline chloride) to rats during the course of prenatal or periadolescent development and then in adulthood we examined their emotional and cognitive responses to the acutely stressful forced swim test or to mild unpredictable chronic stress. After the completion of behavioral tests rats were sacrificed and brains retained for assays of neural plasticity. Our findings compelling revealed antidepressant properties of developmental choline supplementation: supplemented rats exhibited more resistance (less immobility) in the forced swim test and had accompanying increases in hippocampal neurogenesis and growth factor expression. The effects of chronic mild stress were less robust but supportive. Taken together, these findings support our hypothesis and add to the mounting evidence that choline is neuroprotective in models of human diseases.

XAMOTEROL RESCUES MEMORY DEFICIT IN MOUSE MODEL OF DOWN SYNDROME BY ACTIVATION OF BETA-1 ADRENERGIC RECEPTOR. Faizi, M.; Mobley, W.C.; Coutellier, L.; Shamloo, M. Stanford Behavioral and Functional Neuroscience Laboratory, Stanford Institute for Neuro-Innovation and Translational Neuroscience, Stanford University School of Medicine, Stanford, CA 94305, USA. Department of Neuroscience, University of California, San Diego, La Jolla, CA 92093, USA. Down Syndrome is a trisomy of chromosome 21 and is the most prevalent form of intellectual disability caused by genetic abnormalities in humans. The Ts65Dn mouse is a well known genetic model of Down Syndrome and shares a number of physical and functional abnormalities with people with trisomy for chromosome 21. Both Down Syndrome people and Ts65Dn mice have shown abnormalities in Locus coeruleus. It is well established that the Locus coeruleus is the main source of norepinephrine in the brain and has neuronal projections to different areas of the brain, including the hippocampus and frontal cortex. We have recently shown that restoration of norepinephrine level using L-threo-3,4-dihydroxyphenylserine in Ts65Dn mice restored the hippocampal-mediated contextual deficit in fear conditioning as well as nesting behavior. In order to study the role of beta-1 adrenergic receptor as post synaptic target in mediation of these behavioral effects, a selective beta-1 receptor agonist, xamoterol and a beta-1 receptor antagonist, betaxolol were evaluated in battery of learning and memory assays. Xamoterol did not show a significant effect on locomotor activity of male Ts65Dn mice. However, xamoterol rescued the spontaneous alternation deficit observed in T-maze, and its effect was blocked by the selective B1 receptor antagonist, betaxolol. Similarly, xamoterol improved the memory retrieval of Ts65Dn mice in contextual fear conditioning and the effect of xamoterol was completely blocked by betaxolol. In novel object recognition, xamoterol improved the novel object recognition memory in Ts65Dn mice and had no effect on performance of control mice. These results suggest that the hippocampal-related behavioral deficit observed in the Ts65Dn is mediated by a decreased beta-1 receptor signaling in hippocampus, and a selective activation of this receptor could be used as therapeutic approach for enhancement of learning and memory in Down Syndrome or other neurodegenerative disorders.

DOMINANCE AND SUBMISSIVENESS IN THE ETIOLOGY AND TREATMENT OF AFFECTIVE DISORDERS. Pinhasov, A. Department of Molecular Biology, Ariel University Center of Samaria, Ariel, 40700, Israel. Social interactions characterized by relationships of dominance and submissiveness occur at all levels of the animal kingdom, and together with genetic and environmental factors, they contribute to the formation of the social structure. Using the dominant-submissive relationship (DSR) test, we identify mice with dominant or submissive features. In this social interaction test, animals compete for sweetened milk for 5 minutes a day during a 2 week period. Under standard conditions, about 25% of animals develop DSR. Using a selective breeding approach, and based upon the DSR paradigm, we found that the percentage of animals that developed DSR increased in each subsequent generation. Thus, over 98% of the 10th generation of selectively bred animals developed strong and stable DSR. Moreover, the onset of DSR formation began earlier, and the par in drinking time between dominant and submissive animals increased markedly, over consecutive generations. Antidepressants of different types gradually decreased submissive behavior. A mood stabilizer lithium selectively influenced animals of the dominant genotype. The difference between the behavioral phenotypes of these two mice populations, and the dissimilar effects of drugs upon their behavior, are also reflected in their molecular parameters as well as their divergent biochemical responses to stress, a factor granted importance in the etiology of human depression. These two distinct populations of mice represent a valuable and valid model for studying dominance and submissiveness as important elements of social interaction, and for the screening of potential antidepressants and mood stabilizing agents.

6:30-8:30 **Poster Session 1:**

Anxiety and Stress

1. THE EFFECT OF ANXIOGENICS ON PREFRONTAL CORTICAL SINGLE UNIT ACTIVITY, LOCAL FIELD POTENTIAL OSCILLATIONS AND BEHAVIORAL FLEXIBILITY IN FREELY-MOVING RATS. Bondi, CO; Burkowski, AJ; Del Arco, A; Wood, J; Moghaddam, B. Dept Neuroscience, Univ. of Pittsburgh, Pittsburgh, PA. Stress exposure is a major risk factor in many psychiatric disorders and alters various cognitive functions. We were interested in how pharmacological stressors alter prefrontal cortical (PFC) neurophysiology and performance during a test of behavioral flexibility. FG7142 (N-methyl-beta-carboline-3-carboxamide), a benzodiazepine receptor partial inverse agonist, simulates the neurochemical and physiological changes induced by stress. First, we administered several doses of FG7142 to examine the effect of stress-like conditions on single-unit firing and local field potential (LFP) activity in the anterior cingulate cortex (ACC) and orbitofrontal cortex (OFC) of freely moving rats, as well as on operant set-shifting performance. Neural activity was recorded in a home cage environment during a 30 min baseline and for 2 hrs post-injection. FG7142 induced a dose-dependent inhibition of single unit firing rate and a dose-dependent increase in gamma-band oscillatory power in both ACC and OFC. Gamma-range oscillations have been linked to many cognitive processes, while deficits in frontal cortex gamma-band synchrony may contribute to cognitive control impairments in schizophrenia. FG7142 treatment also produced a trend toward enhancing cognitive set-shifting ability, by reducing the total trials to reach criterion and the total errors in the operant task, suggesting increased vigilance. These data indicate that acute FG7142 administration may induce changes in PFC single-unit activity and LFP oscillations that are relevant to optimal cognitive performance.

2. THE ANXIOLYTIC EFFECTS OF CANNABIDIOL IN A POST-TRAUMATIC STRESS MODEL ARE MEDIATED BY 5HT1A RECEPTORS. Campos, AC; Ferreira, FR; Guimaraes, FS. Dept of Pharmacology; School of Medicine of Ribeiro Preto- University of Sao Paulo. Introduction: Posttraumatic stress disorder (PTSD) is an incapacitating chronic syndrome that reflects cognitive, emotional, and physiological processing changes that follow an initial reaction to a traumatic experience. Cannabidiol (CBD), a non-psychotomimetic constituent of Cannabis sativa plant, produces anxiolytic-like effects in animal models and humans. Although its mechanisms of action are not clear, CBD could facilitate the neurotransmission mediated by 5HT1A and CB1 receptors (Campos and Guimaraes, 2008). Although both receptors have been related to anxiety, the effects of CBD in PTSD models have not yet been investigated. Thus, the aim of this study was to investigate if CBD could attenuate behavioral and plastic consequences promoted by a stressful experience in rats submitted to a PTSD animal model. Methods: Male Wistar rats (210-230g) were placed into a box where they were exposed to a toy or a live cat for ten minutes before being replaced in their home cages. Experiment 1: After the exposure the rats started receiving daily systemic injections of vehicle (V), paroxetine (10mg/kg) or CBD (5mg/Kg) during seven days. Twenty four h after the last injection the rats were submitted to the Elevated plus maze test (EPM). Experiment 2: Similar to the first experiment except that the animals received injections of WAY100635 (1mg/Kg), a 5HT1A antagonist, 10 min before CBD injections. After the experiments brain tissues were removed, hippocampus, frontal cortex, amygdaloid complex, and dorsal periaqueductal gray were isolated and processed for posterior RNAm (genes: CB1 and 5HT1A receptors and synaptophysin- SYP) and BDNF analysis. Results: Exposure to the live cat decreased the number of entries and the time spent in the open arms of the EPM. This effect was reversed by CBD and paroxetine. Pretreatment with

WAY100635 prevented CBD effects. Predator exposure decreased 5HT1A and SYP gene expression in frontal cortex. No treatment was able to prevent these effects. Also, CB1 RNAm expression was up-regulated seven days after cat exposure. Sub-chronic treatment with paroxetine, but not of CBD, prevented this up-regulation in the amygdaloid complex. No change in BDNF expression was found in any of analyzed brain structures (hippocampus, frontal cortex, amygdaloid complex and dorsal periaqueductal gray matter). Conclusions: Predator exposure induces long-lasting (7 days) anxiogenic effects and down-regulation of 5HT1A and SYP mRNA expression in frontal cortex but up-regulation of CB1 RNAm expression in the amygdaloid complex. CBD and paroxetine sub-chronic treatment are able to prevent the behavioral but not the mRNA expression changes of SYP, 5HT1A in the frontal cortex. CBD behavioral effects in this model are probably mediated by 5HT1A receptors. Financial support: FAPESP, CNPq.

3. HIGH INTENSITY ACUTE STRESS INDUCES UNIQUE PATTERNS OF ASSOCIATIVE AND NON-ASSOCIATIVE FEAR MEMORY BEHAVIOR IN AN ANIMAL MODEL OF POSTTTRAUMATIC STRESS DISORDER. Corley, M.J.; Takahashi, L.K. Psychology Dept. Univ. of Hawaii, Honolulu, HI, 96822, USA. Two experiments were conducted to test the hypothesis that exposing rats to acute high-intensity footshock stress will induce different patterns of conditioned and sensitized fear behavior in fear memory extinction and habituation tests. In Experiment 1, rats were exposed to acute footshock stress (no shock control, 0.4 mA, or 0.8 mA) followed immediately by auditory fear conditioning training involving the pairing of auditory clicks (CS) with a cloth containing predator odor (US). In the next 5 days of extinction testing, fear behavior measured during presentation of auditory CS in a runway apparatus containing a small hide box showed the 0.8 mA footshock conditioned group exhibited significantly higher levels of freezing and vigilant head out behavior from the hide box than 0.4 mA conditioned and no shock conditioned groups. Thus, high levels of stress induced an associative emotional memory that becomes resistant to extinction. In Experiment 2, rats received footshock as previously described and tested 5 days in the runway apparatus by presentation of novel auditory clicks. Rats in the 0.8 mA group exhibited high levels of freezing and hiding on test day 1 followed by high levels of line crossing or hyperactivity behavior over the remaining test days. Thus, exposure to high intensity stress induces a non-associative sensitized state characterized by hyperactivity behavior. Our novel experiments, highlighting some prominent hallmarks of PTSD - resistance to extinguish fear, hypervigilance, hyperarousal, have relevance in understanding the pathophysiological correlates of the disorder.

4. INTRA-AMYGDALA INFUSION OF A GROUP I AGONIST MODULATES PASSIVE AND ACTIVE DEFENSIVE BEHAVIORS IN A SEX SPECIFIC MANNER De Jess-Burgos M1;Cruz-Santana Y2; and Prez-Acevedo NL1. 1School of Medicine, University of Puerto Rico, Medical Sciences Campus and 2University of Puerto Rico, Cayey campus. Rats display passive and active adaptive defensive behaviors, including, freezing, behavior inhibition (BI) and risk assessment behaviors (RABs) in response to predator odors. These defensive responses are sensitive to anxiolytic as well as panicolytic drugs. Since group I metabotropic glutamate receptors (mGluRs) have been related to defensive behaviors, the present study was undertaken to assess the role of group I mGluRs within the basolateral amygdala (BLA), a region involved in anxiety during cat odor exposure. Ovariectomized females with (OVX+EB) and without (OVX) estradiol replacement and male rats were used to assess whether sex and/or estradiol treatment affect defensive behaviors. We hypothesized that activation of group I mGluRs will increase anxiety in a sex specific manner. After intra-BLA infusion of (S)-3,5-Dihydroxyphenylglycine (DHPG), a group I mGluRs agonist, defensive, as well as, non-defensive behaviors (such as grooming, rearing and locomotion) were measured. In OVX+EB and OVX females ($p=0.05$ and $p=0.002$, respectively), but not in male rats, DHPG increased the time spent in BI. In males, DHPG increased the number of RABs whereas, it decreased in females ($p=0.021$ and $p=0.014$, respectively). DHPG did not alter general locomotion and other non-defensive behaviors ($p>0.05$). Taken together, BLA activation of group I mGluRs by DHPG increases adaptive passive and active defensive responses, this anxiogenic-like response occurs differentially on female and male rats. This study was partially supported by NIH-EARDA (1G11H046326), MBRS-RISE (GM61) and NSF (DBI-0932955).

5. EFFECTS OF AN OREXIN 1 RECEPTOR ANTAGONIST ON INVERSE BENZODIAZEPINE AGONIST-INDUCED PANICOGENIC-RELATED RESPONSES AND CELLULAR RESPONSES IN THE BRAIN Federici L.1; Fitz S.D.2; Hammes N.4; Early M.4; Dietrich A.2; Lowry C.3; Samuels B.C.1; Shekhar A1.; Johnson P.L.1 1. Stark Neuroscience Research Institute, 2. Dept. of Psychiatry, Indiana University School of Medicine, Indianapolis, IN; 3. Dept. of Integrative Physiology and Center for Neuroscience, University of Colorado, Boulder, CO; 4. University of Notre Dame, South Bend, IN. Although the hypothalamic orexin (ORX) system is known to regulate appetitive behaviors and promote wakefulness and arousal [see review (Sakurai, 2007)], this system may also be important in adaptive and pathological anxiety/stress responses. For instance, increasing ORX concentrations in the brain of rats increases anxiety-like behavior (Suzuki et al., 2005). Furthermore, in a Nature Medicine article (Johnson et al., 2010), we determined that the ORX system was hyperactive in an established rat model of panic

vulnerability, and pharmacologically blocking ORX1 receptors or genetically silencing the ORX system blocked provoked anxiety-associated behavior and panic-associated cardiovascular responses. Here we show that systemic injections of two different anxiogenic drugs [i.e., FG-7142 (partial agonist at the benzodiazepine allosteric site on the GABAA receptor, 7.5mg/kg ip) or caffeine (nonselective competitive adenosine receptor antagonist, 50mg/kg ip)] increased cellular responses (i.e., c-Fos induction) in orexin neurons in the dorsomedial/perifornical, but not lateral, hypothalamus. We then determined that systemically blocking the ORX1 receptor (SB334867, 30mg/kg, ip) 30 min prior to FG-7142 attenuated both: 1) anxiety behavior in the open field and social interaction test and; 2) FG-7142-induced increases in cellular responses (i.e., c-Fos induction) in the subregions of the extended amygdala; the dorsal and ventral periaqueductal gray; and rostroventrolateral medulla. Additionally, we are determining the effects of the ORX1 receptor antagonist on panic associated cardiovascular responses to FG-7142. Overall the data here suggest that ORX antagonists constitute a potential novel treatment strategy for anxiety disorders and support the hypothesis that a hyperactive orexin system leads to pathological anxiety. Acknowledgement: IUPUI University Fellowship to LF, Indiana CTSI Project Development Award, NIH Student LRP, and National Alliance for Schizophrenia and Depression Young Investigators Award to PLJ; Indiana CTSI (UL1 RR025761) undergraduate research fellowships to MCE; RO1 MH52619 to AS and RO1 MH065702 to AS and CAL.

6. THE PROTECTIVE EFFECTS OF EXERCISE ON CHRONIC STRESS-INDUCED NEUROTOXICITY. Gerecke, K.M., Kolobova, A. and Allen, S. Rhodes College, Dept. of Psychology and Neuroscience Program, Memphis, TN. Chronic restraint stress has been shown to cause deleterious effects in the brain through chronic elevation of glucocorticoids (GCs), which leave neurons vulnerable to other toxic insults such as oxidative stress and inflammation. This process has been implicated in all neurodegenerative events, including Parkinson's and Alzheimer's diseases. Exercise has been shown to protect against this toxicity, thus we investigated the neuroprotective effects of exercise in a model of chronic stress. In the current study, mice were divided into two housing groups: Standard housing and exercise. Half of the animals in each housing condition were chronically stressed for 2 hours per day for 14 consecutive days. To determine temporal expression, animals were sacrificed 1 and 24 hours after the final stress and tissue was harvested for Western Blot (WB) analysis for expression of the apoptotic protein Bax in the hippocampus and cortex. Our data suggests that stress upregulates Bax and exercise downregulates the expression of this factor in the mouse cortex at 1 hour following the last stress, and that this expression remains elevated for 24 hours. This suggests that chronic stress can induce apoptosis in the cortex that is sustained, and that exercise can decrease neuronal death in this brain region. Thus, chronic stress induces factors related to apoptosis in the brains of chronically stressed mice and exercise provides protection against these deleterious effects.

7. TOXOPLASMA INDUCED OVERLAP IN DEFENSIVE AND REPRODUCTIVE NETWORKS IN THE MEDIAL AMYGDALA. Hari Dass, S.;Vyas, A.School Of Biological Sciences. Nanyang Technological University.Singapore 635771. Toxoplasma gondii, a parasitic protozoan, causes its rodent host to lose fear and even gain an attraction towards its natural feline predator. The medial amygdalar nuclei is important in the processing of innate fear . Another innate response with a very different valency, sexual attraction, is also processed here. Defensive cues recruit the MEApv and VMHdm while reproductive cues recruit the, neighboring, MEApd and VMHvl. Argenine vasopressin (AVP) positive neurons in bed nucleli stria terminalis are involved in social affiliation. We used AVP to visualise reproductive neurons in the medial amygdala. We hypothesised that there is an infection induced overlap between the defensive and reproductive networks. In control animals in the MEApd 39.7 4.4% of the neurons activated post cat odor were AVP positive. As per our hypothesis, in infected animals this was boosted to 56.3 1.7%. There was no difference in infection induced recruitment of AVP neurons in the MEApv (infected mean= 31 5.3 %, control mean = 26.2 5.8 %). Interestingly MEApd is the amygdala region implicated in sexual processes. The medial amygdalar nuclei is rich in testosterone binding androgen receptors (AR). We hypothesis that testosterone via these AR could be sculpting the neural overlap. In accordance with this we found that testicular testosterone was almost doubled in infected males are compared to their control counterparts. In conclusion we have shown that Toxoplasma infection causes an overlap between the defensive and reproductive networks in the medial amygdala. Preliminary results indicate that testosterone might be the mediator.

8. THE PARADOX EFFECT OF NOMIFENSINE ON ACTIVE AVOIDANCE LEARNING IN AN ANIMAL MODEL OF BEHAVIORAL INHIBITION. X. Jiao1*; K.C. H. Pang1, 2; K.D. Beck1,2; R.J. Servatius1,2. 1SMBI and 2Neurobehavioral Research Laboratory, DVA-NJHCS, EO, NJ Comorbid anxiety and depression often complicate the treatment of psychiatric disorders. The Wistar-Kyoto (WKY) rat has long been studied as model of depression. Behavioural and pharmacological evidence suggests that chronic antidepressant treatment reduced the floating time in a forced swim test and increased ambulation in an open field test in WKY rats. We and other laboratories reported that WKY rats demonstrate trait behavioral inhibition (BI), a risk factor for anxiety disorders. Given that avoidance is the core symptom of all anxiety disorders, we recently examined WKY rats in an active

avoidance procedure and found that they acquire avoidance more rapidly and extinguish avoidant responding more slowly than Sprague-Dawley (SD) rats. Thus, WKY rats may be a model of comorbid anxiety and depression. An important question is whether treatment for depressive symptoms would improve the development of anxiety-like behaviors. The present study evaluated the effects of an antidepressant drug on active avoidance learning in WKY and SD rats. Chronic treatment of nomifensine (10mg/kg/day) was administered prior to and during avoidance training. We found that nomifensine enhanced avoidance acquisition and impaired extinction learning in WKY but not SD rats. In addition, nomifensine treatment increased acoustic startle response only in WKY rats, a sign of elevated anxiety. The results from current and previous studies suggest that 1) WKY rats may be a good model of anxiety behavior, and 2) antidepressants may exacerbate the anxiety-like behavior in a model of depression-anxiety comorbidity. The current data provide valuable information to better understand the neurobiological mechanism underlying comorbid anxiety and depression in WKY rats. Supported by DVA Medical Research funds, SMBI and NIH grants.

9. OREXIN-A INJECTIONS INTO THE BNST INDUCES ANXIETY-LIKE BEHAVIOR VIA INTERACTIONS WITH GLUTAMATERGIC RECEPTORS IN THE RAT. Lungwitz, E.1; Johnson, P.2; Harvey, B.3; Deal, R.1; Dietrich, A.1; Minick, P.1; Shekhar, A.2; Truitt, W.1,2. 1-Dept. of Anatomy, 2-Dept. of Psychiatry, 3-Project SEED, IU School of Medicine, Indianapolis, IN. Panic response is an anxiety reaction characterized by sudden onset of autonomic activation. We demonstrate that the bed nucleus of stria terminalis (BNST) is a pivotal region regulating the anxiety component of a panic response. Release of the neuropeptide orexin (ORX) in the BNST is critical for the anxiety-like behavioral component of sodium lactate evoked panic responses in rats. Behavior responses to the sodium-lactate challenge were blocked with intra-BNST pre-infusions of the ORX 1 receptor antagonist SB3344867 but not vehicle. Anxiety-like behavior was induced by unilateral injections of OrxA into the BNST compared to baseline or vehicle injected rats. We determined if the anxiety-like behavior is mediated by the ORX1r by unilaterally injecting the ORX1r antagonist or vehicle into the BNST of rats 10 min prior to a BNST injection of OrxA and assessing anxiety-like behavior via the social interaction test 30 min later. Orexin enhances glutamatergic activity, so to determine if intra-BNST OrxA-induced anxiety involves a glutamatergic mechanism, we infused NMDA receptor antagonist (AP5), AMPA receptor antagonists (CNQX or DNQX) or vehicle into the BNST 10 minutes prior to OrxA infusion. NMDA antagonist blocked the OrxA-induced anxiety, while AMPA antagonists produced a partial response. We demonstrate that OrxA in the BNST increases phosphorylation of NMDA NR1 subunits, suggesting a mechanism by which OrxA and glutamate interact in the BNST to induce anxiety-like responses. Supported by RO1s MH52619 and MH065702.

10. AN INBRED MOUSE MODEL OF IMPAIRED FEAR EXTINCTION: Martin, K.; Lederle, L.; and Holmes, A. Section on Behavioral Science and Genetics, Laboratory for Integrative Neuroscience, National Institute on Alcohol Abuse and Alcoholism, Rockville, MD. CORTICO-AMYGDALA DENDRITIC DYSMORPHOLOGY. Impaired fear extinction is characteristic of Posttraumatic Stress Disorder. We identified an inbred mouse strain, 129S1/SvImJ (S1), with impaired Pavlovian fear extinction (compared to C57BL/6J (B6)), and associated functional abnormalities in a prefrontal-amygdala circuit mediating extinction. The prelimbic (PL), infralimbic (IL), and basolateral amygdala (BLA) regions have been implicated in the processes of fear extinction. Dendritic morphology was assessed in B6 and S1 mice. Average length of apical dendrites was significantly greater in the BLA of S1 than B6, with the most marked differences distal from the soma. Strains did not differ on any measured parameter in either IL or PL. These changes in dendritic distribution may underlie the poor extinction in the S1 mice. EFFECTS OF MEMORY REACTIVATION. Fear memories enter a labile state after reactivation a process termed reconsolidation. A reconsolidation-like process has been hypothesized to explain the recent finding that reactivating fear memories prior to extinction training facilitates long-term extinction in rats (Monfils et al 2009) and humans (Schiller et al 2010). Whether this process facilitates fear extinction in clinical populations or animal models of impaired extinction has not yet been tested. We tested whether fear memory reactivation prior to extinction training produced improvements and rescue of long-term extinction in B6 and S1, respectively, in 3 experimental conditions that typically cause a reemergence of fear: spontaneous recovery, reinstatement, and renewal. Results indicated that, in mice, fear reactivation prior to extinction training does not prevent reemergence of fear. These results add to other recent studies suggesting that the effect of reactivation on extinction is complex and influenced by various procedural variables.

11. EVIDENCE FOR A LACK OF PHASIC INHIBITORY PROPERTIES OF HABITUATED STRESSORS ON HPA AXIS RESPONSES IN RATS. Masini, C.V.*; Day, H.E.W.; Gray, T.; Crema, L.M.; Nyhuis, T.J.; Babb, J.A.; Campeau, S. Dept. of Psychology and Neuroscience. University of Colorado, Boulder, CO 80309 USA This experiment tested the hypothesis that habituation of the hypothalamo-pituitary-adrenocortical (HPA) axis responses to repeated stressor exposures is produced by phasic inhibitory influence on the neural circuitry that normally drives the paraventricular nucleus of the hypothalamus and subsequently the adrenocortical hormone response to

psychological stress. Such a process would be expected to lower acute HPA axis responses to a novel stressor when experienced concurrently with a habituated stressor. Rats were exposed to restraint (n=35) or no stress (n=31) conditions for 14 consecutive days. On the 15th day different groups of rats were exposed to the control condition (no stress), or 30-min restraint, loud noise, or restraint and loud noise combined. Blood was taken and assayed for ACTH and corticosterone, as indices of HPA axis responses. As predicted, the rats that received the same (homotypic) stressor repeatedly and again on the test day displayed low levels of ACTH and corticosterone, similar to the control conditions (i.e., HPA axis response habituation). All rats that received a single novel stressor on the test day, regardless of prior stress history, exhibited high levels of ACTH and corticosterone. The rats that received two novel stressors also displayed high levels of ACTH and corticosterone. Importantly, when a novel stressor was superimposed with a habituated stressor on the test day, no reduction of HPA axis response was observed when compared to rats receiving both superimposed stressors for the first time. These data suggest that habituation of the adrenocortical hormone response to psychological stressors is not mediated by phasic inhibition of effector systems.

12. THE COMBINED EFFECTS OF STRESS AND ENRICHMENT ON POST-PARTUM BEHAVIOUR IN THE RAT. Pichette, N.; Falicki, A.; Mileva, G.; Rees, S.; Bielajew, C. School of Psychology. The University of Ottawa, Ottawa, ON K1N 6N5 Canada. Studies using animal models have shown that gestational stress significantly affects offspring development and maternal behaviour. For example, gestational stress has been shown to elevate maternal and offspring anxiety, decrease maternal care of offspring and introduce cognitive impairments in offspring. Enrichment studies, in contrast, have shown that environmental and social stimulation are generally associated with a decrease in the adverse effects of stress and mood-related disorders in animals. The purpose of this study was to investigate whether prenatal enrichment would reduce the effects of gestational stress in mother rats during the late post-partum period. Groups included mothers that were either stressed or not stressed, and environmentally enriched or not enriched. During the late post-partum period, behavioural tests of anxiety and depression were conducted using the elevated plus maze and forced swim tests. Maternal behaviour was also observed. No group differences were found between stressed rats and enriched rats on the elevated plus maze and forced swim tests. However, mothers who were stressed showed reduced hovering and licking maternal behaviours compared to mothers who were not stressed, independent of enrichment. Furthermore, enrichment, with or without a stress component, was shown to play a critical factor in reducing anxiety- and depression-related behaviours in the elevated plus maze and forced swim tests. Our data indicate that both the physical and social aspects of the environment significantly influence post-partum activity, more specifically anxiety and depression-related behaviours in the mother rat. It would be important to determine at what point during the gestation period environmental enrichment is critical in generating these effects.

13. EFFECT OF STRESSOR CHALLENGE DURATION ON THE BEHAVIORAL AND NEUROENDOCRINE EXPRESSION OF STRESS RESPONSE HABITUATION. Ramsey, R.E.; Spencer, R.L. Department of Psychology & Neuroscience. University of Colorado at Boulder, Boulder, CO 80303 USA. The predisposing and exacerbating influences of stress on physiological and psychological disorders are often difficult to analyze because an individual's perception of a stressor and subsequent responses differ based on prior experience. While habituation develops to a repeated, predicable psychological stressor, manipulating certain parameters of the stress experience may lead to disruption of a stressors predictability and subsequent dishabituation of the stress response. In this experiment, we investigated whether the neuroendocrine and behavioral responses (used as indicators of activity of the Limbic-Hypothalamic-Pituitary-Adrenal-, or LHPA-, axis) to a psychological stressor (restraint) differ when the duration of the stressor given on the test day violates expectations based on prior stress experience. Rats experienced 10 minutes of daily restraint on Days 1-4 followed by either the same duration (10 min) or a longer duration (30 min) of restraint on Day 5. Video recordings were collected for each restraint episode (Days 1-5) and trunk blood and tissue samples (brains for immediate early gene expression analysis [c-fos mRNA]) were collected following restraint on Day 5. Struggling behavior was manually scored as active attempts to escape the restraint device. Rats who experienced the same duration of repeated restraint showed a significant decrease of plasma corticosterone (CORT) compared to the 10 min acute restraint group (habituation). In addition, these rats showed decreased active struggling over repeated restraint trials. Conversely, the rats who experienced a longer duration of restraint on Day 5 showed an increased CORT response (dishabituation). These rats showed a habituated behavioral response during the first 10 min of restraint, however struggling behavior was reinstated once the duration of restraint exceeded the expected duration (with a peak at 12 min). This peak in struggling behavior did not occur during 30 min acute restraint, indicating that the effect was related to memory of previous restraint experience and not simply due to a longer duration of restraint. In addition, the animals showed decreased c-fos expression in the paraventricular nucleus (PVN) and lateral septum (LS), but not the medial prefrontal cortex (mPFC) in response to the increased stressor duration. In conclusion, habituation of the neuroendocrine and behavioral stress responses occurs when the duration of the stressor matches previous experience, while dishabituation of the stress response is triggered (with remarkable temporal precision) by an unpredicted increase in stress duration. Future investigations will determine

whether the mPFC is involved in detecting mismatch between prior experience and current stress conditions, and what cellular mechanisms are necessary for its ability to modulate the physiological and behavioral stress response.

14. IMPACT OF UNPREDICTABLE CHRONIC SOCIAL DEFEAT ON PALATABLE FEEDING AND BEHAVIOURAL MARKERS OF DEPRESSION AND ANXIETY. MacKay, J.C.; Patterson, Z.R.; James, J.; Kent, P.; Abizaid, A.; Merali, Z. The Resident/Intruder paradigm is an ethologically relevant animal model of social stress which is widely used to assess the link between stress and future pathologies. The purpose of this study was to investigate the effects chronic unpredictable social defeat on palatable feeding, anxiety- and depressive-like behaviour as measured by the open field (OF), elevated plus maze (EPM) and forced swim test (FST). Caloric intake, body weight gain, glucose tolerance, and adipocity were also assessed. A 2x2 mixed factorial design was used consisting of: defeat or no defeat x palatable food or no palatable food. Male Long-Evans intruder rats were exposed to a different resident rat for a total of 60 min/daily over 21 days. Rats were allowed to interact for 10 min and then separated by a holed plastic divider for the remaining time. Control rats received daily handling. All rats had ad libitum access to either chow or both chow and the palatable food (TD.08811, Harlan Laboratories). Food intake was measured daily. All rats were tested 24 hrs after the final defeat session in the OF and EPM then in the FST 24 hrs later. After testing, rats were given a 3 week recovery period then FST was repeated and glucose tolerance was assessed. Among defeated rats, total daily calorie consumption was decreased during the stress period and increased during the recovery period. Rats exposed to daily social defeat showed increased immobility in the FST. Compared to chow fed rats, rats fed palatable food showed glucose intolerance. Furthermore, defeated rats with access to palatable food showed greater glucose intolerance and an increased preference for the palatable food compared to controls. No significant results were found in the OF, EPM and adiposity. Results suggest that unpredictable chronic social defeat results in depressive symptomatology and an increased preference for palatable food.

15. THE IMPACT OF JUVENILE STRESS ON PALATABLE FEEDING AND BEHAVIOURAL MARKERS OF DEPRESSION AND ANXIETY IN JUVENILIY. MacKay, J.C; James, J; Cayer, C; Kent, P; Merali, Z. Inst. of Mental Health and Dept. of Psychology. The University of Ottawa, Ottawa ON K1N 6N5 Canada. Approximately 15-20% of juveniles will encounter some form of trauma, with an increased risk of developing a stress-related psychopathology. Stress has also been shown to promote palatable feeding. The current study utilized exposure to juvenile stress to investigate early consequences of juvenile stress on anxiety- and depressive-like behaviour as measured by the open field (OF), elevated plus maze (EPM) and forced swim test (FST) and sucrose preference. Furthermore, the effects of stress on caloric intake and preference for a palatable food were assessed. A 2x2 mixed factorial design was used consisting of: stress or no stress x palatable food or no palatable food. All rats had ad libitum access to chow or chow with 2 hr access to a palatable food (TD.08811, Harlan Laboratories). Based on a modified procedure used by Jacobson-Pick and Richter-Levin (2010), male Wistar rats were exposed to 3 days of consecutive stress on postnatal days (PD) 27-29, while control rats received daily handling. Rats were then tested between PD30-37. Behavioural testing was in the following order: open field and elvated plus maze, then forced swim test. Sucrose preference was assessed on PD32-38. Among all rats, no significant differences in daily chow consumption were found, while in rats with access to palatable food, stressed rats consumed less palatable food during stress exposure. Compared to controls, rats exposed to juvenile stress showed increased immobility in the FST. Stressed chow fed rats showed increased anxiety like behaviour and anhedonia on the OF, EPM and sucrose preference test; effects that were attenuated with consumption of the palatable food. These findings suggest that exposure to juvenile stress results in a behavioural pattern suggestive of depressive and anxiety symptomatology. While consumption of palatable food may be protective against stress-related psychopathology.

16. ANXIOGENIC-LIKE EFFECT OF CANNABIDIOL INJECTED INTO THE RAT PRELIMBIC PREFRONTAL CORTEX. Fogaca, M.V., Campos, A.C., Guimaraes, F.S. Department of Pharmacology, Faculty of Medicine of Ribeirao Preto, University of Sao Paulo (FMRP-USP), Ribeirao Preto-SP, Brazil. E-mail: manoelafogaca@usp.br. Cannabidiol (CBD), a major non-psychotomimetic cannabinoid present in the Cannabis sativa plant, induces anxiolytic-like effects after systemic injections. Similar effects have also been shown in the bed nucleus of the stria terminalis and the dorsolateral periaqueductal gray. The prelimbic prefrontal cortex (PL) is another brain region that has been related to defensive responses. Therefore, the aim of the present study was to investigate if intra-PL injection of cannabidiol would also produce anxiolytic-like effects in rats. Four groups of male Wistar rats (7 to 11/group) with cannulae bilaterally aimed at the PL received microinjections of vehicle (grape seed oil) or CBD (15, 30, 60 nmol). Five minutes later the animals were submitted to the elevated plus maze (EPM) where the percentage of entries (Pea) and time spent in open arms (Pta) and the number of enclosed arm entries (Eae) were recorded for 5 min using the Anymaze software. The former two parameters are proposed to inversely reflect anxiety whereas the latter is related to general exploratory activity. CBD induced an anxiogenic-like effect in the intermediate dose (30 nmol), represented by a decrease in Pea and Pta (Pea: Vehicle: 40.18+/-4.35, CBD: 19.70+/-4.89, F3,34=3.045,

p=0.042. Pta: Vehicle: 24.55+/-5.41, CBD: 6.90+/-2.25, F3,34=3.260, p=0.033, ANOVA). No effects were found in the number of enclosed arm entries (Eae: F3,34=0.256, p=0.856). These results suggest that the effects of cannabidiol in emotional responses depend on the brain region that the substance is injected. The next step of this study is to evaluate if infra-limbic microinjections of CBD is able to produce opposite effects. Financial support. CAPES, CNPq, FAPESP

17. INCREASED LONG-TERM FEAR MEMORY IN MICE LACKING TIP39 SIGNALING: A NEW MODEL FOR THE STUDY OF FEAR-RELATED PSYCHOPATHOLOGY? Coutellier, L.; Usdin, T.B. Section on Fundamental Neuroscience, National Institute of Mental Health, National Institutes of Health, Bethesda, MD, USA. Fear-related psychopathologies are characterized by impaired memory extinction for a fear-provoking event, leading to trauma-induced changes in anxiety- and depressive-like behaviors. To improve therapeutic efficacy for these disorders, it is essential to understand the neural basis of fear memory regulation. Based on previous neuroanatomical and behavioral data we suggest that the neuropeptide tuberoinfundibular peptide of 39 residues (TIP39) and its receptor, the parathyroid hormone 2 receptor (PTH2-R), could modulate the pathways involved in fear memory and could contribute to the expression of long-term consequences of aversive experiences. To explore this possibility, TIP39 knockout (TIP39-KO), PTH2-R knockout (PTH2-R-KO) and wildtype (WT) mice were exposed to an aversive event (foot shock: 1.5mA for 2 seconds). Six or 14 days after the shock, we assessed their long-term fear memory by re-exposing them to the shock context and evaluated signs of anxiety- and depressive-like behaviors employing the elevated-zero maze test, the open-field test, the light/dark box test and the forced-swim test. We observed no difference in fear recall or anxiety- and depression-like behavior between mice with or without TIP39 signaling one week after the shock. Two weeks after the aversive event, TIP39-KO and PTH2-R-KO mice displayed enhanced fear-recall, as demonstrated by a higher level of freezing than WT. This was associated with increased anxiety- and depression-like behaviors. Altogether, our findings suggest that TIP39 signaling may modulate fear regulatory pathways, especially those controlling long-term fear recall. The present data support the idea that mice lacking TIP39 or its receptor, the PTH2-R, could be used as a tool to unravel the mechanisms underlying the long-term consequences of traumatic experiences and open a new avenue to the study of fear-related psychopathologies.

18. NOVELTY-EVOKED ACTIVITY IN THE OPEN FIELD PREDICTS SUSCEPTIBILITY TO HELPLESS BEHAVIOR. Padilla, E.; Shumake, J.; Barrett, D.; Holmes, G.; Sheridan, E.; Auchter, A.; Rothardt, A.; Gonzalez-Lima, F. Institute for Neuroscience, Department of Psychology and Pharmacology, University of Texas at Austin, 1 University Station A8000, Austin, TX 78712, USA. Learned helplessness in animals has been used to model disorders such as depression and post-traumatic stress disorder (PTSD), but there is a lack of knowledge concerning which individual behavioral characteristics at baseline can predict helpless behavior after exposure to inescapable stress. The first aim of this study was to determine behavioral predictors of helplessness using the novel and familiar open-field tests, sucrose consumption, and passive harm-avoidance tasks before learned helplessness training and testing. Individual differences in physiologic responses to restraint stress were also assessed. A cluster analysis of escape latencies from helplessness testing supported the division of the sample population of Holtzman rats into approximately 50% helpless and 50% non-helpless. Linear regression analyses further revealed that increased reactivity to the novel environment, but not general activity or habituation, predicted susceptibility to learned helplessness. During restraint stress there were no mean differences in heart rate, heart rate variability, and plasma corticosterone between helpless and non-helpless rats; however, a lower heart rate during stress was associated with higher activity levels during exploration. Our most important finding was that by using an innocuous screening tool such as the novel and familiar open-field tests, it was possible to identify subjects that were susceptible to learned helplessness.

19. CORTICOSTERONE MEDIATES RISK ASSESSMENT BEHAVIORS IN THE ANTERIOR CINGULATE CORTEX OF RATS. Reis, F. M. C. V. ; Albrechet-Souza, L. ; Franci, C. R. ; Brandao, M. L. . Laboratorio de Neuropsicofarmacologia and Instituto de Neurociencias & Comportamento - INeC, FFCLRP, Departamento de Fisiologia FMRP, University of Sao Paulo - Ribeirao Preto, SP, Brazil. Acute stressors activate the hypothalamic-pituitary-adrenal axis (HPA) producing a rapid increase in plasma corticosterone levels with impact on specific neuronal populations resulting in unique downstream effects. In this respect, the medial prefrontal cortex exerts a marked influence in defensive behaviors. The anterior cingulate cortex, area 1 (Cg1), is especially rich in glucocorticoid receptors. We aimed to determine the extent to which HPA is activated by different degrees of social isolation and whether the levels of plasma corticosterone correlate with the exploratory categories exhibited by rats in the elevated plus maze test (EPM). Male Wistar rats were subjected to different periods of isolation (30 min, 2 h, 24 h and 7 days) and then exposed to the EPM for 5 minutes. Blood samples were collected immediately after completion of the tests. Thirty minutes of isolation produced the greatest increase in plasma corticosterone, which positively correlates with risk assessment behaviors. Long periods of isolation (24 h, 7 days) increased the aversion to the maze even though the plasma corticosterone concentrations have returned to baseline levels. Corticosterone

injected bilaterally into the Cg1 (5ng/side) selectively increased the risk assessment behaviors. These findings suggest the involvement of corticosterone receptors in the Cg1 area in the cognitive and hormonal correlates of the exploratory behavior in the EPM. Furthermore, our results suggest that only the initial cognitive component of the aversive states correlates with HPA activation while the persistence of social isolation induces an emotional shift reflected by a behavioral defense reaction dissociable from hormonal changes. Financial support: CAPES.

20. ABUSE LIABILITY ASSESSMENT OF CINNAMOMUM CASSIA. Shelton, S.; Pope, S.; Birkett, M. Department of Psychology. Northern Arizona University, Flagstaff, AZ 86011 USA. Many compounds that bind to the GABAA receptor possess significant abuse potential (e.g. alcohol, benzodiazepines, barbiturates). Preclinical research has suggested that cinnamon (Cinnamomum cassia) may possess active compounds that bind to the GABAA receptor. Although not currently considered a drug of abuse, we investigated the abuse liability of cinnamon through subjective response questionnaires measuring drug-like effects. This approach is commonly used to screen compounds for potential abuse liability. Forty-four healthy adult participants completed the short version of the Addiction Research Center Inventory (ARCI) 45 minutes after consuming cinnamon (1000mg or 3000mg) or inactive (placebo) capsules. There was no significant difference in euphoria, sedation, or stimulant subscale scores of the ARCI between control and cinnamon groups. Interestingly, differences between groups approached significance on the dysphoria/psychotmimetic subscale ($F=2.693$, $p=.059$), with 1000mg cinnamon associated with increased scores on the dysphoia/psychotomimetic subscale. The results of this pilot study suggest the subjective effects profile and abuse liability of cinnamon is inconsistent with GABAergic drugs of abuse.

21. EFFECTS OF CINNAMON ON PHYSIOLOGICAL RESPONSE TO A COGNITIVE EMOTIONAL STRESSOR. Tebbe, D.; Shelstad, T.; Gilbert, A.; Birkett, M. Department of Psychology. Northern Arizona University. Flagstaff, AZ 86011 USA. The stress response is associated with activation of the sympathetic nervous system and physiological arousal. Physiological changes in response to stress response include increased heart rate, electrodermal response, and blood pressure. Initial research has indicated that cinnamon (Cinnamomum cassia) may bind to GABAA receptors, important inhibitory sites of action within the nervous system. Preclinical research has suggested that cinnamon is effective in producing anxiolytic-like behavioral effects in rodent models exposed to a stressor (elevated plus maze), however the ability of cinnamon to reduce or attenuate physiological measures of stress has not been well described. We hypothesized that cinnamon would reduce physiological measures of stress response to a cognitive/emotional stressor. This stressor has been previously shown to increase physiological measures of stress response, and includes a challenging, timed arithmetic component and an anger recall component. The present pilot study evaluated the ability of cinnamon to reduce or attenuate physiological measures of the stress response in healthy male and female participants exposed to this cognitive/emotional stressor. Two doses of cinnamon (1000mg and 3000mg) failed to prevent increases in heart rate, electrodermal response or blood pressure in response to the stressor. Additional research is needed to more fully evaluate the potential anxiolytic physiological effects of cinnamon.

22. SUBJECTIVE EFFECTS OF CINNAMON ON RESPONSE TO A LABORATORY STRESSOR. Kontnik, M.; Marcus, D.; Birkett, M. Department of Psychology. Northern Arizona University, Flagstaff, AZ 86011 USA. Several plant-derived natural products contain active compounds that bind to GABAA receptors and enhance GABAergic neurotransmission. Some of these products have been shown to reduce measures of anxiety in samples of patients with Generalized Anxiety Disorder and to produce anxiolytic effects in response to standardized laboratory stressors. Cinnamon has been shown to produce anxiolytic-like behavioral effects in preclinical research, likely mediated through activity at GABAA receptors. We hypothesized that cinnamon would reduce subjective measures of anxiety in response to a standardized cognitive/emotional stressor. In the present study, healthy, adult participants received cinnamon (1000mg or 3000mg, p.o.) or control (placebo) capsules 45 minutes prior to exposure to a cognitive/emotional stressor. Participants completed the state portion of the State Trait Anxiety Inventory (STAI) and a visual analog scale (VAS) of anxiety before and after the stressor. There was a significant main effect of gender ($F=5.71$, $p=.007$) on change in VAS score, and treatment group ($F=2.836$, $p=.053$) on change in STAI score, however this research provided mixed support for subjective anxiolytic effects of cinnamon in response to a standardized laboratory stressor.

23. EXPERIMENTAL EVIDENCE THAT DEACTIVATION OF SECURITY-MOTIVATION AFTER EXPOSURE TO POTENTIAL THREAT IS DYSFUNCTIONAL IN OBSESSIVE-COMPULSIVE DISORDER (OCD). Hinds, A1.; Woody, E3.; Schmidt, L2.; Van Ameringen, M1.; Szechtman, H1. 1Dept. of Psychiatry & Behavioural Neurosciences, McMaster Univ., Hamilton, ON, Canada; 2Dept. of Psychology, Neurosci. & Behaviour, McMaster Univ., Hamilton, ON, Canada; 3Dept. of Psychology, Univ. of Waterloo, Waterloo, ON, Canada. The risk of uncertain, but grave potential dangers is proposed to be managed by an evolved special motivational system, termed the security motivation system (SMS) (Szechtman & Woody, 2004). Because it deals with potential (rather than

imminent) threats, security motivation is open-ended in that the environment cannot supply a physical signal for the absence of potential danger; the signal to terminate activated security motivation is generated through performance of precautionary behaviors. Obsessive-compulsive disorder (OCD) is proposed to result from dysfunction in the SMS mechanism by which engagement in security-related precautionary behavior normally terminates SMS activation; such failure would result in the repeated performance of precautionary behaviors such as washing and checking, behaviors which constitute the typical compulsive symptoms in OCD. We tested this prediction using a newly developed paradigm in which mild stimuli suggesting potential harm - contact with seemingly soiled diapers produced a state of activation which returned to baseline only after engagement in corrective hand washing. Participants with OCD (N=57) and normal controls (N=57) made contact with stimuli at varying levels of potential harm (none, very mild, mild). They were then permitted to wash for 30 sec and later for as long as desired; rhythmical sinus arrhythmia (RSA) and subjective ratings were measured to monitor the state of activation and deactivation produced by contact and by washing. Contact produced a similar degree of sensitivity to SMS activation in patients and controls. In contrast, although engagement in corrective behavior for 30 sec produced a deactivation of RSA to baseline in controls, it had no significant effect in patients. This result supports the hypothesis that OCD may stem from a lack of the normal ability to terminate activation of security motivation through engagement in security-related behavior.

24. COMPULSIVE CHECKING BEHAVIOR IN AN ANIMAL MODEL OF OBSESSIVE COMPULSIVE DISORDER. Thompson, B.S.; Greene-Collozi, E.; Andersen, S.L. Laboratory of Developmental Neuropharmacology. McLean Hospital/Harvard Medical School, 115 Mill Street, Belmont, MA 02478 USA. Our recently developed pharmacological animal model of obsessive-compulsive disorder (OCD) demonstrates that drug exposure during a sensitive period of development results in emergence of OCD-like phenotypes. Animals exposed to the tricyclic antidepressant clomipramine (15 mg/kg twice daily) between postnatal days 9 and 16 in the rat demonstrate behavioral and biochemical changes that are consistent with those observed in OCD. In this study, we tested whether early life clomipramine exposure would increase compulsive checking and if exposure to the selective serotonin reuptake inhibitor (SSRI) fluoxetine (10 mg/kg) would produce similar results. Fluoxetine is commonly used in the treatment of OCD in adults and children. Sprague-Dawley rat pups were administered vehicle, clomipramine or fluoxetine (i.p.) during postnatal days 9 and 16. Compulsive checking was tested in a large open space with novel objects located in specific locations in adulthood (P85). Subjects were given a priming dose of the D2 agonist quinpirole (0.5 mg/kg) 25 min prior to testing. Clomipramine and fluoxetine injected rats not only showed more anxious behaviors (e.g., elevated plus, marble burying) in comparison to the vehicles, but also an increased time spent and number of visits to objects placed within the open space. Stereotypical behaviors (grooming, sniffing, chewing and rearing) did not differ during the assessment periods. The results from this study suggest that developmental exposure to elevated levels of serotonin leads to permanent changes in behavior, including enhanced levels of anxiety, but also increased compulsive checking behavior.

25. THE NEUROBIOLOGY OF BENZODIAZEPINE ABUSE AND THE ROLE OF SOCIAL DEFEAT Lilian Doss, Derek van der Kooy. Institute of Medical Science, University of Toronto, Toronto, ON, Canada. Benzodiazepines have a high abuse liability in sub-populations with comorbid anxiety/depression and poly-drug users. However, benzodiazepine abuse remains an understudied area of research and the neural substrates mediating the rewarding effects of benzodiazepines remain undetermined. In order to investigate the rewarding effects of benzodiazepines a conditioned place preference paradigm (CPP) was used. To mimic the clinically relevant comorbid conditions associated with benzodiazepine abuse, mice were chronically socially defeated. This was accomplished by first identifying dominant and submissive animal pairs using the Tube Test of Social Defeat. Following the identification of dominant/submissive pairs, submissive animals were chronically stressed by being introduced to the dominant animals home cage and removed either ten minutes later or if an attack was initiated. Upon removal from the dominant mouses home cage both animals were injected with either .25mg/kg of the short acting benzodiazepine Midazolam or vehicle and conditioned to a distinct environment. Following 6 conditioning sessions (3 with drug and 3 with vehicle) mice were given free access to explore either environment; more time spent in the drug paired environment (vs. vehicle paired environment) was interpreted as a preference for the drug while less time spent in the drug paired environment was interpreted as an aversion. This technique revealed a preference for benzodiazepines in submissive (defeated) animals but not dominant animals. Furthermore, this preference for Midazolam was reversed when animals were pretreated with the Dopamine D1/D2 receptor antagonist Alpha-flupenthixol. Pharmacological and genetic manipulations of the dopamine D1 and D2 receptors have subsequently provided evidence for the involvement of these receptors in benzodiazepine reward in socially defeated animals. This work is supported by the CIHR biological therapeutics training grant.

Development

26. LOW DIETARY PHYTOESTROGEN ELICITS ANXIOGENIC BEHAVIOR IN RAT PUPS MODERATED BY DIETARY PERIOD AND MATERNAL INFLUENCE. Sanstrum, B. J., Totton, R. R., Becker, L.A. Dept. of Psychology and Neuroscience. University of Evansville, Evansville, IN 47722 USA. Previous research found that exposure to a diet containing low amounts of phytoestrogen throughout the neonatal and pre-weaning periods leads to higher ultrasonic vocalizations during maternal separation (Becker et.al, 2005) and more self-comforting behavior in a play behavior paradigm (Bies et.al, 2009) both indicating higher overall anxiety levels. This series of studies delineate critical environmental factors surrounding low phytoestrogen induced anxiogenic behavior. In the first study, dams were fed either low phytoestrogen diet or normal rat chow. Diets were presented during either three weeks of gestation or three weeks during nursing. We also examined potential effects of phytoestrogens on maternal behavior through the method of cross-fostering. Pups were kept with the birth dam or cross-fostered with a dam whose pups are the same age but of a different diet group. One male and one female from each litter were tested on each dependent measure. All offspring were placed on a diet of normal rat chow at weaning. Ultrasonic vocalizations were measured during maternal separation on days 5, 10 and 15, and open field behavior was measured after weaning (days 22-25). Ultrasonic vocalizations and open field behavior were indicative of an increase in anxiety behavior for animals who received the low phytoestrogen diet for only three weeks. Furthermore, low phytoestrogen diet caused behavioral change in the dam, which she then passed to the pups as learned anxiety. Nutrition of both dam and pups should be monitored in developmental studies. (Supported by UExplore grant from the University of Evansville)

27. PRE- AND POST-NATAL ENVIRONMENTAL ENRICHMENT HAS IMMEDIATE BUT NOT LASTING CONSEQUENCES ON OFFSPRING SOCIAL BEHAVIOUR. Mileva, G.; Pichette, N.; Sparling, J.; Baker, S.; Bielajew, C. School of Psychology, University of Ottawa, Ottawa, Ontario K1N5N6, Canada. The pre- and post-natal periods are crucial in the development of proper behavioral and cognitive function. Interactions with both the environment and conspecifics during these periods may significantly affect the animal's behavioral repertoire. In turn, animals raised in isolation may behave differently than their socialized counterparts. In this study, rats were either housed in a social colony (physical and social enrichment) or in standard laboratory conditions (control-isolation) from conception until the juvenile period. Offspring underwent a social interaction test as juveniles (5 week-old) or adults (12 week-old). The social interaction test involved placing a pair of offspring, one from each condition, into an empty square arena during which behavioural indices were measured; general activity (locomotion, rearing, grooming) and social behaviors (sniff, groom, and follow conspecifics, body and over-under contact). Colony juveniles exhibited less social behaviour than their control counterparts, displaying attenuated body and over-under contact. Colony juveniles also displayed less activity and rearing, but more grooming. Behavioural differences observed between housing were moderated by age as adult animals did not show similar differences. Nonetheless, there were notable main effects of age with adults sniffing, making anogenital contact, following, and rearing significantly more often. Further, control juveniles showed the most body contact while colony juveniles showed the least, a trend reversed in adulthood. Therefore, social behaviors differ between control juveniles and colony raised animals during a novel social encounter, but these differences do not persist into adulthood. Possibly, socially reared animals are more accustomed to social interactions and therefore display less of these behaviors during the test.

28. NEONATAL STRESS ALTERS ULTRASONIC VOCALIZATIONS IN BALB/cByJ MOUSE PUPS. Miller, O., Akintola, T., *Hodges, A. and Hohmann, C.F. Department of Biology and *Psychology, Morgan State University, Baltimore, MD 21251. Early life stress has been implicated in mental health disorders such as depression and schizophrenia. Previous studies in our lab, using BALB/cByJ mice and a split-litter design, have shown that neonatal temperature/maternal separation stress permanently alters cortical development and behavior, including social interest in neonatally stressed (STR) but also their non-stressed litter mates (LMC). Ultrasonic Vocalizations (USVs) are distress calls emitted by pups and a social communication to the dam. Neonatal stress was previously found to alter USVs in rodent pups. Based on previous observations, we hypothesized that LMC as well as STR will emit altered USVs. Between PND 2 and PND 7, STR pups were removed from the litter and exposed to 30 minutes of hot (37 C) or cold (5 C), followed by another 30 minutes of maternal separation, on alternating days. A total of 21 STR and 20 LMC (9 litters) and 22 AMC (5 litters) were tested using an U30 bat detector in order to record the USVs for each pup; the number of vocalizations at 71.3KHz per 10 minute period were counted. Our data shows that both LMC and STR mice vocalized significantly more than AMC mice. For all groups USVs increased between PND 2 and PND 7 with peaks at either PND 6 or 7. In the AMC males vs. AMC females, the females vocalized more than the males especially within the later days. Our data support the hypothesis that maternal separation in BALB/cByJ mouse pups causes an alteration in the USVs emitted in the STR pups as well as the LMC pups. Supported by SO6 GM51771 and 5R25GM058904.

29. BEHAVIOURAL AND NEURAL CHARACTERISTICS OF ACUTE AND CHRONIC MEPHEDRONE (4-METHYLMETHCATHINONE, MEOW) TREATMENT IN ADOLESCENT RATS. Craig P. Motbey1, Glenn E. Hunt2, Michael Bowen1, Suzanne Artiss1, Iain S. McGregor1. Mephedrone (4-methylmethcathinone) is a novel recreational drug that has rapidly increased in popularity in recent years. Users report mephedrone as having the stimulant-like qualities of methamphetamine and cocaine, combined with the prosocial, entactogenic effects of MDMA. Anecdotal and case study reports indicate that mephedrone may have the potential to engender compulsive patterns of use as well as toxicity in overdose. However, there have been almost no neuropharmacological investigations of the drug up to this point. Here we compared the effects of two different mephedrone doses (15 and 30 mg/kg, IP) relative to the well-known stimulant methamphetamine (2 mg/kg IP) in adolescent rats. Rats were injected, assessed for locomotor activity for 60 mins, tested in a prisoner social interaction paradigm for 10 min and their brains then processed using Fos immunohistochemistry to show patterns of brain activation. Results showed that mephedrone caused profound locomotor hyperactivity at both dose levels while tending to reduce social interaction. Patterns of Fos expression with mephedrone resembled a combination of those observed with methamphetamine and MDMA, with particularly strong Fos activation in the cortex, dorsal and ventral striatum, nucleus accumbens, ventral tegmental area (typical of both MDMA and methamphetamine) and supraoptic nucleus (typical of MDMA). These results give some empirical basis to observations that mephedrone may be something of a MDMA/methamphetamine hybrid in effect, demonstrate for the first time the powerful stimulant effects of mephedrone in animal models and reveal its ability to activate mesolimbic regions. Results from an in-progress experiment examining behavioural impairments and neural alterations associated with chronic mephedrone use will also be presented. 1School of Psychology and 2Department of Psychological Medicine, University of Sydney.

30. INCREASED IMMOBILITY OF NEONATALLY STRESSED MALE BALB/cByJ MICE IN A MODIFIED FORCED SWIM TEST A POTENTIAL MODEL OF DEPRESSION? Lalith S. Naidu, Octavia Miller, Christine F. Hohmann. Department of Biology, Morgan State University, Baltimore, MD 21251. Neonatal stress exposure effects lasting changes in behavior, morphology, neuroendocrine and molecular mechanisms in rodents. However, these effects are paradigm and strain dependent. The Forced Swim Test [FST] has been successfully used to examine stress responsiveness and therapeutic efficacy of antidepressant drugs owing to its strong endocrine and monoamine correlations. Reduction in Immobility score in FST is correlated to the therapeutic efficacy of antidepressant drugs. In this set of experiments, we have tested the hypothesis that neonatal stress exposure leads to depression like symptoms in the adult males correlating with changes in corticosterone [CORT] and monoamine [MA] levels. Our lab has developed a model of neonatal stress, combining maternal separation [1 hour] and temperature stress [cold (4C) or heat (37C) on alternating days] between Postnatal Days [PND] 2 through 7. Neonatal BALB/cByJ litters were divided into 2 groups, Stressed [STR] and Littermate controls [LMC]. STR pups were stressed using the paradigm mentioned, while LMC pups remained with the dam in the home cage. A cohort of colony reared, age matched mice [AMC] was also included in the FST. Previous experiments by our lab, using this paradigm, demonstrated sexually dimorphic levels of corticosterone during development, altered cortical morphology, exploratory behavior and aggression in adulthood. FST [n=8] was carried out for a period of 15 mins. and the animals were sacrificed after a 15 min interval. Behavior was analyzed using Biobserve-FST (BIOBSERVE GmbH, Bonn, Germany). Trunk blood was collected in EDTA coated tubes for Enzyme Immuno assay of CORT levels. Brain tissue (Cortex and Hippocampus) was harvested and flash frozen for MA analysis with HPLC. Behavioral analysis revealed a significant decrease in swimming and increase in immobility in STR Vis--Vis LMC and AMC. Currently, CORT measurements and MA analysis are being done. Supported by 3-SO6GM51971.

31. ALTERATIONS IN THE HIPPOCAMPAL EPIGENOME AS A RESULT OF PERINATAL EXPOSURE TO ETHANOL IN THE RAT. A.E. Perkins; C. F. Lehmann; R.C. Lawrence; S.J. Kelly. Dept. of Psychology, University of South Carolina, Columbia, SC 29208. Ethanol exposure during development is the leading known cause of preventable mental retardation. Fetal Alcohol Spectrum Disorders (FASD) occur more frequently than Down Syndrome and spina bifida combined, affecting 2-5% of school age children. There are a wide variety of behavioral effects associated with this disorder, including impairments in spatial learning, attention, and social skills. Ethanol exposure leads to morphological and functional changes in the hippocampus that have been associated with some of the behavioral deficits. However, it is unclear what causes these neuronal changes. This study examined the impact that developmental ethanol exposure has on the hippocampal epigenome. A three trimester model of ethanol exposure was used in which pups were exposed to ethanol throughout gestation and from postnatal days (PD) 2 to 10 (ET group). Control groups consisted of non-treated (NC) and intubated control (IC) rats. The activity of two enzymes involved in epigenetic modifications, histone deacetylase (HDAC) and DNA methyltransferase (DNMT) was measured in hippocampal tissue of 21 day old rats. Alcohol exposure led to an increase in DNMT, but not HDAC, activity in both sexes. The present study provides evidence that epigenetic modifications may be a mechanism through which ethanol can alter the developing brain and may underlie many of the behavioral changes associated with FASD. (Funded by RO1 AA11566 to SJK)

32. SELECTIVE ROLE OF NEUROPEPTIDE Y RECEPTOR SUBTYPE Y2 IN THE ANABOLIC STEROID INDUCED SEXUAL BEHAVIOR 1Keyla M. Ramos-Pratts, 2Adhly Huertas, 3Jeffrey Parrilla, 4Jos L. Roig-Lpez, 1Jennifer L. Barreto-Estrada 1Dept. of Anatomy and Neurobiology, Medical Sciences Campus, University of Puerto Rico, San Juan, PR 00936; 2Dept. of Chemistry, University of Puerto Rico, Ro Piedras Campus, San Juan, PR 00931; 3Dept. of Pharmacology, Physiology and Neuroscience, USC School of Medicine, Columbia, SC 29208; 4 Dept. of Science and Technology, Universidad del Este, Carolina, PR 00984. Anabolic androgenic steroids (AAS) were created for clinical purposes, but their abuse among adolescents is of public concern. AAS abuse is associated with physiological and neuroendocrine disorders. The hypothalamus (HYP) is normally under hormonal influence, and neuropeptide modulation in this brain region might account for the behavioral changes after AAS exposure. Neuropeptide Y (NPY) is a common neuropeptide in the CNS and is associated with inhibition of the sexual response. To assess the cellular mechanisms by which AAS affect sexual behavior, male pubertal rats were systemically exposed to 17-methyltestosterone (17-meT) for two weeks. Sexual behavior was analyzed during adulthood (PN-75). AAS exposure decreased the latency to first ejaculation, and increased the number of ejaculations. To assess the pharmacological effects of NPY, pubertal males were systemically exposed to 17-meT, and centrally infused in the HYP with BIIE 0246, a selective antagonist for NPYY2 receptor. AAS decreased the latency to first ejaculation and increased the number of mounts. BIIE 0246 in the HYP decreased the latency to first ejaculation and increase the number of mounts, suggesting a role of NPY in the normal inhibition of sexual behavior. The AAS treatment, together with blockage of Y2R further decreased the latency to the first ejaculation. Preliminary data showed that AAS decreased mRNAs for NPY receptors in the HYP. Our results suggest a role of NPYreceptors in the regulation of sexual function and in the interaction between AAS and neuropeptide systems. NIH-NCRR(2P20RR016470-09), NIH-EARDA(G11HD046326), RCMI(G12RR030551).

33. ENVIRONMENTAL ENRICHMENT ATTENUATES DEVELOPMENTAL LEAD EXPOSURE-INDUCED DEFICITS ON EGOCENTRIC AND ALLOCENTRIC SPATIAL NAVIGATION IN RATS. Goodwill, H.S.; McLean, M.C.; Wheeler, A.P.; Schroeder, J.A. Dept. of Psychology and Behavioral Neuroscience Program. Connecticut College, New London, CT 06320 USA. The neurotoxic effects of developmental lead exposure as well as lead-induced cognitive deficits have been widely documented. However evidence also suggests that exposure to an enriched environment during development is neuroprotective and may be sufficient to overcome lead-induced cognitive deficits. Fischer rats were given lead acetate (0.4%)-laced drinking water and were exposed to an enriched (2-3 peer cagemates, housing enclosures, novel objects) environment or housed alone from post-natal day 21 to day 100. Allocentric spatial navigation was assessed using the Morris Water Maze and egocentric navigation was assessed using the Cincinnati maze. Single-housed, lead-exposed rats displayed significant spatial learning deficits in an allocentric navigation task, whereas group-housed lead-exposed animals performed as well as non-lead exposed control animals. Lead exposure did not affect performance in an egocentric spatial navigation task suggesting that brain areas responsible for cognitive map formation are not as susceptible to lead toxicity. Behavioral results are compared to NeuN histochemical evaluations of neuronal cell counts in the hippocampus and striatum and atomic absorption spectrophotometic determinations of brain and blood lead levels.

34. DEVELOPMENTAL EXPOSURE TO CHRONIC STRESS AND MANGANESE IN RATS, BUT NOT LOW LEVELS OF LEAD, AFFECTS ANXIETY RESPONSES AND EGOCENTRIC LEARNING. Williams, M.T.; Graham, D.L.; Amos-Kroohs, R.M.; Braun, A.A.; Grace, C.E.; Schaefer, T.L.; Skelton, M.R.; Vorhees, C.V. Neurology, Cincinnati Childrens Research Foundation, & Dept Pediatrics, U. Cincinnati COM, Cincinnati, OH Children of lower socioeconomic status are likely to be exposed to a number of environmental factors that influence CNS development. Not only do these children have elevated cortisol resulting from chronic stress such as neglect, impoverishment, or lack of resources, they are also more likely to be exposed to toxic metals such as lead (Pb). Exposure to other metals, such as manganese (Mn), is also of concern because of the harmful effects at high levels and potential interactions with Pb. The purpose of this study was to determine the immediate physiological and long-term behavioral effects in rats exposed to chronic stress (i.e., reduced bedding) in combination with low-level Pb and/or Mn. On postnatal day (P)4, Sprague-Dawley rats were housed in cages containing no woodchip bedding (Barren) or normal bedding (Standard). Pups were gavaged every other day with 10 mg/kg Pb, 100 mg/kg Mn, a combination of Pb and Mn, or control solution from P4 through P28; behavioral testing began at P60. Mn and Barren produced decreased body weight relative to controls. Barren treatment increased basal corticosterone on P11 and exaggerated levels following a stressor on P19. Barren-treated animals showed minor changes in the light/dark test. Mn-treated animals had inappropriate responses in the light/dark test, elevated zero maze, and conditioned fear compared with controls. Mn-treated animals had a blunted acoustic startle response. Barren- and Mn-treated animals exhibited increased errors during egocentric learning in the Cincinnati water maze compared to controls. In the Morris water maze, no effects of Barren or metal treatments were observed, except for an increased latency in Mn-treated animals on day 1 of reversal. The results suggest that developmental stress and Mn have long-term effects on behavior whereas low Pb exposure has less effect. (Supported by RO1-ES015689 and T32-ES07051)

35. EFFECTS OF NEONATAL STRESS ON SOCIAL BEHAVIOR AND CORTICAL AND HIPPOCAMPAL BDNF LEVELS IN Balb/CByJ MICE. *Subedi, K.; *Naidu, L.; ^Azhagiri, A.; ^Pardo, C.A.; *Koban, M.; and *Hohmann, C.F. *Department of Biology, Morgan State University, Baltimore, MD 21251, ^Department of Pathology, Johns Hopkins School of Medicine. Neonatal stress and trauma are risk factors for mental health disorder, such as autism and schizophrenia, which also show altered social behavior. In animal models, neonatal stress leads to permanent neuroendocrine and behavioral changes in adulthood. Expression of the neurotrophic factor BDNF is also altered after stress. Our lab has shown previously, that neonatal stress in Balb/CbyJ mice, results in altered development and plasticity in cerebral cortex. Here we test, if neonatally stressed (STR) BalbC/ByJ mice exhibit altered social behavior in adulthood, and altered BDNF levels in cortex/hippocampus. In a split litter design, half of the pups (STR) from a dam were exposed, for 1h/day, from postnatal day (PND) 2 to 7, to 30 min of cold (4^{o}C) or hot (37^{o}C) stress on alternating days, followed by 30 minutes in room temperature, to reacclimatize body temperature. Littermates (LMC) remained with the dam during the stress period. A cohort of age-matched, colony reared mice (AMC) was also tested. Thirty adult male mice, (10 STR, 10 LMC and 10 AMC) were tested using the Automated Social Approach Task developed by J. Crawley. Detailed analysis of video-taped records, using CleverSys software, showed that STR males exhibited significantly less social interaction and exploratory behavior than LMC and AMC males although self-grooming was significantly higher in STR than LMC mice. Following behavioral testing, brains of all mice were collected, and stored at -70 °C until analysis of BDNF levels via Western blot technique. Levels of mature BDNF, in hippocampus and cortex, were significantly lower in STR males, compared to LMC. In contrast, pro-BDNF levels in both hippocampus and cortex were higher than in AMC and LMC mice. These data suggest that neonatal stress induced altered processing of BDNF in hippocampus and cortex. This could contribute to altered morphology and plasticity in these regions during brain development and consequently impair social and exploratory behaviors in adulthood. Supported by 3-SO6GM51971

36. BEHAVIORAL ANALYSIS OF HDC-KO MICE IN THE BEHAVIORS RELEVANT FOR TOURETTE SYNDROME Authors: Baldan Ramsey, L.C.; Ohtsu, H.; de Araujo,I.; Pittenger, C. Yale University, New Haven, CT 06519 USA Tourette Syndrome (TS) is a neurodevelopmental disorder characterized by stereotyped motor and vocal tics. Understanding its neurochemical underpinnings will lay the groundwork for new pharmacological treatments. In a recent genetic study of a family in which a father and all eight of his children have TS, the State laboratory identified a dominant-acting mutation in the gene encoding the rate-limiting enzyme in the biosynthesis of histamine: histidine decarboxylase (HDC; Ercan-Senicek et al, 2010). We are examining HDC knockout mice as an animal model of this Mendelian form of TS. We found both HDC-KOs and heterozygotes to have deficits in prepulse inhibition (PPI). Administration of a low dose of amphetamine produced stereotypical behaviors in the knockout, but very few in heterozygous and WT mice. HDC-KO mice showed significantly reduced locomotor activity while engaged in stereotypical behaviors. These phenotypes mirror aspects of TS and support the face validity of the model. The model possesses inherent construct validity, since the KO recapitulates the functional disruption found in this TS family. Histamine inhibits midbrain dopamine neurons and modulates dopamine release. We hypothesize that reduction in histamine will increase extracellular levels of dopamine in the dorsal striatum. Levels of dopamine in the striatum of HDC-KO and WT mice were measured using in vivo microdialysis coupled to HPLC-ECD. Preliminary data show increasing dopamine in the dark (active) phase in knockout mice, relative to controls. These experiments will establish a new basis for future studies on the neuropathology of the condition.

8:30-10:45 **Symposium 4:** AUTISM-RELEVANT BEHAVIORS OF MOUSE MODELS OF ASD. Chairperson: **Robert J. Blanchard**

AUTISTIC FEATURES AND THEIR POSSIBLE TREATMENT IN THE FMR1-KNOCK OUT MOUSE**.** S. Pietropaolo and W.E. Crusio. *Institute de Neurosciences Intégratives et Cognitives d'Aquitaine (INCIA), UMR 5287 CNRS, Université Bordeaux, Avenue des facultés, 33405 Talence, Franc.* Autism spectrum disorder (ASD) is a developmental disease with multi-genic bases and a highly complex symptomatology, for which effective treatments are still lacking. There is a general consensus that the diagnosis of ASD mainly depends on a triad of core deficits comprising (i) impaired social interaction, (ii) reduced communication skills and (iii) the expression of repetitive behaviours. Variable symptoms include sensory hypersensitivity, epilepsy, sleep and activity disturbances. The ideal rodent model for autism will include features having conceptual analogies to most of the core human symptoms included in the triad and incorporate some of the variable abnormalities as well. Here we evaluated the ASD-like phenotypes of the *Fmr1* mouse model of Fragile X syndrome, a human disease that is associated to autism. We demonstrated the presence of selected ASD-like core symptoms (deficits in social recognition and interaction, presence of repetitive behaviors, reduced behavioural flexibility) and additional alterations (hyperactivity) in *Fmr1*-KOs. These behavioural abnormalities were more pronounced in the C57BL/6J (B6) than the FVB/N background. Hence, we tested the efficacy of possible treatments in B6 *Fmr1*-KOs.

ENHANCED SOCIABILITY, HYPER-DEFENSIVENESS AND DECREASED COCAINE-INDUCED BEHAVIORAL REACTIVITY IN MALE MICE WITH MECP2-308 MUTATION. Pearson, B.L.; Meyza, K.Z.; Defensor, E.B.; Pobbe, R.L.H.; Bolivar, V.J.; Blanchard, D.C.; Blanchard, R.J. Disruptions in the gene coding the Mecp2 protein, an autism candidate gene with a variety of influences on transcriptional regulation, underlie the majority of cases of Rett syndrome. Mice with mutations of Mecp2 show varying, but often severe phenotypic abnormalities. Emerging work has shown bi-directional alterations in social behavior of Mecp2 mutants. Here, we assessed $Mecp2^{308/Y}$ hypomorphic mice in a battery of tests of social behavior, defense, and responses to hedonic and psychoactive stimuli. In semi-natural and close-proximity social tests, Mecp2 mutants show a pattern of increased affiliative behavior. We next sought to determine if alterations in emotional systems underlie the increased social propensities of these mice. Mecp2 mutants did not differ from their wild-type littermates in anxiety, but they did display a consistent increase in active defensive responses in the mouse defense test battery. Finally, we attempted to discern whether Mecp2 mutation would affect sensitivity to reward. We found decreased locomotor responses to cocaine after repeated, daily injections in $Mecp2^{308/Y}$ mutant mice with no changes in investigation of female mouse estrous urine. We propose that alterations in reward neurocircuitry might influence differential sensitivity to social stimuli. We discuss this possibility as well as ongoing and future projects aimed to test this hypothesis.

SOCIAL AND ENVIRONMENTAL FACTORS RELEVANT TO THE DEVELOPMENT OF SOCIABILITY IN INBRED MICE Yang, M.; Crawley, J.N. Laboratory of Behavioral Neuroscience, Intramural Research Program, National Institute of Mental Health, Bethesda, MD 20892, USA Brief description of talk: While searching for genetic causal factors of autism remains as the most exigent research goal of the scientific community, the roles of non-genetic factors are also of great importance for understanding the etiology and developmental trajectory of the disorder. This talk will focus on our recent research on environmental and social factors relevant to the deficits in social approach behaviors in BTBR mice. Our previous study showed that BTBR tested in the dark phase showed the same social deficits as BTBR tested in the light phase, and B6 tested in both circadian phases showed high sociability. These findings justify light phase testing for most standard social tasks conducted outside the animal's home environment and improved our current knowledge on environmental factors relevant to the outcomes of social behavioral experiments. We then explored if early postnatal maternal environment has major influences on the development of sociability in mice. We reported that cross-fostering has minimal influences on the development of mouse social behaviors in B6 and BTBR mice, either at the juvenile age or during adulthood. B6 and BTBR mice raised by females of the opposite strain exhibited behaviors similar to those raised by their biological mothers. These findings strongly argue against the early Refrigerator Mother hypothesis of autism and contributed to current understanding on essential social-environmental factors relevant to sociability. As a continuation on the search for socio-environmental factors relevant to the development of sociability, we designed a peer social enrichment paradigm to test if housing equal numbers of highly social B6 and asocial BTBR in the same home cage during juvenile/adolescent period could alleviate social deficits in BTBR. Our findings showed that BTBR lived with B6 cagemates developed high sociability, whereas control BTBR which lived with BTBR cagemates continue to show social deficits. Continuing research is focusing in identifying which components of social interaction between BTBR and B6 that led to the significant improvement in sociability in BTBR.

BTBR MICE SHOW AUTISM-LIKE BEHAVIOR CHANGES ON ETHOLOGICALLY-RELEVANT TASKS RELATED TO SOCIALITY. Blanchard, D. Pacific Biosciences Research Center, University of Hawaii, Honolulu, HI 96822 USA. Over several days in a Visible Burrow System affording ample space as well as tunnels and burrows, BTBR male mice in same-sex groups showed high magnitude, consistent, reductions in social behaviors, compared to C57Bl/6J controls. Although many specific behaviors were changed, enhanced avoidance of conspecifics was a consistent theme. When pairs of unfamiliar same-strain males were placed in a small enclosure in which movement necessitated contact with the conspecific partner, BTBRs showed consistent, high magnitude reductions in nose to nose tip contact, accompanied by attempts to crawl under the other animal. Analyses of similar data from pairs consisting of 1 BTBR and 1 B6 male strongly suggested that eye contact may be selectively aversive for the BTBR males. These two tasks, designed to afford ample opportunity for escape/avoidance, or, forcing a substantial degree of proximity between animals, provide different but intersecting views of the social deficiencies in male BTBR mice.

11:15-12:15 **Keynote Speaker: Kerry Ressler**, Emory University
Examining fear and its regulation in mice and men

EXAMINING FEAR AND ITS REGULATION IN MICE AND MEN. Ressler, K.[1]; Choi, C.[1]; Davis, M. [1]; Heldt, S. [1]; Bradley, B. [1]; Hammack, S. [2]; Toufexis, D. [2]; May, V. [2] [1]Emory University, Atlanta, GA. [2]University of Vermont, Burlington, VT. Traumatic events that produce extreme fear and horror are common, but not all individuals develop posttraumatic stress disorder (PTSD) as a result of such exposure. What mediates risk and resilience in the development of stress-related psychopathology is a critical question. Biological factors, such as genotype and neurobiology, interact with environmental factors, such as childhood background and trauma load, to affect vulnerability and resilience in the aftermath of trauma exposure. A core symptom of PTSD is the inability to control fear, which has led some to conceptualize PTSD as a disorder of fear or, more importantly, its inhibition. This talk focuses on translational methods to examine fear conditioning and inhibition of fear in PTSD. We will focus on mechanisms of synaptic plasticity, including the BDNF and NMDA-dependent mechanisms of fear learning and extinction. We will also describe recent findings that an additional neurotrophic factor, pituitary adenylate cyclase-activating polypeptide (PACAP), and its PAC1 receptor play a role in human psychological stress responses, including PTSD. In these new studies, we find that PACAP levels in the blood and a polymorphism within the PAC1 gene are associated with PTSD symptoms. In both cases the association is in women and not men, and we find that the most robust genetic polymorphism in the PAC1 receptor lies within an estrogen response element, potentially explaining the apparent sex-specificity. These data suggest that PACAP levels and ADCYAP1R1 SNPs may serve as useful biomarkers to further our mechanistic understanding of PTSD. Together this work suggests that understanding mediators of synaptic plasticity across translational models of fear will further our understanding and potential therapeutic approaches to fear-related disorders in humans, such as PTSD.

6:30-8:30 **Poster Session 2:**

Cognition

37. ANDROSTENEDIONE IS ASSOCIATED WITH SPATIAL REFERENCE AND WORKING MEMORY IMPAIRMENT IN TRANSITIONAL AND SURGICALLY MENOPAUSAL MIDDLE-AGED RATS Acosta, J.I.1,2; Mennenga, S.M.1,2; Camp, B.W.1,2; Gerson, J. E.1; Villa, Stephanie R.1; Bimonte-Nelson, H.A.1,2 1 Department of Psychology, Arizona State University, Tempe, AZ 85287, United States 2 Arizona Alzheimer's Consortium, Tempe, AZ 85287, United States After natural menopause, the androgen androstenedione becomes a primary hormone secreted by the residual follicle deplete ovaries. Recently we found a positive correlation between higher androstenedione serum levels and spatial working memory errors in rats that have undergone experimentally-induced ovarian follicular depletion via 4-vinylcyclohexene diepoxide (VCD). In a follow-up study, we examined the hypothesis that androstenedione impairs memory by evaluating the cognitive effects of androstenedione administration in a rodent model. Middle-aged ovariectomized (OVX) rats received vehicle or one of two doses of androstenedione, with goal doses resulting in blood levels seen in follicular deplete, ovary-intact animals from our prior study (Acosta et al., 2009, 2010). Rats were tested on a spatial working and reference memory using the water radial arm maze (WRAM). Androstenedione at the highest dose impaired WRAM reference memory performance during learning as well as the ability to handle multiple items of spatial working memory information (WRAM) as memory demand was elevated. Serum androstenedione levels were comparable to the higher serum levels we have shown previously to correlate with impaired working memory, and this correlation was replicated (Acosta et al., 2009, 2010). These findings suggest that androstenedione, a hormone produced by the follicle deplete ovary, is detrimental to spatial learning, reference memory, and working memory.

38. POTENTIAL ENHANCEMENT IN THE TRANSFER OF SYMBOLIC LEARNING IN RAT MOTHERS COMPARED TO VIRGIN FEMALES. Bilinski, T.; Au, A.; Meyer, E.; Kinsley, C.H. Dept. of Psychology and Center for Neuroscience. The University of Richmond, University of Richmond, VA 23173 USA. We know that reproductive experience (parity) enhances various aspects of learning. Positive modifications in the acquisition and usage of information likely provide an advantage to females at a time when other of their faculties are taxed by the burden of carrying or caring-for young. Spatial learning, predation, classical conditioning, and recently, prospective memory, have been shown to be enhanced. Here we are investigating whether or not these enhanced learning capacities will enable maternal females to more efficiently associate a symbol with a reward (a Froot Loop paired with one of four symbols- a circle, a triangle, a cross, or multiple wavy lines). Mothers, virgin females, and males, were trained for four days in an open field maze in which each wall presented one of the above symbols, and had a piece of Froot Loop placed in one of the clean bottle caps located directly under each symbol. After the four days of training, the rats were each tested in an eight-arm radial arm maze to assess the ability and speed of the rats to recognize the symbol paired with the reward from the open field maze and apply it to the new environment to more quickly find a reward. To date, our results are as follows; N = 6, virgins (mean = 279.08s), mothers (mean = 222.25s), and males (mean = 158.63s). Eventually, we will examine differences in the neuroanatomy/function of mothers, virgins, and males. At present, these data suggest that reproductive experience may regulate yet another type of learning, which can be used to benefit both mother and offspring.

39. ENDOGENEOUS HUNGER SUBSTANCE OREXIN A MODULATES SPATIAL PLASTICITY. Yutaka Oomura, Shuji Aou, Koji Fukunaga, and Kazuo Sasaki. Dept. Physiol. School of Medicine, Kyushu University, Fukuoka, Japan. The glucose-sensitive neurons in the lateral hypothalamic area of feeding center produce orexin A (OxA) and send their axons to the CA1 neurons in the hippocampus which expresses orexin receptors OxR1 and R2. OxR1 develops during adult period and OxR2 during young period. OxA released during food intake facilitates food intake by the activation of glucose-sensitive neurons and inhibits the glucoreceptor neurons in the ventromedial nucleus, satiety center. The released OxA then reaches to the hippocampus and modulates spatial plasticity. Namely the Morris water maze tasks showed that 1.0 to 10 mM OxA administered icv retarded spatial learning and memory in young period, but facilitated in adult period. On the same concentrations of OxA, LTP of CA1 neurons in vitro hippocampus slices was suppressed in young period but facilitated in adult period. The phosphorylations of synaptic synapcin 1-3 was not influenced by OxA, but those posphorylations of postsynaptic PKC, CaMKII, ERK and MAR CKS were suppressed dose dependently in young period, but facilitated in adult period. These results indicate that OxA modulates behavioral plasticity with modulations of CA1 LTP. We are then experimenting using OxR1 and R2 knockout mice.

40. RAPID EFFECTS OF INTRAHIPPOCAMPAL DELIVERY OF 17β-ESTRADIOL ON OBJECT PLACEMENT LEARNING IN FEMALE MICE. Phan, A; Molinaro, LP; MacLusky, NJ; Choleris, E. Typically, estrogens effects on learning and memory are studied hours to days after their administration, when their transcriptional responses predominate. In addition to these genomic effects, estrogens also rapidly affect neuronal electrophysiology and morphology within minutes to 1hr of estrogen application. Less is known about the behavioral consequences of these rapid effects of estrogens on learning and memory. We have previously shown that systemic administration of physiological doses of 17β-estradiol and estrogen receptor (ER) α agonist PPT rapidly enhanced learning (object placement, object recognition, social recognition). However, ERβ agonist DPN slightly facilitated object placement, did not affect object recognition, and may impair social recognition at higher doses. Currently, we are working to determine the neural circuits involved in estrogens rapid effects on learning. Microinjections of 17β-estradiol into the hippocampus of young adult ovariectomized female CD1 mice (50nM, 100nM, and 200nM at a volume of 5μL) were performed, 15min prior to testing in an object placement paradigm. Consistent with our systemic experiments, this paradigm was completed within 40min of microinjection and the results were ethologically analyzed. We found that administration of 100nM of 17β-estradiol improved object placement learning in these female mice within the rapid 40min time frame. Thus it seems as though the hippocampus is capable of mediating 17β-estradiols rapid facilitatory effects on object placement learning. Funded by NSERC.

41. POTENTIAL EVIDENCE FOR PROSPECTIVE MEMORY IN PAROUS RATS. Franssen, RA.1; Rafferty KA.2; McDaniel, EM.2; Byce SJ.2; Kinsley CH.2. 1Department of Biological Sciences, Longwood University, Farmville, VA 23909 USA; 2Center for Neuroscience, Department of Psychology, University of Richmond, Richmond, VA 23173 USA. The experience of motherhood exerts innumerable demands on the female rats brain as she copes with the challenges of caring for her young. Improvements in the rats neurobiology provide for advantages such as superior learning and retrospective memory. Here, we identify another change accompanying motherhood, an enhancement in prospective memory (PM). PM, the anticipation of future events, has been identified in scrub jays and humans, but it is rare. We examined PM in nulliparous and primiparous female rats by water-depriving and training them to acquire water in an open field maze (OFM). Individuals were subsequently placed back in their

cages and encountered either replete or no water. Rats without access to water in the cage were predicted to drink more water. Drinking more water in the maze demonstrates anticipation of not subsequently having water in the cage, and thus indicates PM. We found that mothers who did not have cage water drank more in the maze than mothers who did, as well as non-mothers, suggesting that rat-mothers are capable of planning. To control for possible differences in water consumption due to a mothers increased metabolism, we added a 2-hour period between OFM trials in which rats in both conditions had access to abundant water in the cage. Preliminary results suggest that mothers who are water-deprived in the cage, even with the extra 2-hour drinking period, consume more water in the maze than non-mothers. An enhancement in PM would provide the maternal rat with an advantage to compensate for the many demands placed upon her.

42. MEDIAL SEPTAL GABAERGIC CONTROL OF HIPPOCAMPAL ACETYLCHOLINE RELEASE AND SHORT-TERM MEMORY. Roland, J.J.1; Janke, K.L.2; Savage, L.M.3; Servatius, R.J.1,2,4; Pang, K.C.H.1,2,4 .1SMBI, 2GSBS, UMDNJ, Newark, NJ; 3Psych Dept, SUNY, Binghamton, NY; 4DVA Med Ctr, East Orange, NJ. The septohippocampal pathway contains cholinergic and GABAergic projections and is important for learning and memory. Selective damage of the medial septal-diagonal band (MSDB) cholinergic or GABAergic neurons does not impair spatial reference or spatial working memory. However, selective MSDB GABAergic lesions impair delayed match to position. Changes in hippocampal acetylcholine (ACh) have been tied to memory; deficits and enhancements in memory are correlated with decreases or increases of ACh, respectively. Control of hippocampal ACh could occur from MSDB interneurons or feedback from hippocampal-septal projections. Currently we examined MSDB GABAergic lesion effects on hippocampal ACh efflux during spontaneous alternation (Exp 1), delayed nonmatching to position (Exp 2) and nonmatching to position with minimal delay (Exp 3). In Exp 1, we saw no difference between groups on activity, alternation, or ACh efflux. In Exp 2, lesioned animals were behaviorally impaired and had reduced ACh levels during maze training. In Exp 3, lesioned animals were not behaviorally impaired and showed no differences in ACh efflux. These results demonstrate that, 1) increased task demand enhanced ACh in control but not lesioned animals, and 2) this difference in ACh may be the result of increased inhibitory feedback from the hippocampal-septal projection, which is responsible for the memory impairment. Therefore, the interaction of the cholinergic and GABAergic septohippocampal systems may be important for hippocampal-dependent learning and memory. Supported by NIH, DVA & SMBI.

43. WORKING MEMORY IMPAIRMENTS WITH PROLONGED HIGH ALTITUDE RESIDENCE: AN FMRI STUDY. Xiaodan Yan1,3,4#, Jiaxing Zhang1,2#, Qiyong Gong5, Xuchu Weng1 1Laboratory for Higher Brain Function, Institute of Psychology, Chinese Academy of Sciences, Beijing, China. 2Department of Physiology, Medical College of Xiamen University, Xiamen, China. 3Center for Neural Science, New York University, New York, NY, USA. 4Cognitive Science Department, Rensselaer Polytechnic Institute, Troy, NY, USA 5Huaxi Magnetic Resonance Research Center, West China Hospital, Sichuan University, Chengdu, China./// The neuropsychological functioning at high altitude has raised concerns among researchers. Declined performance in short term memory has been reported with acute ascending to very high altitude (6000 meters above the sea level). The current study aimed to investigate the impact of prolonged chronic HA exposure on working memory (WM) capability, especially the neural mechanisms of such impact. 28 high altitude (HA) residents of Qinghai-Tibetan Plateau (2616~4200m) as well as 30 sea level (SL) residents were recruited for a functional magnetic resonance imaging (fMRI) study. The HA subjects showed increased reaction time in both of the verbal and spatial working memory tasks, and decreased response accuracy in the verbal working memory task. In both tasks, the two groups showed activation in the typical regions associated with the two working memory tasks, including the medial frontal cortex, the precentral cortex, as well as the decreased activation in the posterior cingulate cortex and precuneus cluster. With group comparison statistics, in the verbal WM task, the HA subjects showed decreased activation on the left sides of the inferior frontal gyrus, the middle occipital gyrus, the pyramis of vermis, the thalamus, the middle frontal gyrus and the lingual gyrus; in the spatial WM task, the HA subjects had increased activation in the left pyramis, the left superior temporal gyrus and decreased activation in the left middle occipital gyrus. The behavioral performance also showed significant correlations with the BOLD signal change amplitude in the typical regions associated with the two working memory tasks, as well as the Granger causality values between them. In conclusion, the current study revealed impairment in verbal working memory among HA residents, with possible impairment as well as compensation in the spatial working memory.

44. C-FOS EXPRESSION IN THE PERIAQUEDUCTAL GRAY VARIES RELATIVE TO THE METHOD OF CONDITIONED TASTE AVERSION EXTINCTION EMPLOYED. Mickley, G.A.; Wilson, G.N.; Remus, J.; Ramos, L.; Ketchesin, K.; Biesan, O.; Luchsinger, J. & Prodan, S. The Neuroscience Program and The Department of Psychology, Baldwin-Wallace College, Berea, OH 44017 USA. A conditioned taste aversion (CTA) is acquired when an animal consumes a novel taste (CS) and then experiences the symptoms of poisoning (US). Following CTA training, animals will avoid the taste that was previously associated with malaise. This defensive reaction to a

learned fear can be extinguished by repeated exposure to the CS alone. However, following a latency period in which the CS is not presented, the CTA will spontaneously recover (SR). Thomas et al. (2005) have used an explicitly unpaired (EU) extinction procedure that disassociates a light CS and footshock to thwart renewal of a conditioned emotional response. Here we applied similar procedures to the CTA paradigm and also evaluated the ability of EU extinction procedures to affect behavioral indicators of SR and c-Fos expression. Fluid-deprived Sprague-Dawley rats acquired a CTA [3 pairings of 0.3% oral saccharin (SAC; the CS) and 81mg/kg i.p. lithium chloride (LiCl; the US)] followed by extinction trials consisting of multiple exposures to either, (a) CS-only, or (b) CS and US on alternate days (EU extinction). Both extinction procedures resulted in >90% reacceptance of SAC and were followed by a 30-day latency period of water drinking. Rats were then tested for SR with a final exposure to SAC before sacrificing. Brain c-Fos protein expression was evaluated via immunohistochemistry. Rats in the CS-only group exhibited significant suppression of SAC drinking during their SR test compared to their consumption at the end of extinction. However, animals in the EU extinction group did not show such SR of the CTA and drank significantly more than the CS-only rats. The brains of EU-extinguished rats and CS-only extinguished rats did not differ in the number of c-Fos-labeled neurons in gustatory neocortex, medial prefrontal cortex, basolateral amygdala or the central nucleus of the amygdala. However some small, but reliable, differences were detected in Periaqueductal gray (PAG) especially in the dorsolateral region. Thus, behavioral differences in SR between the EU and CS-only extinction animals were not represented by corresponding changes in the neural activity of several brain nuclei classically associated with extinction learning. However a detailed analysis of PAG c-Fos expression provides hints about some of the physiological changes evoked by these 2 extinction paradigms. The findings are clinically relevant as we seek the development of treatments for deficits in fear extinction (e.g. PTSD, phobias). Supported by NIMH grant: 2-R15-MH063720-03.

45. AN L-TYPE CA++ CHANNEL BLOCKER ENHANCES THE ACTION OF DONEPEZIL IN OBJECT RECOGNITION MEMORY IN RATS. Rose, G.M.; Trippodi-Murphy, C. Center for Integrated Research in Cognitive & Neural Sciences, Southern Illinois University, Carbondale, IL 62901 USA. Two basic types of pharmacological treatments are currently approved for symptomatic treatment of Alzheimer's disease (AD) in the United States: acetylcholinesterase inhibitors, such as donepezil, and memantine, a glutamatergic NMDA receptor antagonist. Unfortunately, neither treatment is very effective, likely because AD is not due to the disruption of a single neurotransmitter system or brain circuit. We have been investigating the possibility that combination treatments could provide better efficacy. In particular, we have focused on the effects of combining currently prescribed drugs with MEM 1003, a dihydropyridine L-type Ca++ channel antagonist, since it is known that age-related upregulation of these channels in hippocampal neurons is correlated with memory impairments. The novel object recognition task was used to assess memory in adult male Sprague-Dawley rats. In our paradigm (15 minute training period), strong memory is seen at a 1-hour delay but complete forgetting occurs by 24 hours. Both donepezil and memantine given prior to training enhanced 24-hour memory over a narrow dose range. MEM 1003 also enhanced memory. Individually ineffective doses of donepezil and memantine did not combine to produce significant 24-hour memory. However, the combination of individually ineffective doses of MEM 1003 and donepezil significantly enhanced retention. These results support the idea that combined treatments, particularly those that include both pre- and postsynaptic targets, could provide more effective therapies for age-related memory disorders.

46. IMPACT OF METHYLPHENIDATE ON A RODENT MODEL OF SUSTAINED ATTENTION AND LOCOMOTION: DIFFERENTIAL EFFECTS ON HIGH VERSUS LOW PERFORMERS. R Chu, S Nicholson, JS Shumsky, BD Waterhouse. Dept Neurobiol Anat, Drexel Univ Coll Med, Phila, PA 19129. Methylphenidate (MPH) is the drug of choice for treating Attention Deficit Hyperactivity Disorder (ADHD). However, the dose needed to manage symptoms varies amongst patients and must be titrated over time for each individual. Preliminary studies in our laboratory have shown a similar effect in rats performing a sustained attention task. Prior to drug treatment, trained rats were classified as high and low performers on the basis of baseline performance. Animals were administered oral MPH across a range of concentrations, including ones that produce clinically relevant blood plasma levels, to determine the doses that elicited peak effects for each rat. In the same manner that the optimal dose to manage ADHD symptoms varies amongst patients, the optimal dose that enhanced performance varied amongst rats. At 8 mg/kg, low performing rats showed substantial increases in task performance whereas high performers showed no improvement. Nevertheless, some high performing rats still responded to MPH, suggesting that these results are not due to a ceiling effect. At a dose previously shown to induce hyperactivity (10 mg/kg), high versus low performing animals exhibited different locomotor responses. Overall, these data suggest that only low performing animals receive a substantial benefit from a therapeutically relevant dose of MPH. Furthermore MPH shows subject specific and dose dependent effects on performance in the sustained attention task that may be correlated with its effect on locomotion. Our working hypothesis is that locomotor response may serve as a useful predictor of MPHs impact on vigilance as measured by the sustained attention task.

47. EFFECTS OF CHRONIC INTERMITTENT ETHANOL EXPOSURE ON CORTICO-STRIATAL-MEDIATED DISCRIMINATION AND REVERSAL LEARNING. DeBrouse, L; Plitt, A; Hurd, B; Saksida, L; Bussey, T; Camp, M; Holmes, A. Section on Behavioral Science and Genetics, Laboratory for Integrative Neuroscience, National Institute on Alcoholism and Alcohol Abuse, NIH, Rockville, MD, USA. Alcoholism is associated with the impairment of cognitive and executive functions. However, the precise nature of these impairments and the mechanisms underlying them are not yet fully understood. The goal of the current study was to test the effects of chronic intermittent exposure to vaporized ethanol (CIE) on subsequent, touchscreen-based (striatum-mediated) visual discrimination and (corticostriatal-mediated) reversal learning in C57BL/6J mice. Following operant shaping, mice were subjected to 2 cycles of CIE or a control condition of air-exposure. One CIE cycle comprises 4 consecutive days of 16 hr/day ethanol vapor exposure (target blood ethanol concentration=175 mg/dL) followed by 3 days withdrawal. Preliminary results indicate that CIE produces facilitation, rather than impairment, in reversal learning, as compared to air-exposed controls. Improved reversal after CIE was associated with increased response reaction times, possibly suggesting increased error monitoring and/or enhanced sensitivity to negative reinforcement. Our working hypothesis is that CIE alters the function of corticostriatal systems supporting the formation of well-learned and habit-like behaviors. Neuronal morphology was examined by reconstructing 3-dimensional neurons in the dorsolateral striatum using Golgi Cox staining. Mice exposed to 2 cycles of CIE showed altered distribution of dendritic material. Terminal branches, areas thought to be more plastic, increased. This work could have implications for understanding the mechanistic basis of compulsive behaviors in alcoholism. Research supported by the NIAAA intramural research program.

48. DORSAL STRIATAL DOPAMINE DEPLETION IMPAIRS BOTH ALLOCENTRIC AND EGOCENTRIC NAVIGATION. Braun, A.A.; Graham, D.L.; Schaefer, T.L.; Vorhees, C.V.; Williams, M.T. Successful navigation requires complex interactions among multiple distinct, but overlapping processes that can be subdivided into egocentric (self-oriented, route-based) or allocentric (spatial, map-based) learning. Route-based navigation has been shown to be impaired following acute neurotoxic exposure to the dopaminergic (DA) drugs (+)-methamphetamine and (+)-amphetamine, but not the serotoninergic (5-HT) drugs ()-3,4-methylenedioxymethamphetamine or ()-fenfluramine. The dopaminergic-rich neostriatum (caudate-putamen) is involved in both allocentric and egocentric navigation. This experiment tested whether selective DA loss through neostriatal 6-hydroxydopamine (6-OHDA) injections would impair one or both types of navigation. Two weeks following 6-OHDA injections, rats began testing in the Cincinnati water maze (CWM) and the Morris water maze (MWM) for route-based and spatial navigation, respectively. 6-OHDA treatment significantly increased latency and errors to find the escape in the CWM and path length during acquisition in the MWM with no difference in cued MWM. Neostriatal monoamine levels were determined 2 or 7 weeks post-surgery. 6-OHDA bilaterally depleted DA 80% and increased DA turnover at both time points and decreased norepinephrine (NE) levels 7 weeks post-surgery. 6-OHDA injections did not alter monoamine levels in the prefrontal cortex, but decreased hippocampal NE levels at 7 weeks. 6-OHDA treatment did not have an effect on body weight. Future experiments will determine the dose-response and sub-regional effects of 6-OHDA (dorsolateral and dorsomedial striatum) on navigation as well as test lesions to other structures involved in these circuits.

Reward and Addiction

49. ANTAGONISM OF CARBACHOL-INDUCED 22 kHz VOCALIZATION BY AMPHETAMINE IN THE RAT HYPOTHALAMIC-PREOPTIC AREA. Silkstone, M.; Brudzynski, S.M. Dept. of Psychology, Brock University, St.Catharines, ON, Canada. Intracerebral injections of carbachol, a muscarinic cholinergic agonist, directly into the anterior hypothalamic-preoptic area of the rat brain are known to induce defensive, 22 kHz vocalizations representing an aversive state. Conversely, intracerebral injection of amphetamine can induce 50 kHz calls, which are associated with appetitive behaviour and signal a positive state. The goal of the present study was to test the hypothesis that dopaminergic and cholinergic systems may interact in the brain in induction of these opposite states. Thus, production of the aversive 22 kHz calls should be antagonized by intracerebral amphetamine. Twelve Long-Evans rats were stereotaxically implanted with chronic cannulae in the anterior hypothalamic-preoptic area for injections of carbachol or amphetamine. Injection of 1 μg of carbachol into the anterior hypothalamic-preoptic area induced repetitively long lasting production of 22 kHz calls with an average peak frequency of 21.7 kHz and average call duration of 920 ms, as compared to control saline injection. Pre-treatment of the same injection site with 7 μg of amphetamine, an average effective dose for production of 50 kHz calls significantly antagonized the carbachol-induced response ($p < 0.02$). Not only the total number of vocalizations was decreased but also the average duration of single calls was reduced by a factor of 5 ($p < 0.005$). The results support the hypothesis that activation of the dopaminergic system in the basal forebrain can significantly attenuate cholinergically-initiated production of 22 kHz. The study was supported by Natural Sciences and Engineering Research Council of Canada.

50. THE EFFECT OF PRENATAL METHAMPHETAMINE EXPOSURE ON DRUG-SEEKING BEHAVIOR OF ADULT MALE RATS. Slamberov, R.; Schutov, B.; Hrub, L.; Pometlov, M. Charles University in Prague, Third Faculty of Medicine, Department of Normal, Pathological and Clinical Physiology, Prague, Czech Republic. Methamphetamine (MA) is one of the most frequently used illicit drugs worldwide and also one of the most common drugs abused by pregnant women. There are studies demonstrating that repeated administration of psychostimulants induces behavioral sensitization in response to treatment of the same drugs in rodents. In addition, studies show that abuse of one drug may increase sensitivity to abuse of another drug, which is called cross-sensitization. There are however only few studies investigating possible sensitizing effect of prenatal MA exposure. Our most recent studies demonstrated that prenatal MA (5 mg/kg) exposure makes adult rats more sensitive to acute injection of the same drug. We were interested whether the increased sensitivity corresponds with the increased drug-seeking behavior. The aim of the present study was to examine the effect of prenatal MA exposure on drug-seeking behavior of adult male rats tested in the Conditioned place preference (CPP). The following psychostimulant drugs were used: MA (5 mg/kg), amphetamine (5 mg/kg), cocaine (10 mg/kg). All psychostimulant drugs induced increased drug-seeking behavior in adult male rats. However, while MA and amphetamine-induced increase in drug-seeking behavior did not differ based on the prenatal drug exposure, prenatally MA-exposed rats displayed tolerance effect to cocaine in adulthood. Thus, our results did not confirm our hypothesis that prenatal MA exposure increases drug-seeking behavior in adulthood in the CPP test. Supported by: GACR 305/09/0126, IGA NS10509-3/2009, MSM 0021620816

51. THE PUTATIVE ROLE OF THE NUCLEUS INCERTUS IN FEEDING BEHAVIOUR OF RATS. Rajkumar, R.; Suri, S.; Lee, L.C.; Dawe, G.S. Department of Pharmacology, Yong Loo Lin School of Medicine, National University Health System and Neurobiology and Ageing Programme, Life Sciences Institute, National University of Singapore, Singapore 117456. The Nucleus Incertus (NI), the chief source of relaxin-3 in the brain, is known to be involved in appetite and stress. We presently sought to investigate its role in the novel environment induced suppression of feeding paradigm in rats. Firstly, rats were stereotaxically implanted with 16 channel microelectrode arrays in the NI and localization confirmed by the waveform characteristics of the neurones recorded. Following an one week rehabilitation period, the implanted rats were fasted for 24 hours and exposed to a novel circular arena with standard laboratory feed presented at the centre. The changes in firing rate of NI neurons and behaviour during a 15-min observation period were simultaneously recorded using a wireless recording system coupled to a behavioural video tracking system. Following offline spike sorting using principal component analysis, a place cell type analysis was done to generate a 2D plot of firing rate in different regions of the arena. Secondly, the NI was selectively lesioned using a CRF-Saporin conjugate and in a separate set of rats, sham lesioning was carried out using blank saporin. NI lesioned and sham-lesioned rats fasted for 24 hrs were subjected to the behavioural paradigm described above. Lastly, the effect of NI stimulation on PVN was studied. The results revealed enhanced NI neuronal firing near the wall of the arena and in the feeding area. Likewise, the NI lesioned rats spent less time in the feed area in comparison to the sham lesioned rats. Electrical stimulation of the NI evoked a field potential in the PVN. These results suggest that perturbation of the NI-RLN-3 system can affect feeding behaviour in stressful conditions. In summary, NI affects feeding behaviour, which might involve the hypothalamic centers.

52. ADULT MEDIAL PREFRONTAL CORTEX AND NUCLEUS ACCUMBENS AMPHETAMINE-INDUCED DOPAMINE RELEASE FOLLOWING ADOLESCENT SOCIAL DEFEAT. Burke, A; Forster, G; Novick, A; Roberts, C; Watt, M. Neuroscience Group, Division of Basic Biomedical Sciences, Sanford School of Medicine, University of South Dakota, Vermillion, SD, USA. Final maturation of mesocorticolimbic dopamine systems occurs during adolescence, and exposure to social stress during this period results in behavioral dysfunction and is correlated with substance abuse disorders. Using Sprague-Dawley rats, we have shown that males exposed to repeated social defeat in adolescence exhibit increased conditioned place preference for amphetamine in adulthood. Given the essential role of nucleus accumbens (NAc) dopamine in amphetamine responses, we investigated the effects of social defeat in adolescence on adult amphetamine-elicited NAc dopamine release. We also examined medial prefrontal cortex (mPFC) dopamine responses to amphetamine, as we had previously found that adolescent social defeat decreased mPFC dopamine content in adulthood, and mPFC dopamine activity is known to mediate behavioral and NAc dopamine responses to amphetamine. Rats were exposed to social defeat once per day for 5 days (P35 to P39), with controls placed in empty novel cages at matched times. In early adulthood (P60), amphetamine (1.0 mg/kg, ip.) or saline injections were administered with locomotor activity observed for 90 min. Two days later, rats were anesthetized, microdialysis probes were implanted into the mPFC and NAc core, and amphetamine-induced dopamine release was measured using HPLC. For rats that were exposed to adolescent social defeat, there was a greater degree of adult amphetamine-induced locomotion, and attenuated amphetamine-induced dopamine release in the mPFC compared to amphetamine-receiving controls. Overall, these data suggest that adolescent social defeat may cause blunted mPFC dopamine responses to amphetamine in adulthood, which may be related to enhanced amphetamine-induced locomotion. Support: NIH P20 RR15567 & NIDA RO1 DA019921.

Social Behavior

53. HIPPOCAMPAL GENE EXPRESSION DURING ESCAPE FROM SOCIAL AGGRESSION IN HAMSTERS Arendt DH; Smith JP; Bastida CC; Prasad M; Rasmussen TL; Summers TR; Delville Y; Summers CH Biology & Neuroscience Group Univ S Dakota, Vermillion, SD 57069; Psychology Univ Texas, Austin, TX 78712 USA. Socially stressful aggressive interactions produce adaptive responses such as submission or escape. In these experiments, a small male Syrian hamster is paired with a larger conspecific male resulting in aggressive social interaction. During social interactions in the Learned Escape model, a hole is available that is only large enough for the smaller test hamster to escape through. Training was carried out over six days; an audible tone (conditioned stimulus = CS) preceded social interaction (larger male is the unconditioned stimulus = US). Following training, hamsters escape in response to the CS alone, however, latency differences suggest that learning is a crucial component of using learned escape behaviors. Animals that escape faster have increased levels of BDNF mRNA in the CA1 region of the hippocampus ($r2 = 0.95$, $P < 0.001$). Conversely, slower escapers exhibit inhibited CA1 BDNF gene expression. Hippocampal BDNF and TrkB expression are also influenced by stress hormone levels, and are dependent on behavioral controllability of stress. Animals that do not have the option of escape (no hole available) show a positive correlation between plasma cortisol concentrations and CA1 BDNF gene expression ($r2 = 0.92$, $P < 0.04$). When the escape hole is available to limit stressful interaction, CA1 BDNF and TrkB mRNA levels increase as cortisol concentrations diminished ($r2 = 0.43$, $P < 0.03$). This inverse relationship is dependent on whether the test animal has the opportunity to escape. Learning to escape transforms the relationship between stress and neurotrophin reactivity, potentially facilitating the expression of adaptive learning. Supported by NIH Grant P20 RR15567 and NSF IBO 0518272 (YD).

54. INTRA-VTA PERTUSSIS TOXIN INFUSIONS STIMULATE MATERNAL BEHAVIOR IN ADULT, NULLIPAROUS FEMALE RATS. Bridges, R.S.; Schoen, M.K.; Carini, L.M.; Gleason, E.D.; Lovelock, D.F.; Byrnes, E.M.; Byrnes, J.J. Department of Biomedical Sciences, Tufts University Cummings School of Veterinary Medicine, North Grafton, MA 01536 USA. The onset of maternal behavior in rodents is regulated in part by forebrain dopaminergic mechanisms. The ventral tegmental area (VTA) and its dopaminergic projections to the nucleus accumbens (NA) and medial prefrontal cortex (mPFC) have been shown to play a key role in this regard. Indeed, intact function of these brain regions is necessary for the onset and expression of normal maternal behavior. Moreover, maternal responding is induced by pharmacological stimulation of NA dopamine signaling. These and other findings suggest that enhancement of dopaminergic transmission in the mesocorticolimbic dopamine system can stimulate maternal activity. The current study was undertaken to directly test this hypothesis in a pup-induced, virgin model. Nulliparous female rats were stereotaxically infused with pertussis toxin (PTX; 0, 0.1, or 0.3 g/hemisphere) into the VTA to stimulate the activity of dopaminergic projection neurons. After 3 days of recovery, maternal responding to donor pups was tested daily and latency (in days) to full maternal behavior was recorded. Intra-VTA PTX treatment produced a dose-dependent decrease in maternal behavior latency, and a long-lasting increase in locomotor activity. These effects were associated with significantly decreased dopamine utilization and moderately increased D2 receptor mRNA expression in the mPFC. In the NA, moderate decreases in dopamine D1 receptor mRNA expression were also observed. The findings suggest that enhanced VTA activity stimulates the onset of maternal behavior in rats, perhaps by increasing the motivational aspects of pup stimuli. This effect may involve adaptive responses in both the NA and the mPFC. (Supported by NIH Grant HD19789 awarded to RSB)

55. SOCIAL DECISION MAKING DRIVES BEHAVIORAL AND NEURAL PLASTICITY DURING LEARNED ESCAPE Rasmussen,TL; Summers, TR; Carpenter, RE; Smith, JP; Arendt, DH; Summers, CH Biology & Neuroscience Group Univ S Dakota, Vermillion, SD 57069; Biology Stanford Univ, Stanford, CA 94305 USA. A model of fear conditioning that utilizes social aggression from a large conspecific as the unconditioned stimulus and an opening for escape, provides the opportunity to determine the molecular and behavioral constituents of social decision making. Rainbow trout dependably either escape or remain submissively on presentation of new US stimuli during training. Rapidly diminishing latency to escape is accompanied by complex learning process of 7 steps; the most important of which is social learning regarding the large opponent. Reduction in escape latency is possible when the test animal learns the patrolling pattern of the dominant territorial fish, and escape attempts are limited to occasions when the dominant fish is not watching. This social learning is accompanied by dramatic and rapid upregulation of BDNF gene expression. Fish that do not escape exhibit a similar but significantly reduced increase in hippocampal BDNF mRNA in response to presentation of the conditioned stimulus (tank inflow off) alone. Treatment with the CRF1 receptor antagonist during training induces non-escaping fish to begin escaping. Glutamate AMPA receptor subunit GluR1 (but not GluR2) gene transcription in the hippocampus, which coincides with other conditioned responses, is inhibited by social defeat. While aggressive behavior is inhibited and submissiveness more evident, the conditioned stimulus also produces (after training) increased plasma cortisol, as well as increased serotonin and dopamine activity in limbic and hypothalamic nuclei. Social decision making,

resulting in either complex social escape behavior or social submission is accompanied by neurotrophic activity along with neural and behavioral plasticity. Supported by NIH Grant P20 RR15567 and NSF DDIG IOS 0906691.

56. ANXIETY IS ALLEVIATED BY ESCAPE FROM SOCIAL AGGRESSION IN HAMSTERS Smith JP; Arendt DH; Bastida CC; Rasmussen TL; Summers TR; Delville Y; Summers CH Biology & Neuroscience Group Univ S Dakota, Vermillion, SD 57069; Psychology Univ Texas, Austin, TX 78712 USA. Complex emotional components influence learning strategies developed during aggressive interactions that produce adaptive responses. Social responses to the anxiety induced by aggressiveness include conditioned submission, and Learned Escape. Either response is potentially valuable for reducing social stress, by limiting the duration or magnitude of aggression. These responses are exhibited by a wide variety of vertebrate species. In these experiments, a small male Syrian hamster is paired with a larger conspecific male resulting in aggressive social interaction. During social interactions in the Learned Escape model, a hole is available that is only large enough for the smaller test hamster to escape through. Training was carried out over six days; an audible tone (conditioned stimulus = CS) preceded social interaction (larger male is the unconditioned stimulus = US). Latency to escape dramatically decreased over time, as has been demonstrated in fish using this model. Following training, hamsters escape in response to the CS alone, however, latency differences suggest that learning is a crucial component of using learned escape behaviors. Hippocampal BDNF and TrkB receptor expression appears to explain the capacity of hamsters to escape from aggressive interactions. The aptitude for rapid escape and hippocampal regulation thereof are likely to be influenced by anxiety and amygdalar activity. The availability of an escape route influences both anxiety and aggression during social interaction. Utilization of Learned Escape appears to transform the relationship between stress and neurotrophin reactivity, potentially facilitating the expression of adaptive learning. Supported by NIH Grant P20 RR15567 and NSF IBO 0518272 (YD).

57. A NOVEL ANIMAL MODEL FOR STUDIES IN AGGRESSION AND PATERNAL STRESS. Ten Eyck, G.R. Department of Pharmaceutical Sciences, College of Pharmacy, University of Hawaii Hilo, HI 96720. A novel animal model has been developed for studies in aggressive behavior and paternal care. The Puerto Rican coqu frog, Eleutherodactylus coqui (Leptodactylidae), is a frog that has two evolutionarily derived characters that interestingly make it an ideal candidate for biomedical studies. First, this amphibian undergoes direct development; the free swimming tadpole stage characterized by most frogs has been eliminated and frogs develop directly into the adult phenotype. Secondly, this frog displays paternal care whereby the male broods and aggressively defends the developing eggs/embryos and hatched froglets 3-5 days following hatching. Using this comparative model, studies can be carried out in both laboratory and field environments. This animal displays a relatively complex social organization; males can be territorial, non-territorial, and/or paternal, and radiate auditory signals that communicate to both males and females. This model has been excellent for determining neuroendocrine mechanisms controlling aggressive, territorial behaviors and has provided insight in why some males are more aggressive than others. It has also demonstrated to be a rigorous biological system to test hypotheses on both the behavioral and neuroendocrine effects of stress during paternal care. Using this model, paternal males are exposed to a natural, biological stress, conspecific advertisement calls (since other males are the chief predator on eggs/embryos), whereby both brain and behavior can be examined. Investigations on this animals reproductive and social behaviors are additionally significant since the Puerto Rican coqu was recently introduced on the Island of Hawaii and its widespread distribution is a major environmental and economical concern.

58. PAIR BONDING IN THE BLACK-PENCILLED MARMOSET (CALLITHRIX PENICILLATA): BEHAVIORAL CHARACTERISTICS. 1Birnie, A.K.; 1Smith, A.S.; 1,2French, J.A.; 3Agmo, A. 1Department of Psychology and Callitrichid Research Facility and 2Department of Biology, University of Nebraska at Omaha, USA, 3Department of Psychology, University of Tromso, Norway. The aim of the present study was to describe how the development of a pair bond modifies social, sexual and aggressive behavior in the marmoset. To that end, heterosexual pairs were formed at the beginning of the study. The members of the pair were unknown to each other. At the onset of pairing, social, sexual, exploratory and aggressive behaviors were recorded by experienced observers for 40 min. The animals were then observed for 20 min, both in the morning and afternoon, for 21 days. The frequency and/or duration of behaviors recorded on Day 1 were compared to those recorded at later observations. The behavior displayed shortly after pairing should be completely unaffected by the pair bond, while such a bond should be present at later observations. In that way, it was possible to determine how the behavior between the pair was modified by the presence, or at least the development, of a pair bond. It was found that social behaviors such as allogrooming and huddling increased from Day 1 to Day 26 and all subsequent days observed. Conversely, other behaviors, such as open mouth displays, had a high frequency during the early part of cohabitation but declined towards the end. Consequently, the mental state of pair bonding manifests itself in an increased intensity of social behaviors. It is suggested that the intrinsically rewarding properties of grooming and perhaps other social behaviors turn the pair mate into a positive incentive, activating approach and further commerce when possible. Thus, the pair

bond is nothing more than a motivational state activated by the conditioned incentive properties of the partner. This notion can explain all kinds of pair bonds, including those occurring between individuals of the same sex and in promiscuous species.

59. EFFECTS OF CHRONIC ERβ AGONIST DPN ON A SOCIALLY TRANSMITTED FOOD PREFERENCE IN OVARIECTOMIZED CD1 MICE.. Clipperton-Allen, A.E.1, Mikloska, K.V.2, Roussel, V.R.1, Ying, H.L.2, Choleris, E.1 1Dept Psychology, 2Dept Biological Sciences, University of Guelph, Guelph, Ontario, Canada N1G 2W1. Social living allows for learning from conspecifics, among other advantages, which reduces reliance on risky trial-and-error learning. This is examined in the social transmission of food preferences (STFP) paradigm, in which observer mice are able to expand its food repertoire to include novel food smelled on the breath of demonstrators during a social interaction. We have previously shown that estrogens are involved in the STFP. Chronic administration of estradiol benzoate (EB) and acute pre-acquisition administration of EB or estrogen receptor beta (ERb) agonist prolonged the preference for the demonstrated food, while acute administration of ERa agonist blocked this preference. It is unknown how chronic application of ERa or ERb agonist will affect this paradigm. The current study focused on the latter, implanting ovariectomized (ovx) observer mice with Silastic capsules containing sesame oil vehicle or ERb agonist DPN (3g, 6g, 12g, or 24g per capsule) 9-17 days prior to testing. Observers interacted with demonstrators that had just consumed either cocoa- or cinnamon-flavoured rodent chow, and then underwent an 8h choice test where the two flavours (both novel to the observer) were continuously available. Preliminary results suggest that chronic DPN treatment prolonged the observers preference for the demonstrated food in a similar manner to acute and chronic EB and to acute pre-acquisition administration of an ERb agonist, suggesting similar mechanisms in the acute and chronic involvement of estrogens and their receptors in social learning. Supported by NSERC.

60. NEONATALLY SEROTONIN DEPLETED MICE SHOW EARLY DEFICITS IN SOCIAL BEHAVIOR Ayorinde, M., *Blue, M.E. and Hohmann, C.F. Department of Biology Morgan State University and the *Hugo W. Moser Research Institute at Kennedy Krieger, Inc., Baltimore, MD, USA Autism Spectrum Disorder (ASD) is a developmental brain disorder associated with deficits in social and cognitive behaviors, affecting many individuals. Diagnosis is still on the rise. Serotonin (5-HT) is a neurotransmitter, involved in the growth and plasticity of the brain. Studies suggest that abnormal levels of serotonin may play a key role in the pathophysiology of autism. Our lab has developed a mouse model of selective serotonin-depletions in neocortex and hippocampus, using 5,7-Dihydroxytriptamine (5,7-DHT). This induces, in mature mice, brain and behavioral changes similar to ASD. At one week postnatal (PND7), lesioned mice also show altered cortical volume. We are investigating here, if social deficits can be seen at PND7 as well. Male and female BALB/cByJ mice, from nine litters, received injections of 5,7-DHT (n=15) or vehicle (n=20) into the medial forebrain bundle at birth and were returned to the dam. Age matched controls (AMC, n=23) stayed with their mothers until behavioral testing. At PND 7, all pups were tested on a Homing Task. This task assessed whether the pups show preference for home bedding, versus clean bedding, as an indicator of their normal social behavior. We show that AMC and vehicle pups have significant preference for home bedding, when compared to clean bedding. In contrast, 5,7-DHT mice did not show a significant preference for home bedding. Peppermint scent was found to be aversive to all treatment groups. These data suggest that decreased levels of serotonin in cortical regions, during crucial stages of development, result in early postnatal deficits in social behavior. Supported by: U54MH66417, SO6 GM051971 and 2T34GM007977-23A2.

61. IDENTIFYING THE ROLE OF SEROTONIN IN AUTISM-LIKE BEHAVIOR IN JUVENILE MICE Lewter, L, Hohmann, C.F., and *Blue, M.E. Department of Biology, Morgan State University and the *Hugo W. Moser Research Institute at Kennedy Krieger, Inc., Baltimore, MD, USA. Autism Spectrum Disorder (ASD) is a fast-growing developmental disability, characterized by impaired social interaction and restricted and repetitive behavior. Studies suggest that serotonin (5-HT) plays a key role in the pathophysiology of ASD. Our lab has developed a mouse model of 5-HT depletion in the brain, using neonatal injections of the selective neurotoxin 5,7-DHT into the medial forebrain bundle. Previous results have shown that 5,7-DHT lesioned mice have altered social behaviors, increased anxiety, and altered sensory responsiveness, compared to vehicle injected (Veh) and age-matched control mice (AMC), when tested as adults. Deficits in social cognition, along with altered brain volume, were also seen in mice at PND7. We hypothesize, that 5,7-DHT lesioned mice will continue to show altered social behaviors when tested as juveniles (PND21-PND34). Three litters of AMC (n=15), Veh (n=12), and 5,7-DHT mice (n=10) were tested in a Play Behavior Task. Videotapes were analyzed using CleverSys (Top Scan Version 1.00) software to quantify investigative & affiliative social interactions and non-social interactions, including exploratory & repetitive behaviors. Statistically analyses were conducted using ANOVA (GraphPadPrizm). We show decreased play initiation and affiliative behavior in 5,7-DHT lesioned mice, compared to both AMC and Veh, along with increased investigative/exploratory activity in Veh mice. No significance differences between males and females

were seen. This data suggest that social behaviors in the serotonin depleted, juvenile mice continues to be selectively impaired. Supported by SO6 GM51771, U54MH66417, and 5R25GM058904.

62. NEONATAL BLOCKADE OF GASTRIN RELEASING PEPTIDE RECEPTORS AS AN ANIMAL MODEL OF AUTISM. Johnstone, J.; Mackay. J.C.; Du, L.; Kent, P; Merali, Z. Dept. of Psychology. The University of Ottawa, Ottawa ON K1N 6N5 Canada. Autism spectrum disorders (ASD) are characterized by impairments in communication, learning, social interactions, and stereotyped repetitive behavior. The specific causes of ASD are currently unknown, but evidence suggests a strong genetic component. In this vein, mutations of the gastrin-releasing peptide receptor (GRPr) gene have been shown to be associated with ASD. Developing a rodent model of ASD with altered GRPr function, may help to further our understanding of the role of this receptor subtype in the neurobiology of ASD. We have recently shown that rats treated neonatally with the GRPr antagonist, RC-3095, showed deficits in communication, reduced sociability and enhanced learned fear response compared to vehicle-treated rats. The objective of the present study was to assess the neurochemical consequences of this neonatal insult by measuring the mRNA expression of peptides/neurotransmitters and their respective receptors at brain regions relevant to ASD. From postnatal (PD) days 1-10, male Wistar rat pups (n=8-10/litter) received subcutaneous injections of RC-3095 (1 mg/kg) or saline twice daily. Social interaction was assessed on PD 35 and then animals were sacrificed 24 hours later and brains were harvested and flash frozen. Quantitative PCR analysis revealed a significant decrease in mRNA expression of GRP, a structurally related peptide, neuromedin B (NMB), and several GABAa receptor subunits at the amygdala in rats treated neonatally with RC-3095. Similarly, at the medial prefrontal cortex (mPFC) neonatal RC-3095 treatment decreased mRNA expression of NMB, NMB receptor, and GABAa-a3, b3 and GABAb-R1 subunits. These neurochemical findings are consistent with those observed in individuals with ASD providing further support for neonatal GRPr blockade as an animal model of ASD.

63. ATYPICAL ULTRASONIC VOCALIZATIONS IN A MOUSE MODEL OF DOWN SYNDROME. Pearson, J.N.; Fernandez, F.; Costa, A.C.S. Neuroscience Program, Division of Clinical Pharmacology and Toxicology. Univ. Colorado Denver, SOM, Aurora, CO 80045 USA. Individuals with Down syndrome (DS) show delays in speech production and language acquisition. Roughly 3% of those with DS remain non-verbal into their teenage years. Ultrasonic vocalizations (USVs) in rodents are a primitive form of communication observed throughout the animals lifespan. The aim of the current study was to determine whether male Ts65Dn mice (TS) exhibit altered USVs compared to euploid control mice (CT) and, therefore, whether TS might be useful as an animal model of speech production deficits seen in persons with DS. Accordingly, we acclimated 12 TS and 12 CT (8 weeks old) to a sound attenuating chamber for three consecutive nights. On the fourth night, each male was paired for five minutes with a nulliparious female to stimulate USVs. USVs were recorded, digitized, and analyzed using a specialized high frequency microphone, data acquisition system, and software. A significantly greater proportion of CT than TS vocalized during testing [10/12 vs. 5/12; $\chi 2 = 10.20$, $p = 0.03$]. The quality of the USVs was also significantly different between groups. TS exhibited USVs with higher energy [$t(1, 12) = 5.140$, $p < 0.01$], peak to peak frequency [$t(1,12) = 2.915$, $p < 0.05$], and peak amplitude [$t(1,11) = 2.904$, $p < 0.05$] compared to CT. Taken together, these data indicate that TS vocalizations are both quantitatively and qualitatively different from CT. To our knowledge, this is the first systematic characterization of USVs in this mouse strain. By elucidating altered USVs in these aneuploid mice, we have added another element to the list of available phenotypes that capture components of the human genetic disorder.

candesartan and atorvastatin and their combination against ischemia reperfusion induced behavioral and biochemical alterations in rats.

65. THE 5-HT1A RECEPTOR CONTRIBUTES SUBSTANTIALLY TO THE EFFECTS OF INDOLEALKYLAMINE HALLUCINOGENS ON LOCOMOTOR ACTIVITY AND INVESTIGATORY BEHAVIOR IN MICE. Halberstadt, A.L.; Geyer, M.A. Dept. Psychiatry, Univ. California San Diego, La Jolla, CA 92093-0804 USA. Indolealkylamine hallucinogens are nonselective agonists at 5-HT1A and 5-HT2A receptors. There is extensive evidence, from both animal and human studies, that the characteristic effects of hallucinogens are mediated by interactions with the 5-HT2A receptor. Nevertheless, there is increasing recognition that the 5-HT1A receptor also contributes to the behavioral effects of the indolealkylamine hallucinogens. In the present investigation, we tested whether the 5-HT1A receptor is involved in mediating the effects of the indolealkylamine hallucinogens 5-methoxy-N,N-dimethyltryptamine (5-MeO-DMT) and N,N-dipropyltryptamine (DPT) on locomotor and investigatory behavior in mice in the behavioral pattern monitor (BPM). When tested in C57BL/6J mice, 5-MeO-DMT (10 and 20 mg/kg) and DPT (0.3-30 mg/kg) reduced locomotor activity, rearing, and holepoking, and increased spatial d, a measure of the complexity of locomotor paths. Importantly, the behavioral effects of 5-MeO-DMT and DPT were markedly reduced in 5-HT1A receptor knockout mice on a C57 background. The prototypical 5-HT1A agonist 8-hydroxy-2-(dipropylamino)tetralin (8-OH-DPAT; 1 mg/kg) also reduced locomotor activity, rearing, and holepoking, and increased spatial d, behavioral effects that were significantly attenuated when 8-OH-DPAT was tested in 5-HT1A receptor knockout mice. These findings indicate that the effects of 5-MeO-DMT and DPT on locomotor activity and investigatory behavior in mice are mediated primarily by activation of the 5-HT1A receptor. Although it is generally accepted that most of the effects of hallucinogens are mediated by the 5-HT2A receptor, these findings demonstrate that interactions with the 5-HT1A receptor also contribute to the behavioral effects of indolealkylamine hallucinogens. Acknowledgements: This project was supported by the National Institute on Drug Abuse (DA002925 and DA025412).

66. TOXOPLASMA GONDII MOUSE MODEL OF SCHIZOPHRENIA-LIKE NEUROBEHAVIORAL ABNORMALITIES IN MICE: GENDER-RELTAED EFFECTS AND MOLECULAR CORRELATES. Kannan, G.1,2,3; Xiao, J-C4; Krasnova, I.N.5; Cadet, J.5; Yolken, R.4; Jones-Brando, L.4; Pletnikov, M.V.2,6,7,3 1Baltimore, MD; 2Dept. of Psychiatry and Behavioral Sciences, Johns Hopkins Univ. Sch. of Med., Baltimore, MD; 3Cell. and Mol. Med. Program, Johns Hopkins Sch. of Med., Baltimore, MD; 4Stanley Div. of Developmental Neurovirology, Johns Hopkins Univ. Sch. of Med., Baltimore, MD; 5Natl. Inst. of Drug Abuse, Natl. Inst. of Hlth., Baltimore, MD; 6Dept. of Neuroscience, Johns Hopkins Sch. of Med., Baltimore, MD; 7Dept. of Mol. and Comparitive Pathobiology, Johns Hopkins Sch. of Med., Baltimore, MD. Toxoplasma gondii (T. gondii) infection is implicated in the development of schizophrenia. We wished to determine the contribution of the parasite to behavioral differences seen between men and women with the disease. To do so, we evaluated the effects of one strain of Type II T. gondii, Prugniaud (PRU), on mouse behavior and cortical gene expression in male and female Balb/C mice. We found gender differences in cat odor attraction, a T. gondii- specific manipulation. Female infected mice exhibited more attraction to feline odor than uninfected did. In contrast, male control mice showed more attraction to feline odor than infected males. Furthermore, infected females show increased hyperactivity while infected males show decreased hyperactivity, as compared with controls. No changes in pre-pulse inhibition were observed. No differences in parasite antibody titres were seen between PRU- infected males and females. Microarray analysis revealed differential expression in dopamine receptors between males and females, control and infected. As the neurotransmitter dopamine and its corresponding receptors are associated with schizophrenia, it is possible modulation of these receptors is a mechanism by which T. gondii causes schizophrenia-like behavior.

8:30-10:45 **Symposium 5:** THE USE OF ANIMAL MODELS TO UNDERSTAND MECHANISMS UNDERLYING ENVIRONMENTAL IMPACT ON BRAIN DEVELOPMENT. Co-Chairs: **F. Scott Hall and Susan L. Andersen**

THE EFFECTS OF ISOLATION-REARING AND AMITRIPTYLINE ON GENE EXPRESSION IN THE HIPPOCAMPUS: CAN GENE EXPRESSION STUDIES HELP REVEAL THE UNDERLYING MECHANISMS OF GENE-ENVIRONMENT INTERACTIONS? Hall, F.S.1; Cole, S.W.2; Andrews, A.M3; Knutson, B.4 1Molec. Neurobiol. Branch, NIDA-IRP; 2 Div. Hematology-Oncology, UCLA School Med; 3Penn. State Neurosci. Inst., Penn. State Univ.; 4Depts. Psychol. Neurosci., Stanford Univ. Social isolation, particularly early in life, produces permanent changes in brain function and behavior. The mechanisms underlying these (often) permanent phenotypic changes are largely unknown, but deprivation of different types of social interactions, at different ages, has different consequences. Isolation-rearing produces a syndrome of behavioral and neurochemical effects associated primarily with dopaminergic and serotonergic systems, which model an array of psychiatric disorders, including depression. Chronic antidepressant administration reverses many of these effects, but the underlying mechanisms of both isolation-rearing, and the reversal by antidepressant treatment, are unknown. The permanency of these changes suggests the involvement of long-term changes in gene expression. Sprague-Dawley rats were raised under social or isolated conditions from weaning; half of each group was treated with amitriptyline for 8 weeks and subsequently tested for spontaneous locomotor activity and brain samples were taken for determination of monoamine levels and examination of gene expression. Isolation-rearing resulted in changes in hippocampal serotonin that were reversed by chronic antidepressant treatment. Gene expression changes were also noted in several serotonergic genes in the hippocampus. In addition, numerous genes associated with neurotrophin signaling, cellular stress and apoptosis were also induced in the hippocampus. Some of these changes were also reversed by antidepressant treatment. Analysis of this gene expression data suggests a way to examine the underlying genetic mechanisms mediating gene-environment interactions of isolation-rearing, as well as the mechanism of the antidepressant response, and may be applied other types of early life manipulations that produce permanent phenotypic changes. (Support: NIDA-IRP)

RILUZOLE AND FLUOXETINE MODULATE THE EFFECTS OF MATERNAL SEPARATION ON DEPRESSIVE BEHAVIOR IN A SEX-DEPENDENT MANNER. Andersen SL; Vaccaro K; Thompson BS; Freund N. Laboratory for Developmental Neuropharmacology. McLean Hospital/Harvard Medical School, 115 Mill Street, Belmont, MA 02478 USA. Exposure to stressful experiences early in development is associated with elevated risk for depression. Depressive symptoms do not manifest immediately, but rather emerge during adolescence and significantly earlier than the general population. To investigate the underlying mechanisms associated with vulnerability to depression and its potential reversal, male and female rats were placed individually into cups kept at nest temperature for 4 hours/day between postnatal day 9 (P9) and 16. This period of deprivation is unique for two reasons: 1) maternal separation typically is initiated at P1 or 2, not P9; and 2) this period has been associated with increasing depressive-like symptoms following drug exposure. During this same period, subjects were exposed to a saline vehicle, riluzole (4 mg/kg; which reduces glutamate activity), or fluoxetine (10 mg/kg, an SSRI). Subjects were tested for learned helplessness with the triadic model. The triadic model allows for the further analysis of stressor controllability in response to an escapable shock (ES) or an inescapable shock (IS) in an active avoidance paradigm. Sex differences in sensitivity to the deprivation manipulation and the effects of simultaneous drug exposure were observed in control and deprived animals. First, females had more failures to escape and longer latency to escape than males. Second, maternal deprivation decreased depressive-like behavior in females, but did not affect male behavior. However, fluoxetine exposure increased depressive-like behavior in male, but not female, controls; fluoxetine co-treatment reduced depressive-like behavior in males exposed to deprivation. Riluzole reduced escape behavior in the IS condition in control males, but was without effect in deprived males. The results of this study confirm that sex differences exist in depressive-like behavior. More importantly, they suggest that treatment effects interact with the prior history of the subjects and the degree of stressor controllability. Specifically, rats that were exposed to a stressor that produced minimal effects on depressive-like behavior by itself, sensitizes drug responsiveness in ways that are not predicted based on identical drug exposure in normal animals.

THE EFFECTS OF POST-WEANING SOCIAL ISOLATION ON SEROTONERGIC SYSTEMS AND BEHAVIOR. Lukkes, Jodi L.; Lowry, Christopher A.; University of Colorado, Boulder, CO, USA. Exposure to stressful experiences, such as social isolation, during adolescence can contribute to vulnerability to stress-related psychiatric disorders during adulthood. My previous studies have shown that post-weaning social isolation in male rats causes an up-regulation of corticotropin-releasing factor (CRF) type 2 receptor levels in the dorsal raphe nucleus (DR), alters CRF-mediated serotonin release in the nucleus accumbens (NAc), and increases social anxiety-like and fear behavior in adulthood, which can be attenuated with antagonism of CRF type 2 receptors in the DR. These findings suggest that the social isolation-induced alterations in fear, anxiety, and stress-related behavioral responses could be due to sensitization of stress-related, CRF-dependent activation of a DR-NAc serotonergic circuit. Therefore, we examined how post-weaning social isolation, in combination with a subsequent

stressor such as social defeat in males or a challenge with the anxiogenic drug, N-methyl-beta-carboline-3-carboxamide (FG-7142) in females, affects c-Fos expression in topographically organized subpopulations of serotonergic neurons in the DR in adulthood using dual immunohistochemical staining for c-Fos and tryptophan hydroxylase. Post-weaning social isolation sensitized female rats to anxiogenic drug-induced increases in c-Fos expression in serotonergic neurons in the DR. Furthermore, post-weaning social isolation increased anxiety and promoted a reactive emotional coping style in male rats. These data suggest that post-weaning social isolation alters the effects of stress-related stimuli on serotonergic systems, which have been implicated in the pathophysiology of stress-related neuropsychiatric disorders. Acknowledgements: The project described was supported by Award Numbers F32MH084463 (JLL) and R01MH086539 (CAL) from the NIMH. C.A. Lowry was supported by a 2007 NARSAD Young Investigator Award and is currently supported by an NSF CAREER Award (NSF-IOS #0845550).

SYNERGISTIC INTERACTIONS BETWEEN MILD PRENATAL IMMUNE CHALLENGE AND PERI-PUBERTAL STRESS IN THE DISRUPTION OF ADULT BEHAVIORAL FUNCTIONS RELEVANT TO SCHIZOPHRENIA. Joram Feldon1, Sandra Giovanoli1, Urs Meyer1 1Laboratory of Behavioural Neurobiology, Swiss Federal Institute of Technology (ETH) Zurich, Schorenstrasse 16, 8603 Schwerzenbach, Switzerland. Converging evidence from human epidemiological studies and parallel experimental investigations in animals indicates that prenatal exposure to infection may be a relevant environmental risk factor for schizophrenia and related disorders. However, if prenatal infection does indeed play a significant role in the etiology of schizophrenia, then it likely does so by interacting with other genetic and/or environmental susceptibility factors. Besides prenatal infection, exposure to stressful situations in peri-pubertal stages of life has been repeatedly suggested to represent a significant postnatal environmental factor in the development of psychotic disorders. Against this background, the present study was designed to test the hypothesis whether prenatal viral-like immune challenge may synergistically interact with peri-pubertal stress to facilitate the emergence of schizophrenia-like behavioral abnormalities in adulthood. For these purposes, we combined a well established mouse model of prenatal (gestation day 9) immune challenge by the viral mimic Poly[I:C] (=polyriboinosinic-polyribocytidilic acid, a synthetic analogue of virus-specific double-stranded RNA) with a model of exposure to peri-pubertal stress induced by a sub-chronic variable stress protocol applied in peri-puberty (postnatal days 30 to 40). We found that peri-pubertal stress led to significant behavioral and pharmacological abnormalities specifically in animals which had been subjected to prenatal Poly[I:C]-induced immune challenge at low intensity (1 mg/kg, i.v). These alterations included impairments in sensorimotor gating in the form of prepulse inhibition (PPI) disruption, cognitive deficits in the form of reversal learning impairment, and potentiated sensitivity to the psychotomimetic drugs amphetamine and dizocilpine (MK-801). Importantly, neither prenatal Poly[I:C] treatment at the chosen dose alone nor peri-pubertal stress alone induced such behavioral abnormalities. Hence, our initial experimental research supports the biological plausibility for synergistic interactions between prenatal immune challenge and postnatal stress in the precipitation of brain dysfunctions relevant to schizophrenia. In accordance with an environmental two-hit model of schizophrenia etiology, prenatal immune challenge may render the brain more vulnerable to postnatal stress, thereby facilitating the development of full-blown psychotic disturbances associated with schizophrenia.

11:15-12:15 ***Professional Journeys Series*** **Donald G. Stein**, Emory University School of Medicine. *Progesterone and Brain Injury: From Bench to Bench to Bench to Bench to Bench to Bedside*

PROGESTERONE AND BRAIN INJURY: FROM BENCH TO BENCH TO BENCH TO BENCH TO BEDSIDE. Donald G. Stein. Emory University. I've been involved in recovery of function research for all of my professional career. I started this work at a time when it was forcefully taught that once the adult brain was damaged there was no possibility of true repair. Any improvements in function were seen as merely tricks of compensation. Once a structure was damaged or eliminated, its functions were lost permanently. Unfortunately for me, this is not what I saw when I examined the role of the hippocampus in learning and memorytoo many of my rats performed much better than would be expected following bilateral removals of this structure. What got me started was not a search for a treatment at all, but rather trying to explain why localization of function was not working as planned! It was only much later, after reading case studies suggesting that women recovered better than men after brain injury, that I became interested in sex hormones as a potential treatment for brain injury. When we suggested testing progesterone in TBI, most colleagues thought it was nuts! It was hard enough just to convince relatively unindoctrinated graduate students that it was an area worth considering. It has been a long trail of ups and downs that taught me (and my students) a lot about grantsmanship, paradigms in science, and the politics and sociology of medicine and big pharma. I will discuss some of these issues as they relate to my experience in finding a successful treatment for traumatic brain injury in patients. This work has now led to spin-offs with potential for benefit in stroke and other diseases. This tale of a long, fraught journey to bring lab research to real-world application may not resonate with every scientist, but I believe many will recognize the pattern and I hope some researchers will be encouraged.

3:30-5:00 **Oral Session 2:** Chairperson: **Wim Crusio**

SKYSCRAPERS AND HAYLOFTS: AN EXPLORATION OF DIFFERENTIAL HOUSING IN LONG-EVANS RATS Franssen, CL1, Kaufman, C1, Bardi, M2, Lambert, KG1. 1Department of Psychology, Randolph-Macon College, Ashland, VA 23005 USA; 2Psychology Department, Marshall University, Huntington, WV 25755. Although there is a long history of utilizing environmental enrichments to alter cognitive and emotional responses of rodents, few studies have assessed the characteristics of these enrichments as they relate to the natural world. In this study we assessed emotionality of rodents housed in one of four conditions. Each housing condition was the same size and contained bedding, a place to hide, items to climb on, items to manipulate, and items to chew. The bedding and materials were different between cages, however. In the naturally-enriched habitat, bedding was dirt or mulch, and enrichment items were wood, rock, and other materials gathered from the natural world. The artificially-enriched habitat contained manufactured items made from plastics and rubber. The natural/artificial blended cage contained items from both of these sources. The control cage was matched in size, but contained no enrichment. Rats were tested on a Novel Object Preference Test, a Cricket-Hunting Test, and a Forced Swim Test to assess cognition, foraging and emotionality, respectively. Both of the groups exposed to some degree of natural elements showed behavioral indications that they were bolder and more exploratory; for example, rats housed in the natural habitat had a shorter latency to dive and a trend toward more dives in the swim test, and rats in the natural/artificial blended habitat killed and ate a cricket much faster. Analysis of fecal samples for stress-related hormones revealed that rats housed in the naturally enriched habitat had a significantly higher ratio of dehydroepiandrosterone (DHEA) to corticosterone, suggesting they may have an enhanced resilience to stress. In an initial assessment of neural effects, rats housed in the naturally-enriched habitat had an increase in GFAP immunoreactivity in the dentate gyrus of the hippocampus. These data suggest that the materials which constitute environmental enrichment, namely natural versus artificial, may have significant impacts on the neurobiology of an animal.

ANTERIOR OLFACTORY NUCLEUS SUPPRESSES IPSILATERAL AMYGDALA IN SOCIAL BUFFERING OF CONDITIONED FEAR RESPONSES. Kiyokawa, Y.; Takeuchi, Y.; Mori, Y. Laboratory of Veterinary Ethology, The University of Tokyo, Tokyo 113-8657 JAPAN. In "social buffering", a phenomenon known in various species, stress responses are less distinct when an animal is exposed to a stressor with one or more conspecific animals. We have previously reported that the presence of an associate rat mitigates conditioned fear responses to an auditory conditioned stimulus (CS) via the main olfactory system in male rats. In addition, bilateral lesion of the anterior olfactory nucleus posterior part (AOP) blocked this social buffering. In order to clarify the role of the AOP in the social buffering, we investigated the connectivity between the AOP and the amygdala, the key site for the conditioned responses. The unilateral AOP and either ipsilateral (Ipsilateral) or contralateral (Contralateral) amygdala of the subject was lesioned five days before the conditioning. We also prepared the subject with unilateral lesion of amygdala as a control group (Control). Twenty-four hours after the conditioning, the subjects were re-exposed to the CS with an associate. Control group showed the blockade of freezing to the CS as seen in the previous studies. Whereas Ipsilateral group showed the social buffering phenomenon, Contralateral group showed conditioned fear responses to the CS, i.e., social buffering was blocked. These results suggest that the AOP is an important relay station functionally connecting the olfactory information to the amygdala in social buffering.

NITRIC OXIDE PRODUCING NEURONS IN THE DORSAL RAPHE NUCLEUS ARE ACTIVATED BY RESTRAINT STRESS IN THE WAKING RAT. Vasudeva, R. K.; Waterhouse, B. D. Dept. Neurobiology & Anat. Drexel University College of Medicine, Philadelphia, PA 19129 USA. Afflicting one fifth of US adults, anxiety is a prevalent disorder in our society. Investigations have focused on the serotonergic (5HT) modulation of stress circuitry in the brain, and therapeutics targeting this system tend to be effective treatments. One of the main components of the 5HT system, the dorsal raphe nucleus (DRN), has global projections to forebrain and stress-related regions. This nucleus is not solely serotonergic, and has a significant contingent of nitric oxide producing cells that co-localize or intermingle with 5HT. This unique relationship creates neurochemically distinct subregions in the rostral (5HT only) and caudal (nitric oxide synthase-NOS only) lateral wing (rLW, cLW) of the DRN. Recent investigations (Okere & Waterhouse, 2006) showed anatomical activation of cLW cells via increased staining intensity for NOS following restraint stress, a known psychological stressor. The main focus of the current work is to examine the electrophysiological output of these cells during stressor exposure in the conscious animal. We hypothesized that the apparent anatomical activation of NOS cells in the cLW during restraint will be represented in the form of increased electrophysiological output of this region. Male long evans rats were implanted with chronic electrode bundles in rLW (control) and cLW. Animals were videotaped during baseline behaviors, four hours of restraint stress, and two hours post-restraint. Results demonstrate a behavior-dependant discharge of cells during non-stressed conditions in both recording locations. Restraint stress produced an initial increased discharge in cLW that was absent in rLW, supporting our previous results. The time course of cell responding across the restraint period differed for both subregions and may be dependant on additional behaviors, such as chattering or struggling. These data suggest that increased local release of NO within anatomically discrete subregions of the DRN during stressor exposure. Furthermore, these results prompt us to speculate the role of NO in regulating local DRN function and the operation of circuits within cLW efferent targets. With

future studies we expect to gain insight into the functional mechanisms of current therapeutic treatments for anxiety and the role that NO has in this disorder.

CHRONIC STRESS MODULATES MICROGLIAL-NEURONAL INTERACTIONS IN PREFRONTAL CORTEX: IMPLICATIONS FOR DEVELOPMENT OF DEPRESSION. Walker, F.R.; Tynan, R.; Ng, A.; Nalivaiko, E.; Day, T.A. School of Biomedical Sciences and Pharmacy, University of Newcastle, and the Hunter Medical Research Institute, Newcastle, Australia. Recent reports suggest that psychological stress structurally and functionally alters microglia, cells that are pivotal to the production and maintenance of a neuroinflammatory state in the brain. This is of interest for two reasons: (a) stress is a major risk factor for the emergence of clinical depression, and (b) clinical depression appears to be characterised by enhanced levels of neuroinflammation. These two facts have led to the hypothesis that psychological stress may elicit changes in mood state and cognitive function by driving microglia-mediated neuroinflammatory events. In investigating this hypothesis our research group, using a variety of behavioural approaches, has previously found that chronic stress - sufficient to induce an increase in anhedonia and a decline in cognitive performance - also elicits microglial activation in a select subset of mood regulatory forebrain nuclei (notably the medial prefrontal cortex and the amygdala). We can now further report that pharmacologically preventing microglial activation during chronic stress exposure reduces stress-induced cognitive decline, and stress-induced changes in neuronal activation. Our group is now functionally characterizing, using a variety of ex-vivo techniques, the inflammatory status of microglia within the mood regulatory nuclei where we have observed differences after exposure chronic stress. Collectively, these findings advance our understanding of the cellular mechanisms at play in the brain during the development of stress-induced depression. These studies were supported by funding from the National Health & Medical Research Council of Australia.

ALARM PHEROMONE SUPPRESSES SEXUAL BEHAVIOR IN MALE RATS. Kobayashi,T.; Kiyokawa,Y.; Takeuchi,Y.; Mori,Y. Laboratory of Veterinary Ethology, The University of Tokyo, 1-1-1 Yayoi, Bunkyo-ku, Tokyo. We have reported that a male rat receiving foot shocks releases galarm pheromoneh that aggravates the rise in body temperature in response to novel environment, increases defensive and risk assessment behavior in the modified open-field test and acoustic startle reflex in recipient male rat. In contrast to their effects on individual animals, the effects of this pheromone on social behavior had not been analyzed. Among a wide variety of social behaviors, sexual interaction is one of the most important activities. Therefore, in the present study, we examined the effects of alarm pheromone on sexual behavior in rats. When a pair of male and female rats were exposed to the alarm pheromone during the copulation, the number of mounts preceding ejaculation increased and the hit rate (number of intromissions / number of mounts and intromissions) decreased in the male rat. In contrast, female sexual behavior was not affected by the alarm pheromone. These phenomena were ascribed to the pheromone effects in male rat because we obtained the similar results when we presented the pheromone only to male, but not female rat. In addition, pretreatment of corticotropin releasing factor (CRF) receptor antagonist (CP-154,526) or opioid receptor antagonist (naloxone) blocked the pheromone effects dose dependently, suggesting that CRF and opioid are involved in the alarm pheromone effects. Based on these results, we hypothesize that alarm pheromone suppresses male sexual behavior by inducing CRF secretion which, in turn, evokes opioid secretion in the brain.

6:00-7:00 Keynote Speaker: **Stephen Suomi**, NIH/NICHD
Risk, resilience, and gene-environment interplay in primates

RISK, RESILIENCE, AND GENE- ENVIRONMENT INTERPLAY IN PRIMATES. Stephen J. Suomi, Ph.D., Laboratory of Comparative Ethology, NICHD, NIH, Bethesda, MD, 20892-7971, USA. Recent research with both humans and rhesus monkeys has provided compelling evidence of gene-environment (G x E) interactions throughout development. For example, a specific polymorphism of the serotonin transporter (5-HTT) gene is associated with deficits in infant neurobehavioral functioning, extreme responsiveness to social stressors, poor control of aggression, and low serotonin metabolism during juvenile and adolescent development, and excessive alcohol consumption in early adulthood in monkeys reared with peers but not in monkeys reared by their mother. One interpretation of these findings is that secure attachment relationships somehow confer resiliency to individuals who carry alleles that may otherwise increase their risk for adverse developmental outcomes ("maternal buffering"). Similar patterns of apparent "buffering" have been demonstrated for G x E interactions involving several other genes with functionally equivalent polymorphisms in both humans and rhesus monkeys. Recent research has suggested that much of this "buffering" may be taking place in the context of early face-to-face interactions between rhesus monkey infants and their mothers. Moreover, the allelic variation seen in these genes in rhesus monkeys and humans but apparently not in other primate species may actually contribute to their remarkable adaptability and resilience at the species level.

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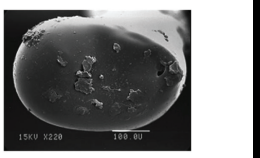

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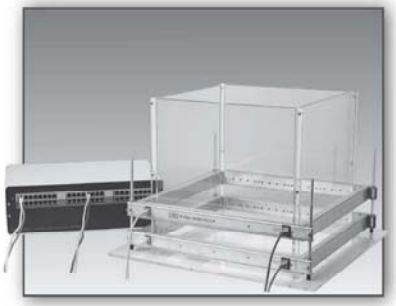

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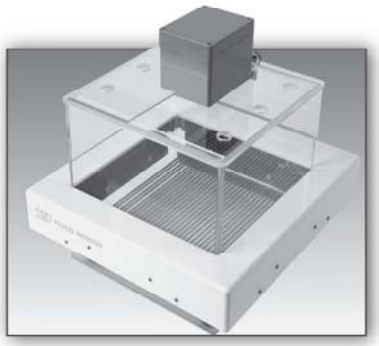

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IBNS Program (short version)

All events will be held in the Storm Peak/Mt. Werner Ballroom unless otherwise noted.

Tuesday, May 24, 2011

9:00-12:00	**Council Meeting** *(Aspen Boardroom)*
4:00-6:30	**Registration** *(Registration Booth, next to Rainbow Room)*
7:00-8:30	**Cocktail Reception** *(Pool Tent)*

Wednesday, May 25, 2011

7:30-8:30	**Continental Breakfast** *(Twilight Room)*
8:15-8:30	**Welcome**: IBNS President, Kelly Lambert. *(Storm Peak/Mt. Werner)*
8:30-10:45	**Symposium 1:** Sex, fear and pheromones…*(Storm Peak/Mt. Werner)*
10:45-11:15	**Break & Exhibit Viewing** *(Meeting Foyer)*
11:15-12:15	**Presidential Lecture:** Kelly Lambert *(Storm Peak/Mt. Werner)*
12:15-2:00	**Break**
2:00-3:00	**Workshop:** *Grants (Rainbow Room)*
3:00-5:15	**Symposium 2:** Orexin/hypocretin's role…*(Storm Peak/Mt. Werner)*
5:30-8:00	**Past Presidents' Symposium** *(Storm Peak/Mt. Werner)*
8:00-9:30	**Student/Postdoc Social** *(Villas Gallery)*

Thursday, May 26, 2011

7:30-8:30	**Continental Breakfast** *(Twilight Room)*
8:30-10:45	**Symposium 3**: Examining the genetic and neural …*(Storm Peak/Mt. Werner)*
10:45-11:15	**Break & Exhibit Viewing** *(Meeting Foyer)*
11:15-12:15	**Keynote Speaker:** Janice Kiecolt-Glaser *(Storm Peak/Mt. Werner)*
12:15-2:00	**Break**
2:00-4:00	**Media and Science Session** *(Storm Peak/Mt. Werner)*
4:00-5:30	**Oral Session 1** *(Storm Peak/Mt. Werner)*
6:30-8:30	**Poster Session 1** *(Sunshine Peak)*

Friday, May 27, 2011

7:30-8:30	**Continental Breakfast** *(Twilight Room)*
8:30-10:45	**Symposium 4:** Autism-relevant behaviors …*(Storm Peak/Mt. Werner)*
10:45-11:15	**Break & Exhibit Viewing** *(Meeting Foyer)*
11:15-12:15	**Keynote Speaker:** Kerry Ressler *(Storm Peak/Mt. Werner)*
12:15-2:00	**Break**
2:00-3:00	**Workshop:** *Science Jobs (Rainbow Room)*
3:00-5:30	**Travel Award Slide Blitz** *(Storm Peak/Mt. Werner)*
6:30-8:30	**Poster Session 2** *(Sunshine Peak)*

Saturday, May 28, 2011

7:30-8:30	**Continental Breakfast** *(Twilight Room)*
8:30-10:45	**Symposium 5:** Animal models to understand...*(Storm Peak/Mt. Werner)*
10:45-11:15	**Break & Exhibit Viewing** *(Meeting Foyer)*
11:15-12:15	**Professional Journeys Series:** Donald G. Stein *(Storm Peak/Mt. Werner)*
12:15-1:15	**IBNS Business Meeting** *(Storm Peak/Mt. Werner)*
1:15-3:15	**Meet the Professionals:** Student/Postdoc event *(Skyline/Sunset Room)*
3:30-5:00	**Oral Session 2** *(Twilight Room)*
6:00-7:00	**Keynote Speaker:** Stephen Suomi *(Storm Peak/Mt. Werner)*
7:00-7:30	**Cash Bar** *(Storm Peak/Mt. Werner)*
7:30-11:00	**Banquet:** Awards, buffet, dancing *(Storm Peak/Mt. Werner)*

Next IBNS Meeting:

June 5-10, 2012

Sheraton Keauhou Bay
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