



2024 Program and Abstract Book

Table of Contents

Schedule 3

Gather.town information 7

Committee members 10

Abstract book 13

 Day 1 Oral Presentation Session abstracts 13

 Day 2 Oral Presentation Session abstracts 16

 Day 1 Poster Session abstracts 19

 Day 2 Poster Session abstracts 43

Future meetings 67

Schedule

Day 1: Thursday September 12th, 2 - 6 pm EST

****1:00 - 2:00 pm: Early access for presenters****

2:00 - 2:15 pm: Opening Statements and Announcements – talk room

- SBN Online Research Symposium Committee Chairs
 - **Tori Riesgo**, graduate student, Willing Lab, Bowling Green State University, USA
 - **Laila Ouldibbat**, graduate student, Kundakovic Lab, Fordham University, USA
- SBN President Opening Remarks
 - **Dr. Margaret McCarthy, PhD**, Professor, University of Maryland, President, Society for Behavioral Neuroendocrinology

2:15 - 3:15 pm: Opening Keynote Lecture – talk room

- **Moderator: Laila Ouldibbat**, graduate student, Kundakovic Lab, Fordham University, USA
- **Speaker: Dr. Troy Roepke, PhD**, Professor, Rutgers University, USA
 - “Supporting My Community with My Science: Preclinical Models of Gender-Affirming Hormone Therapy”

3:15 - 3:30 pm: Announcements and break – talk room, lobby

- SBN Online Research Symposium Committee Co-Chair
 - **Tori Riesgo**, graduate student, Willing Lab, Bowling Green State University, USA

3:30 - 4:50 pm: Oral Presentation Session 1 – talk room

- **Moderator: Sophia Rogers**, graduate student, Bales Lab, UC Davis, USA
- **Speaker 1: Celine M. Camon**, graduate student, Centre for Neuroendocrinology, University of Otago, Dunedin, NZ
 - “DHED, The Brain Specific 17BE2 Prodrug, Affects Gonadal Steroid Receptor Expression but not Metabolic Function in Female Mice”
- **Speaker 2: Dante Cantini**, MS graduate, Choleris Lab, University of Guelph, CAN
 - “Interplay Between Estrogen Receptors and the Oxytocin Receptor in Female Mouse Social Recognition”
 - Poster A-12
- **Speaker 3: Dr. Han Tan, PhD**, postdoctoral fellow, Friedman Lab, Rockefeller University, USA
 - “Leptin Activated Hypothalamic BNC2 Neurons Acutely Suppress Food Intake in Mice”
 - Poster D-3
- **Speaker 4: Dr. Edenia da Cunha Menezes, PhD**, postdoctoral fellow, Nathan Kline Institute for Psychiatry Research, New York University, USA

- o “Effects of gestational hypothyroidism on mouse brain development: Gabaergic systems and oxidative stress”

4:50 - 5:50 pm: Break + Poster Session 1 – lobby, poster rooms A & B

5:50 - 6:00 pm: Closing announcements – lobby

- SBN Online Research Symposium Committee Secretary
 - o **Dr. Alyse Maksimoski, PhD**, staff scientist, de la Cruz Lab, Idaho State University, USA

Day 2: Friday September 13th, 10 am - 2 pm EST

****9:00 - 10:00 pm: Early access for presenters****

10:00 - 10:10 am: Opening Statement and Announcements – lobby

- SBN Online Research Symposium Committee Chairs
 - **Tori Riesgo**, graduate student, Willing Lab, Bowling Green State University, USA
 - **Laila Ouldibbat**, graduate student, Kundakovic Lab, Fordham University, USA

10:10 - 11:30 am: Oral Presentation Session 2 – talk room

- **Moderators: Dr. Brigitta Bonaldo, PhD**, postdoctoral fellow, Espinoza Lab, University of Piemonte Orientale, ITL
- **Speaker 1: Sarah Muh**, graduate student, Hoffman Lab, The University of Texas at Austin, USA
 - “Does Neuropeptide Y Modulate Responses to Social Stimuli in a State-Dependent Manner in a Monogamous Cichlid Fish, *Amatitlania nigrofasciata*?”
 - Poster C-3
- **Speaker 2: Dr. Samantha Lauby, PhD**, postdoctoral fellow, Champagne Lab, University of Texas at Austin, USA
 - “Postnatal Maternal Care Interacts with Prenatal Bisphenol Exposure on Esrrg Gene Expression, Co-Expressed Gene Profiles, and DNA Methylation in the Developing Hypothalamus of Female Rat Offspring”
 - Poster B-15
- **Speaker 3: Bradley Pedro**, graduate student, Romero Lab, Tufts University, USA
 - “Social disruption accelerates perineuronal net formation and impairs vocal learning in songbirds”
- **Speaker 4: Andi Liss**, graduate student, Varodayan Lab, Binghamton University, USA
 - “Cortical astrocyte regulation of glutamate synapses decreases in a sex-dependent manner following ethanol drinking in adolescence and adulthood in male and female mice.”
 - Poster D-10

11:30 am - 12:50 pm: Break and Poster Session 2 – lobby, poster rooms C & D

12:50 - 12:55 pm Announcements – lobby, talk room

- SBN Online Research Symposium Committee Co-Chair
 - **Tori Riesgo**, graduate student, Willing Lab, Bowling Green State University, USA

12:55 - 1:55 pm: Closing Keynote Lecture – talk room

- **Moderator: Dr. Alyse Maksimoski, PhD**, staff scientist, de la Cruz Lab, Idaho State University, USA
- **Speaker: Dr. Farrah Madison, PhD**, Assistant Professor, University of Wisconsin-Madison, USA
 - “Insights from the Songbird Hippocampus into neural mechanisms of Social Navigation”

1:55 - 2:00 pm Closing Statement and Announcements – talk room

- SBN Online Research Symposium Committee Co-Chair
 - **Laila Ouldibbat**, graduate student, Kundakovic Lab, Fordham University, USA

Gather.town information

We will be using the virtual platform [Gather.town](#) for the symposium. Here you can find some useful tips to help you enjoy this platform!

1. Launching Gather.town

The platform can be accessed in a web browser from a computer (**not a tablet/smartphone**) to run properly. Gather.town recommends using **Chrome** as the web browser. To access, you will need to click the link that was sent to you in the meeting invitation email. After clicking the link, you will be directed to a webpage where you can sign in *with the email you registered for the event* once the event has started. You can download the desktop application if you wish, but it is not necessary. After a few moments, the platform will launch in your web browser, or you can click “Or, continue in your browser”.

2. Getting started and customizing your character

In Gather.town, you will use your keyboard to control a simple, customizable avatar in the virtual meeting space. Please allow a few minutes before the start of the meeting to set up your personalized avatar. You will be directed to a webpage where you can enter your name and quickly design the appearance of your character. You will be prompted to allow the platform to use your camera and microphone – select “**Allow**” for these prompts. If you are not used to using Chrome, this may require restarting the browser (this should relaunch all of your open tabs)..

3. Initial user tutorial after entering Gather.town (optional)

After you are finished customizing your character, you will be brought to a short tutorial, which takes approximately 1 minute to complete. This tutorial provides you with basic information on using the Gather.town platform. You can choose to skip this tutorial if you wish. This tutorial will only appear the first time you use Gather.town. If you leave the meeting and come back later, the tutorial will not reappear unless you choose to complete it again.

4. Navigating the meeting space



A: Poster rooms are on the left and right of the lobby. Posters will be set up here during both days of the symposium, although presenters will only be present during their scheduled times. See “6. Interacting with posters and poster presenters” below for more information. Note: once inside the room, the exit is located at the bottom of your screen.

B: The talk room is at the top of the lobby. This is where the oral presentations, keynotes, and most announcements will take place.

C: There are several “private areas” around the Space. This table is one example and there are several others, including benches.

D: Throughout Gather.town, there are sets of kiosks with general information and helpful tips. At any time, you can also access

information by selecting the Main Menu  and then the Help Center .

5. Navigation and interaction with attendees

As you walk around in the lobby and in the poster area, you will be able to interact with other meeting attendees as you pass by. If you are close enough to them, you will be able to speak to them and see their video feed. If your microphone is turned on, you can converse with others if you are close enough. As you increase your distance from other attendees, you will no longer be able to hear or see them.

You will need to provide Gather.town access to your microphone and camera if you use a microphone for communication and/or would like others to see you. A pop-up will appear on your screen prompting you to permit access.

6. Interacting with posters and poster presenters

In the poster area, you will see a poster board set up with filled-in colored ovals that designate “discussion spaces” for viewing the poster and interacting with the poster presenter. You can only interact with the poster presenter, and others in the space, if you are standing on one of the filled-in ovals.

As you get closer to the poster, a pop-up window will appear at the bottom of the page. This pop-up window provides a preview of the poster you are near, including the title of the poster. The video and audio feed of anyone in that “discussion space” will also appear. When you are in this space, if you press the “x” button on your keyboard, you will then enter the full-screen poster mode. You can use the hand icon  to move the poster around and view different parts of the poster. On the right-hand side of the poster, there is a zoom button  that will allow you to zoom in on specific parts of the poster. The audio and video feeds of other people in the discussion space will appear at the top of the screen. When listening to the poster presenter, please turn your microphone off, and then turn it on again when you have questions or if there is a point of discussion.

7. Interacting with talks and speaker presenters

We will have many interesting talks during the meeting! While someone is presenting, please remember to keep your microphone turned off. Do not interrupt the talk. At the end of every talk, the speakers will be glad to answer your questions. If you have a question, you can type your question in the chat, and the moderator will ask it in your place.

8. Accessibility/inclusivity features

Gather.town has several features to make Gather.town a better experience with assistive technology including zoom adjustment, live captioning and participant information outlined at this [link](#).

Smart zoom: Under Accessibility in User Settings, you can reduce motion on the screen and change the label and tooltip font size. Keep an eye on User Settings for other tools (like disabling Smart Zoom) that will allow you to more finely tune your Gather.town experience.

Live captioning: Chrome natively supports live captioning of audio streams, which WebRTC connects to automatically, so you can have live captioning for audio in Gather.town. To enable live captions, open Chrome's Settings. In the Accessibility section under Advanced, toggle on Live Caption.

Participant symbols: Participants in the space can add emoji symbols in their name or in their status to provide more info about themselves. We encourage speakers, attendees etc. to use a chosen symbol in their name or status for quick visual cues of their role in the Space (e.g., ★ SBN ORS Committee; 🎤 = speaker; 🗣️ = hearing impaired). You can add an emoji directly to your name using the emoji picker native to your operating system. Use “Ctrl/⌘ + space” for Mac, or use “Windows key + period (.)” for Windows. Then search for the emoji you wish to add to your name!

9. Navigating technical difficulties

You can contact Gather.town support for individual troubleshooting help or, by selecting Report issues from the main Gather.town menu. You can find some useful tips [here](#).

SBN ORS Committee

The SBN ORS committee is composed of trainee researchers (graduate students, post-docs) across diverse behavioral neuroendocrinology labs. The ORS committee is overseen by the SBN membership committee, including **Dr. Matthew Paul** (University at Buffalo), **Dr. Wendy Saltzman** (University of California, Riverside), **Dr. Stephanie Correa** (UCLA) and **Dr. Damian Zuloaga** (University at Albany).

Victoria Riesgo (co-chair) is a sixth year PhD candidate in the Willing lab at Bowling Green State University. She graduated from California State University, San Marcos with a B.A. in Psychology in 2019, where she studied the influence of neuropeptides on rodent maternal behavior in the D'Anna-Hernandez lab. Upon joining the Willing lab in the fall of 2019, Victoria began work investigating the effects of two common environmental factors, phthalates and maternal infection, on neurodevelopment. Her master's thesis identified that each of these factors introduced perinatally can produce sex-specific reductions in neuron number within the adult prefrontal cortex. Victoria's current research focuses on pubertal hormones and their role in shaping the development of the dopaminergic neurons in the rat midbrain. She is passionate about student mentorship and currently manages a team of six undergraduate research assistants, as well as an additional four students in collaborative projects with other labs. Many of these research assistants have been awarded internal funding for their research, presented at national conferences, and gained authorship on publications under her guidance. As a Latina first-generation academic, she is dedicated to increasing accessibility and inclusivity within neuroscience, guiding similarly under-represented learners through the landscape of higher education with the goal of increasing retention and academic success.

Laila Ouldibbat (co-chair) is a 5th year PhD student in Dr. Marija Kundakovic's lab at Fordham University in New York. Laila graduated from Drexel University in 2016 with a B.S in Nutrition Sciences. Prior to graduate school Laila worked in clinical dietetics and studied preterm neurodevelopment which began to shape her love and interest for neuroendocrinology. The Kundakovic lab is broadly interested in the epigenetic basis of behavior and psychiatric disorders with a focus on hormonal and environmental factors driving sex differences. Upon joining, Laila began a new metabolic research project in the lab. Her research is focused on studying metabolic phenotypes and the molecular mechanisms of ovarian hormone mediated regulation of metabolism in the adult female hypothalamus. Laila is a recent recipient of the Clare Boothe Luce Fellowship for Women in STEM and is passionate about the implications her research has for women's health, as well as advocating for diverse voices and inclusion in science.

Dr. Alyse Maksimoski (secretary) (she/they) is a staff scientist in the de la Cruz lab at Idaho State University. She graduated from Michigan State University in 2017 with a Bachelors in Zoology. During her undergraduate experience, she studied the social behaviors of honeybees, spotted hyenas, and North American rodents. After graduating, they worked in a primatology lab at the University of Massachusetts-Amherst before heading to Zambia to study aggression in male kinda baboons. In the fall of 2019, Dr. Maksimoski started their doctoral studies at the University of Wisconsin-Madison in the Integrative Biology Department in Dr. Lauren Ritters' lab. Dr. Maksimoski's dissertation identified mechanisms that facilitate and reward vocal-social behaviors in European starling flocks, with particular focus on individual variation in social experience. This work provided novel support for the idea that opioids released during social experience may function as a

proximate reinforcer of flocking via coupling the associated positive affective state to the social environment. During her PhD program, Dr. Maksimoski consistently made representation and retention of women and underrepresented students a priority, including mentoring nine such undergraduates, many of whom garnered authorship on posters and publications. Dr. Maksimoski is a recent recipient of the inaugural SBN Elsevier Award and is currently studying how senescence alters the endocrine modulation of pancreatic islets under Dr. Lizbeth de la Cruz at ISU. She will transition this winter into a postdoctoral fellowship in Dr. Devaleena Pradhan's lab, also at ISU, studying neuroendocrine mechanisms behind adult feminization in the sex changing blue banded goby.

Dr. Emily Wright completed her undergraduate studies at UC Davis, where she fell in love with neuroendocrinology as a research assistant in the lab of Dr. Karen Bales. She continued her academic journey at UC Davis, where she pursued her doctoral studies under the mentorship of Dr. Brian Trainor. Dr. Wright's dissertation work identified puberty as an important organizational period for testosterone to reduce both behavioral and neuronal responses to social stress in adult male California mice. Dr. Wright is now a postdoctoral fellow in the lab of Dr. Lin Tian at the Max Planck Florida Institute for Neuroscience. Her current research employs the genetically-encoded serotonin indicator, iSeroSnFR2.0, to monitor changes in serotonin release during fear learning and pharmaceutical manipulation. Her work is supported by a BRAIN Initiative F32 Individual Postdoctoral Fellowship. Dr. Wright is deeply committed to fostering a supportive and inclusive environment in the scientific community, especially for trainees. Inspired by the exceptional mentorship she received early in her career, she prioritizes mentorship both within and outside of the lab.

Aaron Fleischer is a sixth-year graduate student in the Frick lab at the University of Wisconsin-Milwaukee. Aaron graduated with degrees in biology and chemistry from the University of South Dakota in the spring of 2018 and joined Karyn Frick's lab in the summer of the same year. Broadly, the Frick lab studies how estrogens drive memory processes in mice of both sexes, with special, recent emphasis being given to Alzheimer's disease models and developing novel treatments for the menopausal loss of circulating estrogens. Aaron completed his master's thesis in the spring of 2021, where he developed a long-term treatment regimen and behavioral battery to test the efficacy of a highly selective estrogen receptor beta agonist in reducing preclinical indices of menopause-related symptoms in ovariectomized mice. Currently, Aaron is pursuing his dissertation, examining how estrogenic signaling builds upon learning-induced cellular activity to stabilize learned experiences into long-term memory within different hippocampal cell types. Aaron is particularly interested in sex comparisons regarding the roles estrogens play in facilitating memory processes. Importantly, this work has involved many undergraduate research assistants and postbaccalaureate researchers, resulting in many locally and nationally presented posters and talks given by rising scientists from the UWM community. Thus, Aaron is impassioned by mentoring students to find their scientific passions and working with others to facilitate the field's neurobiological understanding of sex comparisons in learning and memory.

Sophia Rogers is a third-year PhD student in the Bales Lab at the University of California, Davis (UCD). The Bales Lab focuses on the comparative neurobiology of monogamy. Sophia's research interests include pair-bonding, adult social relationships, and the effects of prenatal conditions. Her current project investigates

the behavioral consequences of prenatal THC exposure in prairie voles. Additionally, Sophia is dedicated to closing the gender gap in academia, actively promoting diversity and inclusion in scientific research.

Krystyna Rybka is a sixth-year PhD candidate in the Zuloaga lab at the University at Albany in New York. She received her Bachelor of Science degree in Zoology with minors in both Neuroscience and Psychology from the University of Maine in 2018. Her research interests include the effects of stress on HPA-axis regulation and its contribution to stress-related mood disorders, focusing on how sex differences alter their susceptibility. She's particularly interested in the neuroendocrinology aspect of behavioral neuroscience to better understand the hormonal mechanisms that result in sex differences in the prevalence rates of autoimmune diseases that disproportionately affect women.

Dr. Brigitta Bonaldo is a junior postdoc. She completed her undergraduate studies in Neurobiology at the University of Turin. During her undergraduate experience, she studied the role of the anti-inflammatory enzyme A20 in the neuropathology of Multiple Sclerosis. In 2017, she started her doctoral studies in the Neuroendocrinology lab under the mentorship of Professor Giancarlo Panzica at the University of Turin. Her PhD work evaluated the effects on the brain and behavior of oral exposure to bisphenols A or S during critical periods of development. She was particularly interested in studying the possible impairments in maternal, sexual, and anxiety-related behaviors. In October 2023, she started a new postdoc fellowship in the lab of Dr. Stefano Espinoza at the University of Piemonte Orientale in collaboration with the University of Trento and the Istituto Italiano di Tecnologia (IIT) of Genoa, supported by the Simons Foundation Autism Research Initiative (SFARI). The main aim of her current project is to provide Proof-of-Concept for developing a novel RNA-based therapy for autism spectrum disorders (ASD). Even though ASD is a behaviorally defined disorder with a strong sexually dimorphic component, the behavioral phenotype and the sexual dimorphism in the transgenic models of ASD often represent critical points, as they could be mild, poorly described, or not considered; thus, she is now particularly interested in implementing the phenotyping of those aspects as key features to be considered when selecting an experimental model or testing new therapeutic approaches. She fully supports the idea that science is sharing, which goes hand in hand with collaboration, accessibility, and inclusiveness.

Abstracts

Day 1: Oral Presentation Session

1. “DHED, The Brain Specific 17BE2 Prodrug, Affects Gonadal Steroid Receptor Expression but not Metabolic Function in Female Mice”

Celine M. Camon¹, Jenny Clarkson¹, Caroline Decourt¹, Katalin Prokai-Tatrai², Rebecca E. Campbell¹, Mike Garratt¹

¹ Centre for Neuroendocrinology, University of Otago, Dunedin, NZ. ² Department of Pharmacology and Neuroscience, University of North Texas Health Science Center Fort Worth, Texas, USA.

Menopausal hormone therapy (MHT) alleviates symptoms including hot flushes, mood disturbances and weight gain and contains estrogens such as 17-Beta estradiol (17BE2). However, peripheral estrogen receptor activation by MHT can increase reproductive cancers and cardiovascular event risk in some patients. As protective metabolic effects of 17BE2 are mediated through the arcuate nucleus of the hypothalamus, restricting 17BE2 actions to the brain could serve as a safer mechanism of MHT in the treatment of weight gain and other metabolic disturbances associated with menopause.

10b,17B-dihydroxyestra-1,4-dien-3-one (DHED), is a prodrug of 17BE2 which is enzymatically converted to estradiol exclusively within the brain. DHED has demonstrated positive benefit in rodent models of hot flushes, cognitive decline and stroke (1). We hypothesise that DHED treatment in female mice will act within the hypothalamus to provide the same beneficial metabolic effects as 17BE2, while avoiding peripheral actions. Female mice placed on a high fat diet to induce metabolic dysfunction were split into either control, DHED, or 17BE2 treatment groups. Body weight, food intake, uterus weight and glucose tolerance was recorded along with estrogen and progesterone receptor expression in the brain. Findings to date indicate that DHED influences the expression of steroid receptors in the hypothalamus, but does not elicit the same protective metabolic effects as 17BE2. Further optimisation may be required to fully establish whether DHED can provide protection against metabolic dysfunction.

2. “Interplay Between Estrogen Receptors and the Oxytocin Receptor in Female Mouse Social Recognition”

Dante Cantini¹, Miji Cha¹, Catherine Schmidt¹, Elena Choleris¹

¹ University of Guelph Department of Psychology, Neuroscience and Applied Cognitive Science Program

In the social-brain-network the estrogen, 17 β -estradiol, has been shown to rapidly facilitate social recognition in mice. The medial amygdala of mice expresses the three known estrogen receptors: ER α , ER β , and G Protein-Coupled ER (GPER). Selective agonists for each of the three estrogen receptors rapidly facilitate social recognition in the medial amygdala of female CD1 mice. Oxytocin receptors within the medial amygdala are necessary for the rapid effects of 17 β -estradiol on social recognition to occur, suggesting a 17 β -estradiol/oxytocin receptor interplay. Our objective was to elucidate which of the estrogen receptors interplay with oxytocin receptors in the medial amygdala of female mice to rapidly facilitate social recognition. Female mice were ovariectomized and had bilateral cannulae implanted into the medial amygdala. A sub-effective dose of oxytocin receptor antagonist (the highest dose of the antagonist that per se does not inhibit social recognition) was infused into the medial

amygdala before infusion of an estrogen receptor agonist, either ER α agonist PPT, or ER β agonist DPN, or GPER agonist G1). A social recognition paradigm designed to measure the rapid facilitating effects of treatment was administered. If the facilitating effect of the estrogen receptor agonist is impaired by the administration of the oxytocin receptor antagonist, we can infer that the specific estrogen receptor is implicated in the interplay with oxytocin receptors. A sub-effective dose of oxytocin receptor antagonist administered before each estrogen receptor agonist prevented the facilitation of social recognition by the agonist, demonstrating that each estrogen receptor interplays with oxytocin receptors in this process.

o Poster A-12

3. “Leptin Activated Hypothalamic BNC2 Neurons Acutely Suppress Food Intake in Mice”

Han L. Tan¹, Luping Yin², Yuqi Tan³, Jessica Ivanov¹, Kaja Plucinska⁴, Anoj Ilanges¹, Brian R. Herb⁵, Putianqi Wang¹, Christin Kosse¹, Paul Cohen⁴, Dayu Lin², Jeffrey M. Friedman¹

¹ Laboratory of Molecular Genetics, Howard Hughes Medical Institute, The Rockefeller University, New York, NY, USA. ²

Department of Psychiatry, Neuroscience Institute, New York University Langone Medical Center, New York, NY, USA. ³

Department of Microbiology and Immunology, Stanford University School of Medicine, Stanford, CA, USA. ⁴ Laboratory of

Molecular Metabolism, The Rockefeller University, New York, NY, USA. ⁵ Institute for Genome Sciences, Department of

Pharmacology, University of Maryland School of Medicine, Baltimore, MD, USA

The balance between hunger and satiety is crucial for maintaining energy balance, largely regulated by the hormone leptin. Leptin, produced by adipose, targets the hypothalamus's arcuate nucleus (ARC) to control food intake. Two neuron types in this system, orexigenic AGRP and anorexigenic POMC, express leptin receptors (LepR), and are central to hunger regulation, representing a ‘yin-yang’ model. Leptin promotes satiety by inhibiting AGRP and activating POMC neurons. While AGRP neurons regulate feeding on a rapid time scale, acute activation of POMC neurons has a minimal effect, suggesting that there is a heretofore unidentified leptin-regulated population that acutely suppresses appetite. Through single nuclei RNA-seq, we identified a novel population of leptin-target neurons expressing basonuclin 2 (BNC2). Functional studies revealed BNC2 neurons suppress food intake with kinetics similar to the swift induction of feeding after AGRP neuron activation by monosynaptically inhibiting AGRP neurons. Furthermore, activating BNC2 neurons induces positive valence, suggesting that it suppresses the negative emotional effects of AGRP activation. Acute activation of BNC2 neurons also regulated peripheral glucose metabolism by decreasing glucose levels and increasing insulin sensitivity, an effect opposite to that of AGRP neuron activation. Finally, deleting LepR in BNC2 neurons of adult mice leads to marked hyperphagia and obesity, similar to the effects observed in LepR knockouts in AGRP neurons. These findings reveal that BNC2-expressing neurons are a crucial missing component in the neural circuit that maintains the balance between hunger and satiety, providing the ‘yang’ to the AGRP neurons’ ‘yin’ in the regulation of feeding.

o Poster D-3

4. “Effects of gestational hypothyroidism on mouse brain development: Gabaergic systems and oxidative stress”

Edênia da Cunha Menezes¹, Fabiula Francisca de Abreu², Jada B Davis³, Sara V Maurer³, Venezia C Roshko³, Angela Richardson⁴, Jonathan Dowell³, Sarah N Cassella⁴, Hanna E Stevens⁵

¹ Psychiatry Department, Emotional Brain Institute, Nathan Kline Institute for Psychiatric Research, Orangeburg, NY, United States; Psychiatry Department, Iowa Neuroscience Institute, University of Iowa Carver College of Medicine, Iowa City, IA, United States. ² Departamento de Fisiologia, Programa de Pós-Graduação em Ciências Fisiológicas, Universidade Federal de Sergipe, São Cristóvão, Brazil. ³ Psychiatry Department, Iowa Neuroscience Institute, University of Iowa Carver College of Medicine, Iowa City, IA, United States. ⁴ Neuroscience Department, Loras College, Dubuque, IA, United States. ⁵ Psychiatry Department, Iowa Neuroscience Institute, University of Iowa Carver College of Medicine, Iowa City, IA, United States.

Hormonal imbalance during pregnancy is a risk factor for neuropsychiatric impairment in the offspring. It has been suggested that hypothyroidism leads to dysfunction of cortical GABAergic interneurons and inhibitory system development that in turn underlies impairment of the central nervous system. Here we investigated how gestational hypothyroidism affected offspring GABAergic system development as well as redox regulation parameters, because of previous links identified between the two. Experimental Gestational Hypothyroidism (EGH) was induced in CD-1 mice with 0.02% methimazole (MMI) in drinking water from embryonic day 9 (E9) until tissue collection at embryonic day 14 (E14) or E18. We examined GABAergic cell distribution and inhibitory system development gene expression as well as redox relevant gene expression and direct measures across all embryos regardless of sex. Intrauterine restriction of maternal thyroid hormones significantly impacted both of these outcomes in brain, as well as altering redox regulation in the placenta. GAD67+ neuronal migration was reduced, accompanied by a disruption in gene expression influencing GABAergic cell migration and cortical inhibitory neural system development. EGH also altered embryonic brain gene expression of Gpx1, Nfe2l2, Cat levels in the dorsal E14 brains. Additionally, EGH resulted in elevated TBARS, Gpx1 and Nfe2l2 in the ventral E18 brains. Furthermore, EGH downregulated placental Gpx1 gene expression at E14 and increased protein oxidation at E18. These findings support the hypothesis that sufficient maternal thyroid hormone supply to the fetus influences central nervous system development, including processes of GABAergic system development and redox equilibrium.

Day 2: Oral Presentation Session

1. Does Neuropeptide Y Modulate Responses to Social Stimuli in a State-Dependent Manner in a Monogamous Cichlid Fish, *Amatitlania nigrofasciata*?”

Sarah A. Muh¹, Camille P. Brown¹, Ross S. DeAngelis^{1,2}, Hans A. Hofmann^{1,2}

¹ Department of Integrative Biology, University of Texas at Austin, ² Institute for Neuroscience, University of Texas at Austin

How the brain regulates behavioral responses to salient situations depends on an individual's behavioral state. For example, in pair-bonding species, unbonded individuals readily approach unfamiliar opposite sex individuals, yet once a pair bond is established, they may avoid or even attack them. Similarly, pair-bonded individuals may respond to offspring cues only after they have reproduced. While the role of both neuropeptides and dopamine in regulating the formation of these enduring bonds has been studied in detail, our understanding of how other peptide systems mediate the responses to aggressive or affiliative stimuli in a state-dependent manner is very limited. Here, we use the monogamous biparental Convict Cichlid, *Amatitlania nigrofasciata*, to investigate how Neuropeptide Y (NPY) may regulate responses to social stimuli in bonded or parental convict females. Our results show that pair-bonded males and females coordinate their responses to social challenges, with context-dependent sex differences. Through immunofluorescent labeling of the neural activity marker phospho-S6 ribosomal protein (pS6) and NPY, we demonstrate that differentially motivated aggressive and affiliative responses are mediated by brain region-specific activity patterns of this neurohormonal system. These results show how information about the social world is represented in the brain across hierarchically organized levels to generate behavioral state-dependent context-appropriate behavior.

o Poster C-3

2. “Postnatal Maternal Care Interacts with Prenatal Bisphenol Exposure on Esrrg Gene Expression, Co-Expressed Gene Profiles, and DNA Methylation in the Developing Hypothalamus of Female Rat Offspring”

Samantha Lauby^{1,2}, and Frances Champagne^{1,2}

¹ University of Texas at Austin Department of Psychology, ² University of Texas at Austin Center for Molecular Carcinogenesis and Toxicology

Environmental exposures co-occurring during early-life have a profound influence on neurodevelopment. Our previous work in rats suggests that postnatal maternal care modulates the effects of prenatal exposure to bisphenols, an estrogenic endocrine disrupting chemical, on offspring neurodevelopment. High postnatal maternal care and prenatal bisphenol exposure have known opposing effects on estrogen receptor alpha (Esr1) expression in the medial preoptic area (MPOA) of the hypothalamus. We hypothesized that postnatal maternal care would interact with prenatal bisphenol exposure on transcription profiles in the developing MPOA, including Esr1, and affect DNA methylation modifications downstream. We administered two doses (50 ug/kg/day and 150 ug/kg/day) of a bisphenol mixture (BPA, BPF, BPS) and a corn oil control to pregnant rat dams. Postnatal maternal care was measured using home-cage videos. We found a significant interaction between the 150 ug/kg bisphenol group and maternal care (compared to controls) on estrogen-related receptor gamma (Esrrg) expression but not Esr1 in females. We also found interactions between the 150 ug/kg

bisphenol group and maternal care on five co-expressed gene profiles in females. Four co-expressed gene profiles were enriched for estrogen-responsive genes. Finally, we found substantial influences of postnatal maternal care on differentially methylated regions in all prenatal bisphenol-treated groups in males and females. Overall, this work suggests that postnatal maternal care influences *Esrrg* expression, genome-wide transcription profiles, and DNA methylation modifications in the MPOA of bisphenol-exposed female offspring. Since *Esrrg* has a high affinity for BPA, it will be important to explore the downstream consequences of *Esrrg* activity.

- o Poster B-15

3. “Social disruption accelerates perineuronal net formation and impairs vocal learning in songbirds”

Bradley P. Pedro¹, Yuqi Guo¹, L. Michael Romero¹, Mimi H. Kao¹

¹ Department of Biology, Tufts University, Medford MA

Vocal learning occurs in a sensitive period early in development during which social interactions and sensory experience actively shape brain structure and function. In humans and songbirds, early life adversity, such as social isolation or sensory deprivation, profoundly impair development of auditory circuits. In addition, there is growing evidence that early life stress alters development of brain regions involved in memory, cognition and emotion, such as the hippocampus, prefrontal cortex, and amygdala. How early life stress shapes motor circuits, however, is not well understood. Using the vocal learning zebra finch, we test the hypothesis that social experience modulates learning by regulating expression of perineuronal nets (PNNs), extracellular matrix structures that restrict neural plasticity, in a motor circuit specialized for producing learned vocalizations. We show that repeated separation of juvenile males from their families for just a few hours each day over a two-week period accelerates the formation of PNNs in the song motor circuit. Moreover, we find that song learning is impaired in separated males compared to age-matched controls, consistent with premature closure of the sensitive period. In contrast, separation did not drive premature elevation of circulating testosterone nor chronic increases in corticosterone. Together, our results show that even short bouts of social separation can induce structural changes that restrict plasticity in motor circuits, with long-lasting effects on behavior. More generally, they highlight a role for extracellular matrix structures in modulating the brain's ability to respond dynamically to changing social experience.

4. “Cortical astrocyte regulation of glutamate synapses decreases in a sex-dependent manner following ethanol drinking in adolescence and adulthood in male and female mice.”

Andi Liss¹, Florence P. Varodayan¹

¹ Behavioral Neuroscience Program, Department of Psychology, Binghamton University-SUNY, Binghamton, NY, USA
Prefrontal cortex (PFC) pathology is associated with the cognitive symptoms of alcohol use disorder (AUD); however, the underlying mechanisms remain understudied. Preclinical studies in male rodents have shown that ethanol exposure in adolescence or adulthood alters glutamatergic transmission in the medial PFC (mPFC), and that astrocytes can also influence glutamatergic transmission by buffering calcium signaling and recycling glutamate. Following adolescent ethanol drinking, cortical astrocytes in adult male mice show reduced surface area to volume ratios and synaptic contacts, suggesting a decreased ability to regulate synapses. To date, no studies have examined the role of

astrocytes in ethanol's synaptic actions in the mPFC of either sex, or if this change in astrocyte morphology following adolescent drinking produces long-term functional changes at glutamate synapses. Here, we assessed the role of astrocytes in ethanol's regulation of glutamate transmission in prelimbic (PL) mPFC layers 2/3 and 5 pyramidal cells in adult male and female C57BL/6J mice using ex vivo electrophysiology following chronic ethanol drinking in adolescence or adulthood. We found that acute ethanol increased glutamate release in PL layer 2/3 in water-drinking male adults and layer 5 in water-drinking female adults. Pharmacological reduction of astrocyte activity blocked these sex- and layer-specific effects. After chronic ethanol drinking in adolescence or adulthood, acute ethanol synaptic regulation in adulthood was no longer observed. Collectively, our findings demonstrate that mPFC astrocytes play critical roles in the cellular and behavioral actions of ethanol in both male and female mice. Ongoing molecular studies are examining the specific astrocyte mechanisms involved.

- o Poster D-10

Day 1: Poster Session

A-1. “Vagal tone and neural systems underlying emotional reactivity in young children”

Nicolas Murgueitio¹, Kathryn Garrisi¹, Michelle Shipkova¹, Margaret Sheridan¹

¹ Department of Psychology and Neuroscience, University of North Carolina at Chapel Hill

The parasympathetic nervous system (PNS) plays a key role in emotional reactivity (ER). For example, increased respiratory sinus arrhythmia (RSA) at is linked with better ER and decreased psychopathology in children. However, little data exists linking PNS at rest and neural function supporting ER in young children. The present study aimed to explore associations between resting RSA and children’s neural processes during ER. Resting RSA was assessed using noninvasive ECG recordings. Participants (N = 50; Mage = 6.29 years, SD = 0.84; 27 female), were instructed to sit quietly without moving for 3 minutes. We used an fMRI paradigm to assess ER. During this task children passively view negative and neutral pictures. We examined associations between resting RSA and activation during the task using whole-brain analyses with cluster-level corrections (voxel level correction $z = 3.1$) to compare BOLD responses as children observed negative (vs. neutral) stimuli (i.e., ER). Children with higher resting RSA showed heightened recruitment of the temporal fusiform cortex and parahippocampal gyrus when viewing negative compared to neutral images. We observed that children with higher resting RSA, an index commonly associated with decreased ER, also showed increased activity in regions associated with visual processing when viewing negative vs. neutral images. It may be that the enhanced visual processing observed for these children reflects their ability to situate emotions, naturally controlling strong reactivity. Taken together, these findings suggest that as expected, resting PNS predicts neural processes associated with ER in young children.

A-2. “Relative reward processing of ethanol following sucrose exposure in non-food-restricted female rats”

Erika Shultz¹, Kristina Thompson¹, Howard Casey Cromwell¹

¹ Bowling Green State University

Alcohol Use Disorder (AUD) is a disease that has a devastating effect on millions of individuals around the world. While around 50% of the risk for developing AUD has been determined to be genetic, examining predictors of addictive-like behavior and individual variance in models without a family history of addiction is also important. Factors such as impaired reward sensitivity and response to natural reward may play a role in an individual’s propensity to develop alcohol addiction. Additionally, little research has been done on predictors of alcohol addiction without the use of food or water deprivation to coax animals into addiction. This study examined the appetitive and consummatory behavior of female Wistar rats in self-administration tasks of sucrose and ethanol solutions. The rats had ad-libitum access to food and water throughout the study. Time-based manipulation of reward value was utilized to evaluate relative reward effects and reward sensitivity was assessed on both an inter-session and intra-session basis. This allowed for evaluation of motivational changes across different time periods and how those changes contribute to addictive behavior. Sucrose consumption during the shortest time access predicted both 15% and 30% ethanol consumption in the home cage during 30-minute exposure. The current study allows a closer

examination of the motivational processes shared by alcohol and sugar that could result in addiction. Using natural reward sensitivity to predict future addiction could aid significantly in preventing and treating substance use disorders.

A-3. “Altered Social Communication in Adult Vasopressin-deficient Brattleboro Rats”

Chloe N Cordes¹, Cole P Fredericks¹, Lingling Liu¹, Destiny J Brakey¹, Derek Daniels², Matthew J Paul¹

¹ Department of Psychology, University at Buffalo SUNY, ² Department of Biological Sciences, University at Buffalo, SUNY
Vasopressin (AVP) has been implicated in social communication across a diverse array of species. Many rodents communicate basic behavioral states through high-pitched vocalizations above the human hearing range (ultrasonic vocalizations; USVs). Previous studies have found that infant, juvenile, and adolescent Brattleboro (Bratt) rats, which have a mutation in the *Avp* gene, exhibit deficits in their USVs. The present study tested whether Bratt rats continue to exhibit USV deficits in adulthood. USVs of adult male and female Bratt and wild type (WT) rats were recorded in two contexts: a novel environment (empty arena) and a social context (arena filled with bedding soiled by same-sex conspecifics). The number, frequency, and duration of 50 kHz USVs were quantified by DeepSqueak after validation with manual scoring. Twenty-two kHz measures were quantified by manual scoring because DeepSqueak failed to accurately detect USVs in this frequency range. Adult Bratt rats did not exhibit deficits in the number of 50kHz USVs: male Bratt rats emitted similar 50kHz USVs as male WT rats, whereas female Bratt rats emitted more USVs than female WT rats. USV frequency and duration were altered in adult Bratt rats, but in a context-dependent manner. Twenty-two kHz USVs were less affected by the Bratt mutation. The present study demonstrates how chronic AVP deficiency impacts social communication across the lifespan. The present findings reveal a complex role for AVP in vocal communication, whereby disruption to the *Avp* gene leads to sex-, context-, and developmental phase-specific effects on the quantity and spectrotemporal characteristics of rat USVs.

A-4. “Relationships between estrogen, menopause, and genetic risk for Alzheimer’s disease in the frame of neural network dynamics and emotional memory performance – A cross-sectional study in human females.”

Hannah Ballard¹, Madelyn Castro¹, Lorena Ferguson¹, Ashwathi Nair¹, Alexis Bailey¹, & Stephanie Leal^{1,2}

¹ Psychological Sciences Department, Rice University, ² Integrative Biology & Physiology, University of California, Los Angeles

Given substantial evidence of an interaction between menopause and Alzheimer’s disease (AD), interrogating the linkage between endocrine aging and risk for AD is critical for uncovering biological factors that may elicit distinct aging trajectories between sexes. However, work on brain-behavior responses during menopause is lacking, especially in the context of AD. To correct this imbalance, our project examined the effects of estrogen decline with menopause on emotional memory performance and neural network dynamics in cognitively normal females that carry genetic risk for AD. We used salivary assays to identify allele status for the apolipoprotein (ApoE) gene and measure 17 β -estradiol (17 β -E) levels in pre- and post-menopausal females ages 35+. Reproductive stage was classified

using the Stages of Reproductive Aging Workshop criteria. We administered a mnemonic discrimination paradigm during high-resolution functional magnetic resonance imaging (fMRI) to assess emotional memory and activity within the medial temporal lobe network, as both are sensitive to age-related decline. We observed a predictive effect of 17β -E on detailed memory performance, where higher 17β -E was associated with better memory. Further, a trending effect of ApoE4 status was present; the positive association between 17β -E and detailed memory appeared blunted in those with increased AD risk (i.e., E4 carriers). Relative to premenopausal females, postmenopausal females demonstrate reduced performance in terms of detailed memory, particularly for negatively-valenced stimuli, whereas memory for neutral or positive stimuli remained consistent across groups. Notably, these relationships were not observed with a more general memory measure. Upcoming analyses incorporating fMRI results may reveal underlying neurological contributions.

A-5. “SEX-SPECIFIC ADOLESCENT DEVELOPMENT OF SOCIAL REWARD MOTIVATION IN FEMALE AND MALE *OCTODON DEGUS*”

Shealin H. Murray¹, Amanda M. Leonetti¹, Andrew C. Sheehan¹, Cheryl M. McCormick¹, & Paula Duarte-Guterman¹

¹ Department of Psychology, Brock University, St. Catharines, ON.

Octodon degus are a small desert rodent known for their unique social behaviour profiles, and protracted development relative to other laboratory models. We have previously demonstrated that motivation for social reward fluctuates across adolescence in Long-Evans rats, and thus investigated the role of puberty onset on social motivation in juvenile male and female *degus*. We utilized an operant conditioning apparatus in which *degus* could “nose-poke” into two ports that opened a gate to allow five seconds of access to either a social reward (sex and age matched cagemate) or an empty chamber. On a fixed ratio (one nose poke = one reward) prepubertal males (~7 weeks old) demonstrated a social preference in gate opening, whereas there was no difference in gate opening for postpubertal males (6-8 months), or either age of females tested. However, on a progressive ratio (increasing number of nose-pokes required to access reward), irrespective of sex, prepubertal *degus* opened the social gate more than the nonsocial gate and had more overall openings than postpubertal *degus*. Lastly, we tested the animals in an extinction trial wherein no social reward was provided but both gates could be opened on a fixed ratio. In extinction, irrespective of age, males continued to open the previously social gate more than the nonsocial gate, whereas there was no gate opening preference in females. Therefore, we have demonstrated that in adolescent *degus* motivation for social reward is present prior to puberty and declines with maturation, and this occurs to a greater extent in male than in female *degus*.

A-6. “Pannexin1 Channels Regulate Synaptic Plasticity and Memory by Mapping Transcriptional Responses to Neuronal Activity in Larval Zebrafish”

Fatema Nakhuda^{1,2}, Georg R Zoidl^{1,2,3}

¹ Department of Biology, ² Centre for Vision Research, ³ Center for Integrated and Applied Neuroscience, York University, Toronto, ON M3J 1P3, Canada

Long-lasting memories are established through changes in neuronal connectivity driven by gene expression and protein synthesis, processes that are intricately regulated by neuroendocrine factors.

Our research has focused on the role of Pannexin1 (Panx1) channels in these processes, revealing their critical function in synaptic plasticity and memory formation. We have previously demonstrated that Panx1 ablation in mice leads to altered learning and memory. Recently, we found that Panx1 channels are integral to a signaling pathway that links NMDA receptors in chemical synapses to Connexin-36 in electrical synapses, both crucial for the modulation of learning and memory. In zebrafish, we explored the roles of two Panx1 ohnologs—Panx1a and Panx1b—using behavioral paradigms to assess memory in 6-day-old larvae. We observed that Panx1a ablation disrupts habituation to light stimuli, while Panx1b knockout does not. Early-stage memory testing indicated that Panx1a is essential for the transcriptional responses underlying memory formation. Pharmacological blocking suggests the interplay of Panx1a with GABAergic signalling to maintain excitation-inhibition balance. Subsequent in-vivo transcription and post-hoc fosab fluorescent labeling studies confirmed Panx1a's role in regulating gene expression during habituation. Our findings suggest that Panx1a modulates the excitation-inhibition balance critical for learning and memory, impacting the transcriptional regulation of these processes. This highlights Panx1a's potential as a target for influencing memory-related neuroendocrine pathways.

A-7. “Vasopressin-deficiency of juvenile Brattleboro rats alters behavioral arousal but not social preference in the 3-chamber preference test”

Grace C. Parmelee¹, Emma G. Bailey², Tyree L. Langley², Chloe N. Cordes², Kelcie C. Schatz², Matthew J. Paul^{1,2}

¹ Department of Neuroscience University at Buffalo, The State University of New York, ² Department of Psychology, University at Buffalo, The State University of New York

The neuropeptide, vasopressin (VP), plays a major role in regulating social behaviors of many species. The mechanism by which VP regulates social behavior, however, is not fully understood. Social behavior is complex and regulated by many processes, including social motivation, sensorimotor processing, cognition, and behavioral states. We previously found that juvenile Brattleboro rats, which lack VP due to a mutation in the VP gene, exhibit atypical social behavior characterized by decreased active social behaviors (e.g., play and ultrasonic vocalizations), but increased passive social behaviors (e.g., huddling). This finding raises the possibility that the absence of VP throughout development results in a hypoaroused phenotype that alters the type of social behavior exhibited, while leaving social motivation intact. The present experiment tested whether male and female juvenile Brattleboro rats exhibit deficits in their preference for social over non-social stimuli using the 3-chamber social preference test during the light phase (Exp. 1) or dark phase (Exp. 2) of the light/dark cycle. Brattleboro rats exhibited robust social preferences, the magnitude of which did not differ from wild type rats. Consistent with previous findings, Brattleboro rats exhibited decreased behavioral arousal as measured by decreased locomotor activity (Exp. 1) and decreased self-grooming (Exp. 2). While behavioral paradigms requiring greater effort to gain access to the social stimulus may uncover subtle deficits, present results are consistent with previous studies indicating that disruptions to VP do not impact social preference. The present findings are consistent with the hypothesis that VP regulates social behavior through actions on arousal.

A-8. “Androgens and Facial Emotion Recognition: Fear Perception Differences Between Women with and without Polycystic Ovary Syndrome Symptoms”

Shree Venkateshan¹, Kirsten Oinonen¹

¹ Lakehead University Psychology Department

Recent research suggests that androgens, influence facial emotion recognition. Over 80 percent of women with Polycystic Ovary Syndrome (PCOS) experience elevated androgen levels and androgenic symptoms such as hirsutism. However, there is a lack of research directly exploring the relationship between PCOS symptoms and emotion recognition. This study addressed this gap by investigating facial emotion recognition and self-reported PCOS symptoms. Women and men were asked to identify emotions (anger, disgust, fear, happiness, sadness, surprise, or neutral) in images of emotional facial expressions. PCOS symptom severity in women was assessed using self-report measures, including the Polycystic Ovary Syndrome Questionnaire (PCOSQ). Consistent with previous research, women were more accurate than men. Furthermore, individuals with a provisional PCOS diagnoses had significantly lower (less accurate) facial emotion recognition scores than those without provisional PCOS diagnoses. The largest difference was in fear recognition. A significant linear trend emerged for overall facial emotion recognition, revealing men as the least accurate, followed by women with provisional PCOS, and then women without PCOS. These findings are consistent with the theory that emotion recognition is affected by androgens, and may help with understanding mental health issues in women with PCOS symptoms.

A-9. “Resource scarcity alters postpartum maternal defense in rats”

Sydney Ku¹, Molly Dupuis², James Flowers II², Debra Bangsser¹

¹ Georgia State University, ² Temple University

Stress is a major risk factor for development of postpartum disorders, which can be mimicked using a limited bedding and nesting (LBN) manipulation. LBN dams and pups reside on a metal grate from postnatal day (PND) 2-10. Previously, we found that LBN increases dam's pup-directed behavior and decreases self-care. To determine if LBN impacts additional postpartum behaviors, we randomly assigned Long Evans dams (100-150 days old) to standard (n = 15) or LBN (n = 12) housing, then ran a resident-intruder task on PND10 with a male intruder to elicit aggression. LBN dams have lower total attack durations (p = 0.01), which is driven by significant decreases in offensive (pin, wrestle, shove; p = 0.01), but not defensive (kick, box, bite; p = 0.36) durations. LBN consistently decreases attack duration across all offensive measures, though only pin (p = 0.008) and wrestle (p = 0.03) are significant (shove; p = 0.49). We conducted whole-brain cFos to determine regions underlying these behavioral shifts. LBN dams show decreased cFos in the medial (p = 0.04) and lateral habenula (p = 0.06). Ongoing work utilizes immunohistochemistry to discern what types of neurons in LHB are driving our stress-induced MA effect.

A-10. “Early-life adversity alters microglial engulfment of excitatory synapses in the central nucleus of the amygdala in male and female mice”

Nneka C. Otuonye¹, Michelle K. Sequeira^{1,2}, Hannah D. Lichtenstein¹, Sara Correa¹, Peter Clements¹, Jessica L. Bolton¹

Early-life experiences are important for the typical development of organisms, and early-life adversity (ELA) is a risk factor for developing neuropsychiatric disorders later in life. The central nucleus of the amygdala (CeA) is a region of the brain that is responsive to stress. We recently discovered that ELA-induced anhedonia-like behaviors are mediated by corticotropin-releasing hormone (CRH) overexpression within the CeA. In another stress-related region of the brain, we discovered that decreases in microglial engulfment of excitatory synapses present on CRH+ neurons in the paraventricular nucleus of the hypothalamus contribute to the altered stress responsivity and hyperexcitability observed in male mice exposed to ELA. Here, we hypothesize that, like the PVN, microglial engulfment of excitatory synapses is impaired in the CeA by ELA. To test this hypothesis, we induced ELA in mice utilizing limited bedding and nesting from postnatal day (P)2-10. We used male and female CX3CR1-GFP; CRH-tdTomato mice to analyze microglial synaptic engulfment and excitatory synapse count through confocal microscopy and microglial 3-D reconstruction. We find that ELA increases the number of excitatory synapses present on the CeA-CRH+ neurons at P24/5. Additionally, microglial engulfment of excitatory synapses in the amygdala was decreased at P8. However, we discovered there are few to no CRH+ neurons located in the CeA at P8. We predict that there will be increased synapses and decreased microglial engulfment in the CeA by P14, a timepoint in which there are CRH+ neurons in the CeA.

A-11. “Potential Influences of Perinatal Exposure to Anti-Aging Supplements in *Rattus Norvegicus*”

Hallie Ruby¹, Victoria Riesgo¹, Ashley Chisnell¹, Jari Willing¹

¹ Bowling Green State University

Disorders due to cognitive aging are of continuous concern, resulting in the development of multiple “anti-aging” supplements. Currently, two supplements gaining prominence are nicotinamide mononucleotide (NMN) and resveratrol. Previous studies observed that, in animal models, both NMN and resveratrol slowed the cognitive aging process through a reduction of apoptosis. As research on these supplements continues, companies continue to market NMN and resveratrol as beneficial for overall brain health, which creates a diverse population of consumers. This population may include pregnant mothers, which raises concerns as apoptosis during early embryonic and postnatal development is critical in optimizing neural circuits. In the present study, we divided Long Evans dams into four exposure groups: NMN-exposed animals, resveratrol-exposed animals, animals exposed to both supplements, and control animals. Dosing for all groups began on embryonic day 0 and continued until postnatal day (P)10. Offspring underwent neonatal behavioral testing on P5, P7, and P10, while neonatal male and female brain tissue was collected on P12. Additional adult behavioral testing occurred on P80 to observe possible long-lasting effects on hippocampal and PFC-regulated behaviors. We also examined the effects of these supplements on reproductive success, body weight, and total brain weight. Current results support the idea the idea that perinatal exposure to these “anti-aging supplements” may alter neurodevelopment and neonatal and adult behaviors. Our findings may provide a better understanding of how these “anti-aging” supplements affect the mechanisms of neurodegenerative disorders and may influence future research models investigating neurodevelopmental disorders.

A-12. “Interplay Between Estrogen Receptors and the Oxytocin Receptor in Female Mouse Social Recognition”

Dante Cantini¹, Miji Cha¹, Catherine Schmidt¹, Elena Choleris¹

¹ University of Guelph Department of Psychology, Neuroscience and Applied Cognitive Science Program

In the social-brain-network the estrogen, 17 β -estradiol, has been shown to rapidly facilitate social recognition in mice. The medial amygdala of mice expresses the three known estrogen receptors: ER α , ER β , and G Protein-Coupled ER (GPER). Selective agonists for each of the three estrogen receptors rapidly facilitate social recognition in the medial amygdala of female CD1 mice. Oxytocin receptors within the medial amygdala are necessary for the rapid effects of 17 β -estradiol on social recognition to occur, suggesting a 17 β -estradiol/oxytocin receptor interplay. Our objective was to elucidate which of the estrogen receptors interplay with oxytocin receptors in the medial amygdala of female mice to rapidly facilitate social recognition. Female mice were ovariectomized and had bilateral cannulae implanted into the medial amygdala. A sub-effective dose of oxytocin receptor antagonist (the highest dose of the antagonist that per se does not inhibit social recognition) was infused into the medial amygdala before infusion of an estrogen receptor agonist, either ER α agonist PPT, or ER β agonist DPN, or GPER agonist G1). A social recognition paradigm designed to measure the rapid facilitating effects of treatment was administered. If the facilitating effect of the estrogen receptor agonist is impaired by the administration of the oxytocin receptor antagonist, we can infer that the specific estrogen receptor is implicated in the interplay with oxytocin receptors. A sub-effective dose of oxytocin receptor antagonist administered before each estrogen receptor agonist prevented the facilitation of social recognition by the agonist, demonstrating that each estrogen receptor interplays with oxytocin receptors in this process.

A-13. “Sex Differences in Neuronal Signaling Within Estrogen Receptors in the Hippocampus and its Effects on Memory Consolidation in Rodents”

Morales, C.J.¹, Fleischer, A.W.¹, Frick, K.M.¹

¹ Department of Psychology, University of Wisconsin-Milwaukee, Milwaukee, WI, 53211

Neuronal activity within the hippocampus underlies the formation and consolidation of episodic memories. Estrogens are crucial in lowering the threshold for excitatory neuronal signaling, which triggers molecular cascade responses within hippocampal cells. The exact localization of those effects in interneurons is not entirely understood. Thus, we incorporated several methods to pinpoint the cellular targets of estrogenic regulation of memory. Male and female mice either underwent gonadectomy surgery (GDX) and or sham surgeries, allowing us to explore sex and gonadal status differences in the expression of the major estrogen receptor isoforms, ER alpha and beta, in three interneuronal subtypes, Somatostatin (SST), Parvalbumin (PV) and Vasoactive Intestinal Peptide (VIP). Following the GDX or sham surgeries, the mice were handled and lavaged daily to track the estrous cycle in females; males were handled similarly to control for the lavage procedure in female mice. Females were euthanized via isoflurane overdose when the intact females were in phases of low circulating estrogen and had their brains collected and flash frozen. Both intact and GDX males were euthanized similarly near the end of their light cycle to capture low levels of circulating androgens. Pilot immunohistochemistry and RNAscope studies were conducted to optimize

antibodies for markers of different interneuronal populations and detect estrogen receptor subtypes, respectively. Having successfully optimized our protocols through the pilot studies, our next step is to localize the estrogen receptors' gene expression to different interneurons to pinpoint a specific site of action for estrogenic regulation of neuronal activity within the hippocampus.

A-14. “PARENTAL EXPERIENCE DIFFERENTIALLY REGULATES SPATIAL MEMORY AND ANXIETY-LIKE BEHAVIOUR IN FEMALE AND MALE *DEGUS*.”

Anton Dinh¹, Gurprince Attlas¹, Julia Mazur¹, Nicholas Gadea¹, Madeleine Maheu², Brian Cartmell¹, Dimitri Skandalis¹, Paula Duarte-Guterman¹

¹ Department of Psychology, Brock University, Canada, ² Department of Health Sciences, Brock University, Canada

The experience of being a parent leads to various neural, physiological, and behavioural adaptations. In rats, motherhood experience is associated with improved spatial learning and memory after the pups are weaned, while research is equivocal on anxiety. In biparental rodents, fathers show enhanced spatial learning in some studies but not others. To date, studies have focused on maternal experience in monoparental species and paternal experience in biparental species. The objective of this study is to compare the effects of parental experience on spatial cognition and anxiety-like behaviour in females and males of the same species, the degu. Degus are biparental rodents allowing us to examine parental experience in both sexes as well as differences between the motherhood experience of a biparental pair and single motherhood when the male is removed. Adult degus were randomly assigned to one of three groups: (1) breeding pairs (biparental group), (2) breeding with male partner removal (single mother group) or (3) non-breeding animals (naïve group). After weaning the pups, all adult degus were subjected to behavioural tests to assess spatial learning and memory (Barnes maze) and anxiety-like behaviours (elevated plus maze). Preliminary results suggest experienced mothers have enhanced spatial learning compared to naïve females, while fathers display impaired spatial memory and reduced anxiety-like behaviour compared to naïve males. This study suggests that parental experience differentially affects memory and anxiety in females and males. Future work will examine potential neural mechanisms and provide insights on how such an important life experience remodels the adult brain.

A-15. “Early-Life Behavior Phenotypes and Cortisol Responses to Common Lab Stressors in a Cichlid Fish”

Alyssa P. Alvey^{1,2}, Elijah R. Stern¹, June Lee³, Abigail Parrish³, Tessa Solomon-Lane¹⁻³

¹ Scripps College, ² Pitzer College, ³ Claremont McKenna College

The stress response is highly conserved across species, and a key component is increased glucocorticoid release (cortisol in fishes). In the highly social cichlid fish, Burton's Mouthbrooder (*Astatotilapia burtoni*), stress axis activity is associated with juvenile social behavior and status, and it mediates early-life social effects, yet little is known about early-life stress physiology. Here, we measured water-borne cortisol, a non-invasive method, in juveniles less than 1-week old. We first tested whether juveniles habituate to the beaker confinement necessary for collection. We found that repeated exposure to a beaker did not affect cortisol compared to handled and unhandled controls. In a separate cohort, we measured behavior in an open field exploration and social cue investigation, followed by a test of whether common lab stressors elevated cortisol. Controls were undisturbed in a

collection beaker (90 min). For the stressor treatments, we collected three sequential samples (30 min each) to try and quantify stress response peak and recovery; however, we found no effect of time. Stressors included handling, brief net confinement, and brief gentle movement. These stressors led to a significant increase in cortisol, with the highest levels resulting from confinement. The behavior tests revealed a bold-shy axis across both tests, describing the behavior of most individuals. A distinct group of socially-motivated juveniles showed a dramatic switch, staying in the territory during the open field but leaving to investigate the social cue. However, behavior did not predict cortisol levels. This work provides insight into early-life behavior and stress axis development and function.

A-16. “FUNCTIONAL SEX DIFFERENCES AND STRUCTURAL ANALYSES OF CORTICAL NORADRENERGIC SIGNALING IN C57BL/6J MICE”

Marcis V. Scroger¹, Alexandria C. Athanason¹, Noah Paperny¹, Andrea Liss¹, Mahum T. Siddiqi¹, Molly Batchelder¹, Anushree Karkhanis¹, Florence P. Varodayan¹

¹ Binghamton University-SUNY Behavioral Neuroscience Program, Department of Psychology, Binghamton, NY, 13902, USA

Norepinephrine (noradrenaline; NE) is a primary stress signal involved with regulating emotional and behavioral processes. NE is predominantly released from the locus coeruleus (LC), which innervates several structures including the medial prefrontal cortex (mPFC). Notably, the mPFC is divided into subcortices; the infralimbic subregion (IL) drives extinction of drug seeking, mood, and fear memory, and its prelimbic subregion (PL) promotes drug seeking, as well as working and episodic memory. While adrenergic receptors are expressed in both layer 2/3 and 5 of the mPFC, the specific effects of NE on mPFC activity across subregions, layers, and sexes, are not well understood. Therefore, we explored how NE affects glutamatergic neurotransmission in PL and IL layer 2/3 and 5 pyramidal neurons in naïve adult male and female C57BL/6J mice using ex vivo electrophysiology. We found that NE increased glutamate release in PL layer 5 and IL layer 2/3 in both sexes, but only in PL layer 2/3 and IL layer 5 in males. To assess how NE regulates mPFC-related cognitive behavior, we paired systemic injections of atipamezole, an α_2 adrenergic autoreceptor antagonist that increases NE release, with Barnes maze and novel object in place (NOIP) tasks. We found main effects of drug and sex during Barnes maze reversal, and trends towards similar main effects on the NOIP test day. We also assessed adrenergic receptor expression and basal NE levels in the mPFC that showed no differences. Current findings support that NE modulates the mPFC in a nuanced sex-dependent subregion- and layer-specific manner.

A-17. “Influence of Adolescent Stress on Behavior in Adulthood in an Alzheimer’s Disease Mouse Model”

Emily Groom¹, Christina Thrasher¹, Ava Herzog¹, Abigail Salinero¹, Charly Abi-Ghanem¹, Riane Richard¹, Damian Zuloaga², Kristen Zuloaga¹

¹ Department of Neuroscience and Experimental Therapeutics, Albany Medical College, Albany NY, 12208, ² Department of Psychology, University at Albany, Albany NY, 12222

Stress is a modifiable risk factor for AD and increases dementia risk 2-fold. During the stress response, the hypothalamic-pituitary adrenal (HPA) axis is activated which stimulates the release of

stress hormones called glucocorticoids. Studies on early-life stress (pre-adolescence) have shown a glucocorticoid dependent vulnerability towards late-life inflammation and neurodegenerative diseases. However, adolescence may be a more critical period for stress-induced vulnerability for late-life neurodegenerative diseases given that adolescents experience longer circulating levels of glucocorticoids following stress exposure as well as greater stress responsivity. Additionally, there are sex differences in AD prevalence and stress vulnerability with women being affected more than men. This suggests that there may be sex differences in stress-induced changes in AD pathology. We hypothesized that adolescent stress would exacerbate AD pathology later in life and that this effect will be stronger in females. Male and female Tg-SwDI mice underwent chronic social stress during adolescence. Mice were assessed at 2 and 6 months of age for anxiety-like behavior, cognitive function, and social behavior. Brains were collected at 6 months of age to assess neuropathology. Adolescent stress had a sex- and age-dependent effect on anxiety-like behavior and social behavior where females exhibited greater anxiety, and males exhibited greater social avoidance. Additionally, adolescent stress impaired spatial recognition memory in adulthood independent of sex. Neuropathology assessments are ongoing. This data is in line with clinical studies showing stress as a risk factor for AD and sex differences in anxiety-like behaviors and sociability.

A-18. “Unraveling the role of histone acetylation in adolescent reward processing disorders in male and female mice”

Sydney Zimmerman¹, Geramiah Drew¹, Peyton Bendis¹, Polymnia Georgiou¹

¹ Psychoneuroendocrinology Laboratory, Department of Psychology, University of Wisconsin Milwaukee, Milwaukee, WI, United States.

Reward processing disorders such as major depressive disorder afflicts ~13% of adolescents in the United States. Despite some effectiveness of the traditional antidepressant treatments, they often result in low remission rates and carry potential adverse effects. This emphasizes the need for novel pharmacotherapies for adolescent reward disorders. In response to this need, we conducted extensive bioinformatics analyses and drug repurposing screenings to identify drugs and compounds capable of reversing or mimicking the molecular signatures of adolescent depression, using RNAseq data from the human dorsolateral prefrontal cortex. Our study demonstrated that histone deacetylase inhibitors can reverse the molecular changes observed in adolescents with depression, indicating that increased histone acetylation affects gene expression. Notably, gene ontology analysis revealed significant downregulation of cholesterol metabolic and biosynthetic processes in adolescent patients compared to adults. Since cholesterol is a precursor for most steroid gonadal hormones, pathway enrichment analysis also showed downregulation in steroid biosynthesis. These results suggest that increased histone acetylation may lead to decreased synthesis of steroid gonadal hormones during adolescence, potentially increasing the risk of developing depression. To validate our findings, we subjected adolescent male and female mice to acute foot-shock stress and assessed the development of anhedonia, social preference deficits, and anxiety-like behavior using the sucrose preference, social interaction, and open field tests. Stressed adolescent mice exhibited social interaction deficits and anxiety-like behaviors, which were reversed by administering a histone deacetylase inhibitor before the acute stress. These findings indicate that targeting histone acetylation could represent a novel strategy for treating adolescent depression.

A-19. “Evaluating a Semi-Virtual Trier Social Stress Test: Evidence from Clinical & Non-clinical Samples of Young Adult Humans”

Madeline Divine¹, Melissa Miller¹, Ciara McAfee¹, Shelby Sears¹, Brandi Reynolds¹, Frances A. Champagne¹

¹ Department of Psychology, University of Texas at Austin, Austin, TX, USA

The Trier Social Stress Test (TSST) is a well-established protocol for inducing acute psychosocial stress which typically relies on an in-person interaction involving five people (including the participant). Here, we evaluate a modified semi-virtual version of the TSST (SV-TSST) that enhances the test's capacity for social distancing and reduces logistical barriers without the need for specialized equipment. Across two independent studies, participants (N = 87) underwent the SV-TSST, wherein the participant is guided through the baseline, anticipatory, challenge, and recovery phases of the test by an in-person experimenter, but confederate judges involved in the challenge phase of the protocol connect with the participant via video teleconference. Self-reported subjective stress and cortisol levels were assessed throughout the SV-TSST, including before and after the stressor to capture dynamic changes in stress reactivity. Across both studies, the SV-TSST protocol was found to induce a significant increase in subjective stress and salivary cortisol, with significant reductions in cortisol during the recovery phase. Results indicate that the SV-TSST is an effective protocol for acute stress induction that can serve as an alternative to the classic and virtual reality adaptations of this methodology. This adaptation has significant implications for research flexibility, allowing for broader participant inclusion and enhanced experimental control while maintaining robust stress induction. Further research should explore its applicability in diverse populations and settings.

A-20. “Sex differences in behavioral and physiological responses to chronic ethanol intake in ethanol dependent and non-dependent mice.”

Daniela Carrizales¹, Heather Aziz¹, Turner Lime¹, Daniel San Miguel¹, Regina Mangieri¹

¹ The University of Texas at Austin College of Pharmacy

Ethanol dependent and non-dependent male mice show behavioral differences and functional plasticity of nucleus accumbens (NAc) dopamine D1 receptor-expressing medium spiny neurons (D1MSNs). This study examined potential sex differences in ethanol intake using a 2-bottle choice model (2BC, 15% ethanol). Following 21 days of drinking, ethanol dependence was induced over 4 treatment cycles in which mice were exposed to chronic intermittent ethanol (CIE) vapor, no treatment (3 days), and 2BC testing (5 days). Ethanol non-dependent and naïve mice received air, rather than CIE vapor. 24 hours after concluding CIE/air vapor exposure, brain slices were prepared for whole cell patch clamp electrophysiological recordings to measure NAc D1MSNs membrane properties and excitability. Both male and female ethanol experienced mice demonstrated escalating ethanol consumption compared to their baselines, however, only male CIE mice showed greater escalation relative to male air mice, indicative of ethanol dependence. Although male CIE mice maintained expected consistent CIE vapor-induced blood ethanol concentrations (BECs) throughout the experiment, female CIE mice BECs escalated throughout vapor exposure, confounding interpretation of the female behavioral data. Regardless, Sex by Condition interactions in 2-way ANOVAs were found for both threshold potential and input resistance. Female CIE mice had

significantly decreased threshold potential and input resistance compared to female non-dependent mice; opposite trends were observed for male mice. Although sex differences were found in both behavioral and physiological responses to chronic ethanol exposure, providing consistent ethanol vapor exposure for both sexes in additional cohorts of mice would facilitate interpretation of findings in future studies.

A-21. “Neuronal Cilia Gene Expression Across Different Social Experiences in the Monogamous Prairie Vole (*Microtus ochrogaster*)”

Sabrina L. Mederos¹, Albatool Al Khazal², Adele A. M. Seelke², Karen L. Bales²

¹ University of California, Davis Animal Behavior Graduate Group, ² University of California, Davis Psychology Department
Primary cilia are microtubule-based organelles that project from most vertebrate cells, extending into the extracellular space and serve as key sites for cell communication. This study investigates the gene expression of cilia-related and other genes related to social behavior in response to different social experiences in the monogamous prairie vole, a well-studied translational model for studying the neurobiology of pair bonding. Previous comparative research in our lab found distant species that share a monogamous mating system, seahorses and prairie voles, have significantly similar gene expression patterns in cilia-related genes. Here, prairie voles were divided into same-sex sibling pairs and male-female breeder pairs to explore how different social environments influence gene expression in the brain. After 20 days of cohabitation, brains were extracted, sliced, and analyzed using a fluorescent in situ hybridization (HiPlex) assay. The analysis focused on the nucleus accumbens, a critical region for social bonding, and examined the expression of genes related to neuronal primary cilia, and others (such as oxytocin, vasopressin, and prolactin). The results of this study provide novel insights into the molecular mechanisms underlying social behavior, highlighting the role of primary cilia and other key signaling pathways in the regulation of pair bonding. This research contributes to a broader understanding of the neurobiological basis of social behaviors and the importance of cilia-related signaling in the brain.

A-22. “Early resource scarcity in rats leads to female-specific changes in prefrontal cortex neurovascular gene expression”

E. ANDREWS¹, E. HARRIS¹, S.N. CHEHIMI², R.C. CRIST², M.WIMMER³, D. BANGASSER¹; B.C. REINER²

¹ Neuroscience Institute, Georgia State Univ., Atlanta, GA; ² Dept. Psychiatry, Univ., Philadelphia, PA; ³ Dept. Psych and Neuro., Temple Univ., Philadelphia, PA

Early resource scarcity is a risk factor for psychiatric disorders. In rats, this is modeled through a limited bedding and nesting manipulation (LBN) where dams and offspring are exposed to limited resources from PND 2-9. This is a sensitive period of brain development where changes in the environment have lasting effects into adulthood. In fact, we have previously demonstrated that LBN changes cognition and motivated behavior that relies on medial prefrontal cortex (mPFC) in adult rats, often in sex-dependent ways. To investigate the persistent effects LBN has on mPFC, we used single nucleus RNAseq to measure changes in cell-type gene expression compared to control male and female rats. Nuclei are clustered into 18 known cell types. Clusters included excitatory neurons, inhibitory neurons, neuroglia, and vasculature. We compared differentially expressed genes (DEGs)

within sex such that control vs LBN were compared for males and separately for females. In general, we found LBN caused more transcriptional changes in females than males across most clusters. One cluster that showed transcriptional changes in LBN females was vasculature. To follow up on this data we plan to use qPCR to investigate blood brain barrier (BBB) integrity. These results provide insight into blood brain barrier permeability suggesting an impact of LBN on BBB integrity in females. Few studies have investigated early life adversity and its effects on the developing neurovascular environment, which may be an important yet unexplored mechanism of female vulnerability to stress.

B-1. “Exploring Biomarkers for the Menopausal Status within Human Post-Mortem Pituitary Gland Tissue”

Anaya Ger¹, Maria Tickerhoof¹, Marija Kundakovic¹

¹ Department of Biological Sciences, Fordham University

Physiological changes during the menopausal transition occur for years prior to the cessation of menstruation. The decrease in ovarian hormone levels triggers compensatory changes in regulatory hormones in the hypothalamus and pituitary gland. These hormonal fluctuations increase female risk for neuropsychiatric disorders. The pituitary gland, playing a central role in regulating ovarian hormone release, still remains under-explored in reproductive aging research. The Kundakovic lab previously studied 39 biomarkers in human post-mortem blood, hypothalamic, and pituitary tissues. Specifically, FSH protein levels and FSH and GNRHR gene expression in the pituitary were strong markers for classifying samples as pre- or post-menopausal. This project aims to integrate pituitary-specific markers into a single measure for classifying menopausal status in forty-two post-mortem pituitary samples from the Human Brain Collection Core, initially categorized by age as pre- (<40 years), peri- (45-55 years), or post-menopausal (>55 years). The expression of genes involved in regulating sex hormone release at the level of the pituitary, including TGFBR3L, FST, LHB, and AMHR2, was examined using qRT-PCR. Among those, TGFBR3L showed significant differences in expression between the pre- and post-menopausal groups, proving to be another good marker for predicting menopausal status. Employing Principal Component Analysis, FSH protein, FSH, GNRHR, and TGFBR3L gene expression were combined into a single component score to classify samples as pre- or post-menopausal-like. The results from this project aids in classifying the menopausal status of post-mortem human brain tissue, which is critical in further investigating the effect of the menopausal transition on female risk for psychiatric disorders.

B-2. “Assessing the Effects of Prenatal Alcohol Exposure on Emotionality in Sprague Dawley Rats”

Kingston Wong¹, Dustin Cordeiro¹, Landon Daschuk¹, Matthew Epp¹, Kanishka Wijesundara¹, Sunny Qureshi¹, Victoria Vella¹, Carolina Luft¹, Tamara Bodnar², Parker Holman¹, Charlis Raineke¹

¹ Department of Psychology, Brock University, St. Catharines, Ontario, Canada. ² Department of Biological Sciences, University of Calgary, Calgary, Alberta, Canada

Individuals affected by Fetal Alcohol Spectrum Disorder show increased risk for mental health issues and social behavior deficits. This is reflected in the preclinical literature where Prenatal Alcohol Exposure (PAE) has shown to induce anxiety- and depressive-like behaviors as well as deficits in

social interaction. However, changes in emotional and social behavior are often assessed individually which prevents evaluating the effect of PAE on overall emotional status. This study aims to examine the effect of PAE on overall emotional status using an emotionality score - which includes domains such as anxiety, depression, and social interaction. Pregnant dams were randomly assigned to: PAE – liquid ethanol diet ad libitum or Control – pelleted control diet ad libitum. Adult male and female rats were evaluated for anxiety-like behavior through the open field and dark-light emergence tests, depressive-like behavior through the sucrose preference test, and social behavior through the social interaction test. Principal component analysis was used to identify correlated scores across all behavioral assays which included duration spent in the center, duration spent in and latency to the light chamber, ratio of sucrose preference, and non-affiliative exploration. These scores were compiled into a single emotionality score where higher scores indicated poorer emotionality. Male animals had significantly higher emotionality scores than female animals. Regardless of sex, PAE animals had significantly higher emotionality scores compared to control animals. These findings support the use of an emotionality score for assessment of socioemotional domains affected by PAE.

B-3. “Investigating Differences In Activation of the Vasopressin Neural System Between Soldier and Worker Naked Mole-Rats”

Firaas Mir¹, Xinye Peng¹, Melissa Holmes¹

¹ University of Toronto Mississauga Department of Psychology

The naked mole-rat (*Heterocephalus glaber*) is a eusocial rodent species characterized by a highly organized social hierarchy. Among its hierarchy is the nonreproductive subordinate caste, which can be further subdivided into subcastes. Members of the aggressive soldier subcaste defend the colony from foreign threats while nonaggressive, prosocial workers maintain the colony through tasks such as alloparenting of pups. In other rodent species, the neuropeptide arginine vasopressin (AVP) has been shown to promote aggressive behaviour towards unfamiliar conspecifics in a species and sex-dependent manner. To determine if AVP also plays a role in modulating the characteristic aggressive behaviour of soldier naked mole-rats towards unfamiliar conspecifics, soldiers and workers were paired with either a familiar or unfamiliar same-sex conspecific (also a worker), and their interaction was recorded and scored for aggressive behaviours with BORIS. Following the interaction, brains were extracted and immunohistochemistry was conducted to label the immediate-early-gene c-Fos. C-Fos density was used to quantify neural activation in parts of the vasopressin neural circuitry, which included the hypothalamic paraventricular nucleus (PVN), supraoptic nucleus (SON), anterior hypothalamus (AH), and ventromedial hypothalamus (VMH). At present, results are inconclusive as behavioural analyses and comparisons of c-Fos density of these areas have not been completed, though preliminary data suggests generally greater c-Fos density in these vasopressin-related regions in soldiers relative to workers.

B-4. “Are You Hurt? Exploring the Role of Opioid Receptors in Female Rodent Social Approach Behaviour Towards Siblings in Pain”

Zainab Zakaria¹, Navdeep Lidhar¹, Crystal Mui¹, Sana Khan¹, Loren J. Martin¹

¹ University of Toronto Mississauga, Department of Psychology

Observing someone in pain often compels us to approach and offer comfort. The social relationship and biological sex of both the observer and the individual in pain are key determinants of this behaviour, yet the underlying neurological mechanisms remain poorly understood. In this study, we used a social affective preference (SAP) test with CD-1 mice to examine their preference for approaching a demonstrator mouse in pain (induced by intraperitoneal injection of 0.9% acetic acid) versus a demonstrator mouse not in pain. The SAP test was conducted under two conditions: one with sibling mice and the other with stranger mice. We found a sex-specific effect, whereby female, but not male, observers spent significantly more time investigating their siblings in pain compared to those not in pain. No significant preference was observed in either male or female mice towards strangers, regardless of pain status. Given the observed effect of social relationship and pain status exclusively in female mice, we tested the role of the opioid system by administering opioid antagonists to female observers prior to the SAP test. This treatment reversed their preference for siblings in pain, suggesting that the endogenous opioid receptor system plays an important role in modulating social approach behaviours toward conspecifics in pain.

B-5. “The Role of the Hypothalamus in Tamoxifen-Induced Hot Flashes in Female Mice”

Hannah Azadi¹, Jae Park¹, Weronika Budek¹, Amanda Misque¹, Stephanie Correa¹, Ed van Veen¹

¹ UCLA Department of Integrative Biology and Physiology

Tamoxifen is a drug used as a post-surgical adjuvant therapy in patients with estrogen receptor positive breast cancer. It is classified as a selective estrogen receptor modulator, meaning it binds to estrogen receptors to modulate cellular activity. Many patients who take tamoxifen report experiencing hot flashes, which are acute episodes consisting of a decrease in core body temperature and an increase in skin temperature. Disruptive hot flashes can decrease quality of life and cause many patients to stop taking tamoxifen. It has been shown in mice that hypothalamic expression of estrogen receptor alpha (ER α) is required for tamoxifen-induced thermal dysregulation. Although, the specific hypothalamic nuclei and the mechanism in which tamoxifen acts to cause these side effects is currently unknown. We hypothesized that tamoxifen acts through ER α to cause transcriptional changes, including modifying estrogen response element (ERE)-dependent gene transcription and Tac2 gene expression in the arcuate nucleus. To test this hypothesis, we observed the effects of tamoxifen administration on both ERE-dependent gene transcription and Tac2 mRNA expression in the arcuate nucleus. Tamoxifen administration was not associated with significant changes in transcription through either assay. Future experiments will investigate whether the mechanism in which tamoxifen exerts its effects is occurring through protein signaling rather than nuclear transcriptional mechanisms. This will include looking at neurokinin B protein expression as well as membrane ER α signaling. By understanding this mechanism, combination therapies may be developed to counteract the negative side effects experienced, while maintaining tamoxifen's ability to prevent the recurrence of breast cancer.

B-6. “The Effect of Thyroid Hormone Levels on Brain-wide Responses to Acute Social Stressors in Male Mice”

Rexhebi Kadriu¹, Jovian Cheung¹, Sanjeev Janarthanan¹, Laura Lynch¹, Alissa Le¹, Danielle Roberts¹, Shannon Bennett¹, Annegret Falkner¹, Catherine Peña¹

¹ Princeton University Neuroscience Institute

Thyroid dysfunction during adulthood affects behavior, while early life suppression could have effects on brain development that also lead to later behavioral changes. Specifically, thyroid hormone production contributes to brain development and thyroid hormone levels in adulthood are known to influence mood, although little is known about how different brain circuits may be impacted by developmental or adult thyroid dysregulation. This project focuses on the manipulation of thyroid hormones and its effects on brain-wide responses towards acute stressors. We hypothesize that suppression of thyroid hormone levels, either during early life and/or in adulthood, alters brain circuit-level response to social defeat stress in mice.

B-7. “Social Novelty Representations in the Ventral Hippocampus of Male Mice”

Benjamin Dykstra¹, Gordon Berman¹, Malavika Murugan¹

¹ Emory University

Prior research has determined that the ventral hippocampus is necessary for mice to preferentially investigate novel conspecifics over familiar conspecifics. However, few studies have recorded endogenous activity in the ventral hippocampus during social interactions so it largely remains unknown what social information the ventral hippocampus encodes. We hypothesize that the ventral hippocampus encodes social recognition information and contains neurons that preferentially fire for novel and familiar conspecifics. To test this hypothesis, we performed one-photon calcium imaging to record endogenous activity in the ventral CA1 and ventral CA3 subfields of the hippocampus in male mice. We then developed a novel behavioral assay called the linear approach assay in which novel and familiar conspecifics are presented to the imaging mice at the same spatial location. This approach is critical to disentangle social from spatial encoding in the hippocampus. We trained support vector machine classifiers to decode social targets from population neural activity. Support vector machine classifiers were unable to decode two novel conspecifics above chance levels however they were able to decode novel and familiar conspecifics above chance levels. Interestingly, decoding accuracy is significantly higher in the ventral CA3 compared to the ventral CA1. Additionally, individual ventral hippocampus neurons preferentially fired for both novel and familiar conspecifics. Together, these results provide evidence that the ventral hippocampus encodes social novelty. Additionally, this research suggests that there are functional differences in the ventral CA1 and ventral CA3 with the latter encoding social recognition information more robustly.

B-8. “Neuroendocrine Circuit in Mice for Sleep-Dependent Growth Hormone Release”

Xinlu Ding¹, Fuu-Jiun Hwang², Daniel Silverman¹, Peng Zhong^{1,4}, Bing Li¹, Chenyan Ma¹, Lihui Lu¹, Grace Jiang¹, Zhe Zhang^{1,5}, Xun Tu¹, Melissa Tian¹, Jun Ding^{2,3}, and Yang Dan¹

¹ Division of Neurobiology, Department of Molecular and Cell Biology, Helen Wills Neuroscience Institute, Howard Hughes Medical Institute, University of California; Berkeley, CA 94720, USA. ² Department of Neurosurgery, Stanford University; Stanford, CA 94305, USA. ³ Department of Neurology and Neurological Science, Stanford University; Stanford, CA 94305, USA. ⁴ Present address: Department of Neurological Sciences, University of Nebraska Medical Center; Omaha, NE 68106, USA. ⁵ Present address: Institute of Neuroscience, State Key Laboratory of Neuroscience, Key Laboratory of

Growth hormone (GH), which stimulates tissue growth and regulates metabolism, is released predominantly during sleep, but the underlying circuit mechanism is unknown. We demonstrate how GH release, which is markedly enhanced during both rapid eye movement (REM) and non-REM (NREM) sleep, is regulated by sleep-wake-dependent activity of distinct hypothalamic neurons expressing GH-releasing hormone (GHRH) and somatostatin (SST). SST neurons in the arcuate nucleus suppress GH release by inhibiting nearby GHRH neurons that stimulate GH release, whereas periventricular SST neurons inhibit GH release by projecting to the median eminence. GH release is associated with strong surges of both GHRH and SST activity during REM sleep but moderately increased GHRH and decreased SST activity during NREM sleep. Furthermore, we identified a novel feedback pathway, in which GH enhances the excitability of locus coeruleus noradrenergic neurons and suppresses sleep. These results elucidate a circuit mechanism underlying bidirectional interactions between sleep and hormone regulation.

B-9. “The effect of estrogen on the expression of metabolic genes in the hypothalamus of female mice”

Liz Leeser¹, Laila Ouldibbat¹, Marija Kundakovic¹

¹ Department of Biological Sciences, Fordham University

Obesity and eating disorders are more frequent in women than in men, although this sex difference is underexplored. Studies have revealed that women become more susceptible to metabolic dysfunction and obesity as estrogen levels decline. However, the molecular mechanisms driving this sex disparity remain poorly understood. To better understand mechanisms through which estrogen impacts metabolic function in females, we studied ovariectomized (OVX), estrogen-depleted female mice undergoing “cyclic”, chronic estradiol treatment alongside sham surgery (ovary-intact) females. The examined groups included OVX mice receiving 1 µg of estradiol benzoate (EB) every 4 days for 6 weeks (OVX-EB-1µg); OVX mice receiving vehicle injections of corn oil (OVX-Veh), and sham (ovary-intact) females. Using quantitative qRT-PCR, we analyzed the expression of three candidate genes, *Mc3r*, *Fto*, and *Ncoa1*, in the hypothalamus, a brain region involved in energy homeostasis and metabolism. *Mc3r* encodes G protein-coupled receptors that regulate energy expenditure; *Fto* mutations have been linked to obesity risk; and *Ncoa1* encodes a steroid-receptor coactivator that modulates energy balance. All three genes are involved in estrogen-mediated metabolism; however, they have not been studied using the ovariectomy and estrogen replacement model. We found no significant differences in gene expression between OVX-Veh, OVX-1µg, and sham groups; however, a trend of higher expression in OVX-EB-1µg compared to OVX-Veh and the sham cycling group was observed for all genes. Given hypothalamic heterogeneity, our results indicate that the higher-resolution, single-cell gene expression assays may be more suitable for further investigation into the molecular mechanisms of estrogen-driven, metabolically-relevant gene regulation in the hypothalamus.

B-10. N/A

B-11. “Impact of Prenatal Alcohol Exposure on Cognitive and Immune Signaling in Aging Male and Female Sprague Dawley Rats: A Longitudinal Study”

Sunny Qureshi¹, Carolina Luft¹, Madeleine Maheu¹, Jordan Albanese¹, Kingston Wong¹, Victoria Vella¹, Parker J. Holman¹, Tamara S. Bodnar², Paula Duarte Guterman¹, Charlis Raineke¹

¹ Department of Psychology, Brock University, St. Catharines, ON, Canada. ² Department of Biological Sciences, University of Calgary, Calgary, AB, Canada

Prenatal alcohol exposure (PAE) can lead to long-lasting detrimental effects on the developing brain, resulting in cognitive deficits and alterations in immune function. However, the progression of these deficits with aging and their relationship to sex-specific changes remains understudied. To explore this, we utilized a PAE rat model to examine the relationship between immune function, cognitive performance, and sex differences in aging animals. Pregnant rats were randomly assigned to either an ad libitum PAE (liquid ethanol diet) or control (pelleted diet) group throughout gestation. Their offspring were tested on the Barnes Maze and Novel Object Recognition (NOR) tasks at 6 (6M) and 12 months (12M) of age. Blood was collected for circulating cytokine measurement and brains were collected for immunofluorescence of microglia (Iba-1). NOR analysis revealed that by 6M, PAE females exhibited recognition memory deficits which continued into 12M of age. However, PAE males only displayed these deficits at 12M of age. Barnes maze analysis showed no differences in spatial learning and memory, however, age-related improvements were observed with repeated testing. Cytokine data showed that PAE males had lower levels of IL-4 at both ages, while PAE females exhibited reduced IL-4 levels only at 12 months. Additionally, at 12M, PAE females displayed significantly lower levels of IL-10, IL-13, and IFN- γ compared to controls, suggesting sex-specific alterations in immune signaling. Furthermore, microglia analysis revealed an increased number of microglia in the hippocampus only in PAE females at 12M of age, indicating potential sex-dependent neuroinflammatory mechanisms contributing to cognitive decline.

B-12. “Shifts in ethanol drinking mediated through bidirectional fecal microbial transplant between alcohol preferring and non-preferring rats”

Sudhan Pachhain¹, Brittany Halverstadt², Ekenedilichukwu Anekwe¹, Vipaporn Phuntumart¹, Jon Sprague³, Christopher Ward¹, Howard Cromwell²

¹ Department of Biological Sciences, Bowling Green State University, Ohio, USA, ² Department of Psychology, Bowling Green State University, Ohio, USA, ³ Ohio Attorney General's Center for the Future of Forensic Sciences, Bowling Green State University, Ohio, USA

Alcohol Use Disorders (AUDs) constitute a global health crisis with limited effective treatments. Alterations in gut microbiome have been shown to be major contributors to substance use disorder and mental illness. Fecal Microbial Transfer (FMT) is emerging as a promising method for manipulating the Brain-Gut-Microbiome Axis in diverse health and disease states including AUD and has potential as a clinical treatment. The well characterized behavioral genetics of Alcohol Preferring (P) and Non-Preferring (NP) rat model offer valuable insights into the underlying mechanisms of AUD. In the present study, an FMT between P and NP rats was utilized to explore influences of gut microbiome composition on ethanol drinking behaviors. NP rats exhibited significantly lower ethanol consumption and preference compared to P rats, throughout pre, during and post-FMT treatment.

Following FMT, significant differences in drinking behaviors were found among testing weeks indicating P rats reduced ethanol consumption post-FMT. Microbiome analysis showed that P rat ethanol-induced dysbiosis involved increased relative abundance of *Akkermansia muciniphila* and reduced Bacteroidetes and Lactobacilli while the FMT treatment altered their levels. Gene abundance analysis resulted in an increase in alcohol metabolizing genes in P rats following three weeks ethanol access, indicating that the gut favors alcohol metabolizers after ethanol intake, which significantly decreased after FMT. The findings of distinct microbial communities and alcohol consumption phenotypes in P and NP rats underscore the systemic interaction between the gut microbiome and neurobehavior. FMT effects obtained highlight its potential as an innovative clinical approach for addressing AUD and mental illness.

B-13. “A Role for De Novo mPFC Estradiol Synthesis in Spatial and Object Recognition Memory Consolidation in Female Mice”

Miranda R. Schwabe¹, Hailey A. Beaty¹, Elaina M. Milkie¹, Karyn M. Frick¹

¹ Department of Psychology, University of Wisconsin - Milwaukee

De novo E2 synthesis of the hormone 17- β estradiol (E2) via the enzyme aromatase in dorsal hippocampus is essential for object recognition (OR) and object placement (OP) memory consolidation in ovariectomized (OVX) female mice. Other regions, such as medial prefrontal cortex (mPFC), play critical roles in these types of memory, but less is known about the involvement of mPFC E2 synthesis in memory. Because exogenous E2 infusion in the mPFC enhances OR and OP memory consolidation, we hypothesized that de novo E2 synthesis in mPFC is likely also necessary for memory consolidation. Female C57BL/6 mice (8-9 weeks of age) were OVXed and implanted with cannulae targeting mPFC. After recovery, mice underwent OR and OP testing. Immediately after object training, mice received an infusion of vehicle (1% DMSO in saline) or the aromatase-inhibitor letrozole (0.025 μ g or 0.05 μ g/hemisphere). Results indicate that letrozole-infused mice exhibit impaired OR and OP memory consolidation relative to controls, supporting our hypothesis. We next investigated the effect of object training on aromatase-expressing cells in mPFC. A separate cohort of female mice were OVXed or underwent sham OVX, because the presence of ovarian hormones may affect aromatase expression. Mice were exposed to a session of object training (or remained in homecage as control) then euthanized 1-hour post-training. In mPFC tissue, cells expressing the gene *cyp19A1* were identified via RNAscope and c-fos protein as a marker of activation by training. Data indicate that *cyp19A1* expression increases after training in sham, but not ovx mice.

B-14. “The fluid inhibitory effect of estradiol is not associated with an elevation in excitatory cell activation in the subfornical organ of the female rat.”

Lasni Nishshanke¹, Andrea Edwards¹, Jessica Santollo¹

¹ Department of Biology, University of Kentucky, Lexington, KY 40506

Estradiol's (E2) inhibition of water intake is well documented, yet the underlying mechanisms remain unclear. To understand how E2 treatment decreases water intake in female rats following water deprivation, this study investigated the associated drinking microstructure changes and explored whether E2 modulates the activation of excitatory cells in the SFO, which are associated with stimulating drinking during dehydration. Ovariectomized rats were administered estradiol benzoate

(EB) and underwent 24 hours of water deprivation. Water intake and licks were then monitored for 2 hours. In a second group, brains were extracted and processed for FOS and CaMKII to examine neuronal activity in the excitatory cells in the SFO. As expected, EB treatment reduced intake compared to oil controls. This occurred through a decrease in burst number ($p < 0.05$), while burst size remained unaffected. Furthermore, water deprivation increased FOS expression in the SFO, and water-deprived rats exhibited a higher number of CaMKII cells expressing FOS ($p < 0.05$). This response, however, was mediated by the oil-treated rats. EB-treated rats showed no elevation in FOS expression in the CaMKII cells due to an unexpected increase in activated CaMKII cells in the non-deprived state. Despite EB decreasing water intake in water deprived rats, corresponding excitatory cell activation was not significantly different between the groups. The decreased burst number in EB-treated rats suggests that E2 influences water intake through alterations in post-ingestive signaling, indicating that satiety signals, instead of the cells that drive intake, may be the target of E2, which future studies will address.

B-15. “Postnatal Maternal Care Interacts with Prenatal Bisphenol Exposure on Esrrg Gene Expression, Co-Expressed Gene Profiles, and DNA Methylation in the Developing Hypothalamus of Female Rat Offspring”

Samantha Lauby^{1,2}, and Frances Champagne^{1,2}

¹ University of Texas at Austin Department of Psychology, ² University of Texas at Austin Center for Molecular Carcinogenesis and Toxicology

Environmental exposures co-occurring during early-life have a profound influence on neurodevelopment. Our previous work in rats suggests that postnatal maternal care modulates the effects of prenatal exposure to bisphenols, an estrogenic endocrine disrupting chemical, on offspring neurodevelopment. High postnatal maternal care and prenatal bisphenol exposure have known opposing effects on estrogen receptor alpha (Esr1) expression in the medial preoptic area (MPOA) of the hypothalamus. We hypothesized that postnatal maternal care would interact with prenatal bisphenol exposure on transcription profiles in the developing MPOA, including Esr1, and affect DNA methylation modifications downstream. We administered two doses (50 ug/kg/day and 150 ug/kg/day) of a bisphenol mixture (BPA, BPF, BPS) and a corn oil control to pregnant rat dams. Postnatal maternal care was measured using home-cage videos. We found a significant interaction between the 150 ug/kg bisphenol group and maternal care (compared to controls) on estrogen-related receptor gamma (Esrrg) expression but not Esr1 in females. We also found interactions between the 150 ug/kg bisphenol group and maternal care on five co-expressed gene profiles in females. Four co-expressed gene profiles were enriched for estrogen-responsive genes. Finally, we found substantial influences of postnatal maternal care on differentially methylated regions in all prenatal bisphenol-treated groups in males and females. Overall, this work suggests that postnatal maternal care influences Esrrg expression, genome-wide transcription profiles, and DNA methylation modifications in the MPOA of bisphenol-exposed female offspring. Since Esrrg has a high affinity for BPA, it will be important to explore the downstream consequences of Esrrg activity.

B-16. “Effects of Chronic Stress on Avoidance Behavior in the Estrogen Receptor Alpha Knockout (ERKO) Mouse”

Victoria Appel¹, Troy A. Roepke¹

¹ Department of Animal Sciences, School of Environmental & Biological Sciences, Rutgers University, New Brunswick, NJ

Chronic stress has long been implicated in the development of mood disorders. Research has demonstrated that cisgender women are more susceptible than cisgender men to developing such conditions, and that these sex differences may, in part, be attributed to endocrine pathways. The mechanisms behind these pathways are progressively being elucidated, with focus on estrogen signaling. Using estrogen receptor alpha knockout (ERKO) mice, we examined the role of chronic stress on avoidance behavior across the sexes. Experimental mice underwent six weeks of chronic variable mild stress (CVMS) to induce a depressive phenotype, while control mice were not stressed. Four behavior tests were used to assess avoidance behavior: the open field test, elevated plus maze, light-dark box, and novelty suppressed feeding. We demonstrated effects of sex and stress on behavior where chronic stress has an anxiogenic effect in both sexes, that was more prominent in females. When compared to our published work in wild-type mice, our findings suggest that the deletion of estrogen receptor alpha increases the susceptibility to chronic stress, which may also involve estrogen receptor beta or G-protein estrogen receptor 1 to elicit the observed avoidant responses. This work lends itself to further investigation into the intersection of chronic stress and steroid hormone signaling pathways.

B-17. “Maternal gut Microbiome in Human Females is Associated with Somatic Symptoms During Pregnancy”

Brooke DeRonne¹, Nicolas Murgueitio¹, Michelle Graf¹, Sarah Short², Rebecca Knickmeyer³, Cathi Propper¹

¹ University of North Carolina at Chapel Hill, ² University of Wisconsin-Madison, ³ Michigan State University

Pregnancy is a period with a high prevalence of mental health problems, including somatization (i.e., the expression of psychological or emotional factors as physical symptoms). Pregnancy necessitates emotional and physical adaptations, which increase the potential for somatization. The gut microbiome, a key link in the brain-body connection, also undergoes adaptations in pregnancy, and has been associated with somatic symptoms in healthy and clinical populations. The present study investigated associations between the prenatal microbiome and maternal somatic symptoms. 180 pregnant women reported somatic symptoms via a web-based questionnaire and provided fecal samples. Differential abundance analyses assessed associations between taxonomic genera abundance and symptomology. P-values were corrected for multiple comparisons using the Benjamin-Hochberg method, with a q-value threshold for significance of .25. Multiple linear regressions were conducted to assess the association between alpha diversity and somatic symptoms. All analyses controlled for diet, medication use, and illness. Several genera were significantly associated with somatic symptoms including *Parascardovia* (q = .09), *Lawsonella* (q = .13), *Nissabacter* (q = .13), and *Plesiomonas* (q = .14). In summary, the gut microbiome might influence a subset of mental health symptoms, somatization, via the presence and abundance of specific genera. Specific species of bacteria within the *Lawsonella* genus have been associated with rare cardiovascular diseases. Species within the *Plesiomonas* genus have been associated with central nervous system pathology. Therefore, gut bacteria that drive tissue dysfunction might contribute to or exacerbate somatization during pregnancy.

B-18. “Sex differences in offspring risk and resilience following 11 β -hydroxylase antagonism in a rodent model of maternal immune activation”

Julia Martz¹, Micah A Shelton², Laurel Geist¹, Marianne L Seney², Amanda C Kentner¹

¹ Massachusetts College of Pharmacy and Health Sciences School of Arts & Sciences, ² University of Pittsburgh
Department of Psychiatry

Maternal immune activation (MIA) puts offspring at greater risk for neurodevelopmental disorders associated with impaired social behavior. While it is known that immune signaling through maternal, placental, and fetal compartments contributes to these phenotypical changes, it is unknown to what extent the stress response to illness is involved and how it can be harnessed for potential interventions. To this end, on gestational day 15, pregnant rat dams were administered the bacterial mimetic lipopolysaccharide (LPS; to induce MIA) alongside metyrapone, a clinically available 11 β -hydroxylase (11 β HSD) inhibitor used to treat hypercortisolism in pregnant, lactating, and neonatal populations. Maternal, placental, and fetal brain levels of corticosterone and placental 11 β HSD enzymes type 1 and 2 were measured 3-hrs post treatment. Offspring social behaviors were evaluated across critical phases of development. MIA was associated with increased maternal, placental, and fetal brain corticosterone concentrations that were diminished with metyrapone exposure. Metyrapone protected against reductions in placental 11 β HSD2 in males only, suggesting that less corticosterone was inactivated in female placentas. Behaviorally, metyrapone-exposure attenuated MIA-induced social disruptions in juvenile, adolescent, and adult males, while females were unaffected or performed worse. Metyrapone-exposure reversed MIA-induced transcriptional changes in monoamine-, glutamate-, and GABA-related genes in adult male ventral hippocampus, but not in females. Taken together, these findings illustrate that MIA-induced HPA responses act alongside the immune system to produce behavioral deficits. As a clinically available drug, the sex-specific benefits and constraints of metyrapone should be investigated further as a potential means of reducing neurodevelopmental risks due to gestational MIA.

B-19. “Estrogenic re-mapping of the metabolic response to an energy deficit in female mice”

Paul B. Vander¹, Jae W. Park¹, Alexandra V. Ahlschlager¹, Fangmiao Sun², Weizhe Hong²,
Stephanie M. Correa¹

¹ Department of Integrative Biology & Physiology, University of California, Los Angeles, ² Department of Biological Chemistry, David Geffen School of Medicine, University of California, Los Angeles

In response to an energy deficit, mice can rapidly conserve energy by entering a hypometabolic state known as torpor. We have found that circulating 17 β -estradiol (E2), a potent estrogen, strongly inhibits fasting-induced torpor use in female mice – keeping energy expenditure high even during a dangerous energy deficit. The signaling mechanisms underlying this phenomenon have not been studied, however. Estrogen-sensitive neurons in the medial preoptic area of the hypothalamus (MPO) have been identified as central regulators of torpor in mice – suggesting that circulating estrogens may influence torpor usage by acting directly on this brain area. To test this hypothesis, we delivered E2 directly to the MPO and found that E2 treatment was sufficient to inhibit fasting-induced torpor in gonad-intact female mice, but, surprisingly, not ovariectomized females. We then knocked out estrogen receptor alpha (ER α) in the MPO and found that this manipulation did not block the effect of circulating estrogens on fasting-induced torpor usage. Together, these data demonstrate that

estrogen signaling in the MPO is neither sufficient to fully recapitulate the effects of circulating estrogens on torpor nor is it required to mediate them. However, we have shown that the MPO can tune torpor usage in response to local estrogen in certain contexts, suggesting that the MPO is integrated into the regulatory circuit, even if its influence is subtle and context-dependent. In conclusion, these findings advance our understanding of how estrogens influence torpor in mice – a critical step towards the larger goal of understanding how estrogens influence energy balance across species.

B-20. “Vasopressin deficiency enhances central GLP-1-mediated suppression of fluid intake in the Brattleboro rat”

Sydney A. David¹, Destiny J. Brakey², Matthew J. Paul¹, and Derek Daniels¹⁻³

¹ Department of Psychology¹, Department of Biological Sciences. ² Center for Ingestive Behavior Research. ³ State University of New York at Buffalo, Buffalo, NY USA.

Physiologically and behaviorally, fluid and food intakes are entangled. There is overlap in the brain regions that control the two types of ingestive behavior, and signaling peptides that affect one often affect the other. For example, glucagon-like peptide-1 (GLP-1) suppresses both food intake and fluid intake. The vasopressin-deficient Brattleboro rat has emerged as a potential model organism to separate fluid intake from food intake and could help isolate brain regions particularly involved in fluid intake control. Brattleboro rats drink copious amounts of water, but eat a similar amount of food when compared to wildtype rats. Brattleboro rats are hypersensitive to the fluid intake suppression caused by central administration of a GLP-1 receptor (GLP-1R) agonist, exendin-4 (Ex4), with no differences in sensitivity to the food intake effects. To evaluate if the hypersensitivity is directly related to the untreated vasopressin deficiency in these rats, we implanted osmotic mini pumps containing desmopressin (ddAVP, a vasopressin type 2 receptor agonist) and tested for fluid intake suppression by central administration of Ex4. We found that ddAVP reduced, but did not completely prevent, the hypersensitivity to Ex4, suggesting both correctable and uncorrectable changes underlying the response to Ex4. We also found that acute and chronic treatment with tolvaptan (a vasopressin type 2 receptor antagonist) in wildtype rats does not produce the hypersensitivity to Ex4 observed in Brattleboro rats. Together with previous work, these data provide useful context that will help identify the underlying cause of the different responses to Ex4.

B-21. “The prelimbic cocaine self-administration acquisition engram mediates well-trained cocaine seeking in male and female rats”

Bo W. Sortman^{1,2}, Brandon L. Warren¹

¹ University of Florida Department of Pharmacodynamics, ² University at Buffalo Department of Biology

The prelimbic cortex (PL) mediates cocaine seeking in reinstatement models post-extinction. Additionally, engram work has shown that after acquisition of cocaine self-administration the PL mediates cocaine seeking. Here we sought to investigate if the engram that mediates acquisition of cocaine self-administration continues to mediate well-trained cocaine seeking. We used a dual viral approach in rats that targets neuronal ensembles. Ensembles are constitutively activated neurons wired together after repeated cue-outcome associations identified by recent activity via Fos production. The Fos targeted recombination in active populations (Fos-TRAP) approach uses Fos

dependent CreERT2 expression in conjunction with a Cre-dependent tool. Here we injected AAV-FosCreERT2 with AAV-DIO-hM4Di-mCherry to tag neurons using 4-hydroxy-tamoxifen, one day after cocaine self-administration acquisition either in a novel context to tag a random subset of neurons, or, in the self-administration context, to tag the cocaine seeking engram. Rats are trained for 10 additional days to become well-trained and undergo a 30-min non-reinforced cocaine seeking test. On test day we injected either vehicle or the DREADD agonist compound 21 (C21). Rats that had the cocaine seeking engram tagged and received C21 had significantly attenuated cocaine seeking behavior compared to their vehicle counterparts, as well as rats that received C21 but had a random subset of neurons tagged in the novel context. Our results indicate that inhibiting activity in a random subset of PL neurons does not affect cocaine seeking in general. Additionally, the results of this study suggest that the engram formed after behavioral acquisition is maintained once rats are well-trained.

B-22. “Seasonal regulation of adolescent neural development: vasopressin innervation of the lateral septum”

Thalia J. Lopez¹, Jillian Woodhams², Zoey Forrester-Frontsin², and Matthew J. Paul²

¹ Department of Biological Sciences, University at Buffalo SUNY, Buffalo, NY, USA. ² Department of Psychology, University at Buffalo SUNY, Buffalo, NY, USA

Adolescence is a time of marked neural and behavioral development. Many changes are thought to be regulated by pubertal increases in gonadal steroids. One primary candidate is the vasopressin (VP) pathway that innervates the lateral septum (LS). This pathway develops postnatally, is sexually dimorphic (males > females), and is dependent on the presence of gonadal steroids. However, developmental increases begin, and sex differences emerge, prior to puberty, raising the possibility of contributions from puberty-independent mechanisms. Siberian hamsters are a useful model for studying the role of puberty in adolescent development. In this species, pubertal timing is regulated by seasonal changes in daylength: long daylengths (LDs) accelerate, whereas short daylengths (SDs) delay puberty. The present experiment tested the hypothesis that SD-induced delays in puberty would be accompanied by delayed development of VP innervation of the LS. Male and female hamsters were gestated and reared in either a LD or SD and development of LS VP innervation was assessed by immunohistochemistry. Paired testes and uterine weights were recorded to confirm photoperiod regulation of puberty. As reported for other species, LS VP fiber density was greater in male hamsters compared to female hamsters. SD-rearing suppressed LS VP innervation in both sexes. In males, this suppression persisted through 285 days of age, the latest time point assessed. These findings are consistent with the hypothesis that increases in pubertal steroids direct the development of VP innervation of the LS. More broadly, these findings demonstrate

Day 2: Poster Session

C-1. “Exploring C-Fos Expression Following Precipitated Glucocorticoid Withdrawal in Male Mice”

Savannah R. Pointe¹, Christen N. Snyder^{2,3}, Shany E. Yang^{2,3}, Joanna Spencer-Segal, M.D., Ph.D.^{3,4}

¹ NURO Program, Neuroscience Graduate Program, University of Michigan, Ann Arbor, MI. ² Neuroscience Graduate Program, University of Michigan, Ann Arbor, MI. ³ Michigan Neuroscience Institute, University of Michigan, Ann Arbor, MI. ⁴ Division of Metabolism, Endocrinology, and Diabetes, Department of Internal Medicine, University of Michigan Medical School, Ann Arbor, MI

Glucocorticoid withdrawal syndrome (GWS) develops after immediate cessation of prolonged exposure to endogenous or exogenous glucocorticoids. The symptoms, including fatigue, muscle and joint pain, nausea, and/or anorexia, contribute to a decreased quality of life. Currently, no effective treatment exists due to a limited understanding of its mechanisms. To explore the neural mechanisms of GWS, we developed a mouse model by administering twice daily injections of the glucocorticoid, dexamethasone, for ten days followed by a single dose of the glucocorticoid antagonist, mifepristone (5, 20, or 50 mg/kg), to precipitate withdrawal. During withdrawal, mice were tested for mechanical pain sensitivity in the von Frey test; findings suggested that mifepristone at 5 and 20 mg/kg doses increased mechanical pain sensitivity. To investigate neural activity changes during precipitated glucocorticoid withdrawal, immunohistochemistry using 3,3'-diaminobenzidine labeling for c-fos, a marker of neural activity, was performed on brain sections, and c-fos+ cell density was quantified in the motor and somatosensory cortices, paraventricular thalamus, hippocampal CA1 and dentate gyrus, basolateral amygdala, and piriform cortex. Mifepristone had a significant effect on the density of c-fos+ cells in the basolateral amygdala ($F(3,15)=4.559$, $p=0.0184$), motor cortex ($F(3,15)=5.680$, $p=0.0084$) and somatosensory cortex ($F(3,15)=3.447$, $p=0.0438$). Mice that received an injection of mifepristone 20 mg/kg showed decreased c-fos+ cell density in these areas compared to controls. Further research will look at additional brain regions and whole-brain analysis. Determining which areas of the brain are affected by glucocorticoid withdrawal will provide researchers with future therapeutic targets to treat GWS and improve patient quality of life.

C-2. “Marked sex differences are observed in heroin acquisition and affective state in rats, but converge to similar levels of footshock stress-induced reinstatement”

Claire Deckers¹, Chen Li¹, Zeinab Ahmed¹, Lynn Kirby¹

¹ Center for Substance Abuse Research, Department of Neural Sciences, Lewis Katz School of Medicine at Temple University, Philadelphia, PA, 19140, USA

Negative affective states, particularly those driven by stress, are known to trigger the development of opioid addiction and relapse. Previous preclinical research has identified sex differences in vulnerability to stress and resulting compulsion to consume drugs. Twenty-two- and 50-kHz ultrasonic vocalizations (USVs), reflecting negative and positive affective state, respectively, were recorded during a heroin self-administration and footshock stress-induced reinstatement model in Sprague-Dawley rats. We hypothesized that females would display increased vulnerability towards heroin use disorder-like profiles. Females show elevated acquisition of heroin self-administration

compared to males, but these sex differences are not associated with gonadal hormone levels. During heroin acquisition, males emit significantly more 50 kHz calls compared to females, reflective of clinical data demonstrating that drug consumption in males is driven by positive reinforcement. Despite marked sex differences in heroin self-administration, similar levels of stress-induced reinstatement of heroin seeking are observed in both sexes. Successful stress-induced reinstatement in both sexes is accompanied by a distinct pattern of USVs, whereby a high number of anticipatory 50 kHz calls are emitted prior to the onset of reinstatement sessions, followed by 22 kHz call production during reinstatement sessions, perhaps reflecting drug seeking to relieve negative affect or negative affective responses to drug unavailability. These findings indicate sex-specific vulnerabilities to the development of heroin use disorders. Future studies will focus on exploration of potential underlying mechanisms and the involvement of the serotonergic dorsal raphe nucleus, a sexually dimorphic system that contributes to affective components of drug addiction that drive drug consumption and relapse.

C-3. “Does Neuropeptide Y Modulate Responses to Social Stimuli in a State-Dependent Manner in a Monogamous Cichlid Fish, *Amatitlania nigrofasciata*?”

Sarah A. Muh¹, Camille P. Brown¹, Ross S. DeAngelis^{1,2}, Hans A. Hofmann^{1,2}

¹ Department of Integrative Biology, University of Texas at Austin, ² Institute for Neuroscience, University of Texas at Austin

How the brain regulates behavioral responses to salient situations depends on an individual's behavioral state. For example, in pair-bonding species, unbonded individuals readily approach unfamiliar opposite sex individuals, yet once a pair bond is established, they may avoid or even attack them. Similarly, pair-bonded individuals may respond to offspring cues only after they have reproduced. While the role of both neuropeptides and dopamine in regulating the formation of these enduring bonds has been studied in detail, our understanding of how other peptide systems mediate the responses to aggressive or affiliative stimuli in a state-dependent manner is very limited. Here, we use the monogamous biparental Convict Cichlid, *Amatitlania nigrofasciata*, to investigate how Neuropeptide Y (NPY) may regulate responses to social stimuli in bonded or parental convict females. Our results show that pair-bonded males and females coordinate their responses to social challenges, with context-dependent sex differences. Through immunofluorescent labeling of the neural activity marker phospho-S6 ribosomal protein (pS6) and NPY, we demonstrate that differentially motivated aggressive and affiliative responses are mediated by brain region-specific activity patterns of this neurohormonal system. These results show how information about the social world is represented in the brain across hierarchically organized levels to generate behavioral state-dependent context-appropriate behavior.

C-4. “A single-cell atlas of chronic stress- and ketamine-induced transcription across the mouse brain”

Devin Rocks¹, Grace Paquelet¹, Conor Liston¹

¹ Department of Psychiatry, Weill Cornell Medicine, New York, NY 10065, USA.

Depression is an episodic psychiatric disorder often characterized by recurring mood-state transitions between depression and euthymia. The neural and molecular mechanisms of factors associated with

induction and remission of depressive episodes, such as chronic stress and antidepressant treatment, respectively, are poorly understood. To address this, we administered chronic corticosterone (Cort, 0.1mg/ml in 1% ethanol) or vehicle (Veh, 1% ethanol) drinking water to mice for 3 weeks to model chronic stress conditions. All Veh mice were given an acute (3h) saline injection (i.p.), while Cort mice were divided into three groups: saline (i.p. 3h), ketamine-acute (10mg/kg i.p. 3h), and ketamine-extended (10mg/kg i.p. 48h). We found that this dose and duration of corticosterone treatment reduced motivated escape behavior in both males and females in the tail suspension test, and this behavioral effect is rescued by ketamine treatment. Using split-pool combinatorial barcoding, we then performed single-nucleus RNA-sequencing (snRNA-seq) on mice from these four groups (n=6/group, 3 male and 3 female) across four stress-sensitive and antidepressant-responsive brain regions: medial prefrontal cortex, anterior insula, ventral hippocampus, and basolateral amygdala, generating a total of 96 snRNA-seq samples. Analysis of this comprehensive dataset will yield crucial insights into the cell type- and sex-specific transcriptional effects of chronic stress and ketamine across multiple brain regions, enabling us to test hypotheses about the neural mechanisms mediating the behavioral effects we observed. By improving our understanding of the mechanisms underlying mood-state transitions, this work will enable the generation of more effective treatment protocols for depression.

C-5. “Effects of Environmental Enrichment on Neonate Ultrasonic Vocalizations and Sex-Biased Maternal Response in House Mice (*Mus musculus*)”

Lucas F. Fowler^{1,2}, T. Nadine Burry^{1,3}, Meagan Hinks¹, Alison M. Randell⁴, Kerri M. Sparkes¹, Stephanie Salia¹, Jessica Vaters¹, & Ashlyn Swift-Gallant¹

¹ Department of Psychology, Memorial University of Newfoundland and Labrador, ² Cognitive & Behavioural Ecology Program, Memorial University of Newfoundland and Labrador, ³ Department of Chemistry, Memorial University of Newfoundland and Labrador, ⁴ Department of Psychology, Brock University

Diverse taxa, including whales, frogs, and rodents, use high-frequency (i.e., >20kHz) ultrasonic vocalizations (USVs) to communicate. In neonatal rodents, USVs elicit maternal retrieval and are thought to ensure survival. Pup sex mediates USVs and maternal care, with males typically making more USVs with higher durations and lower frequencies and receiving more attention than females. However, stressful conditions can alter USVs and maternal responses in a sex-dependent manner. To assess whether sex and environment interact to modulate neonate USVs and subsequent maternal response, we assigned female mice to standard (ST) or environmentally enriched (EE) housing and bred them with males. Resulting litters were culled to two pups of each sex. In a modified three-chamber apparatus, maternal response and pup USVs were recorded. Maternal response, defined as time spent interacting with pups, and pup USV parameters, including call number, duration, and frequency, were assessed. Preliminary results show that housing affects maternal response, with ST dams spending more time with pups than EE dams. Yet, EE pups made more and longer calls at lower frequencies than ST pups, but these measures did not correlate with maternal response. EE dams also preferred male pups over female pups, an effect absent in ST counterparts. Dam serum will be assessed for corticosterone, estradiol, and progesterone to delineate potential endocrine mechanisms driving these differences in maternal response, and future work will evaluate call types

(e.g., short, flat, chevron, upward, downward, etc.) to further understand the role of sex and environment in neonate rodent communication.

C-6. “The Effect of Histamine on Water Intake and Locomotor Activity in Female Rats”

Emalie Mullane¹, Lasni Nishshanke¹, and Jessica Santollo¹

¹ University of Kentucky Biology Department

The hormone estradiol controls water intake in a bidirectional manner. When food is available estradiol decreases water intake in ovariectomized (OVX) rats, but when food is unavailable estradiol increases water intake. The mechanism, however, by which estradiol influences water intake is unknown. The neuromodulator histamine increases water intake and estradiol increases histamine signaling. Our long-term goal is to determine if estradiol enhances the dipsogenic effect of histamine. The goal of this project is to determine the behavioral specificity of histamine treatment. Specifically, we tested the hypothesis that central treatment of histamine and a histamine antagonist at doses that influence water intake do not affect locomotor activity. This is an important consideration, as changes in motor function can influence ingestive behavior. Sixteen female rats were ovariectomized and a cannula was surgically placed into the lateral ventricle of the brain. After recovery, water intake and locomotor activity were determined by observing the effect of histamine and the histamine antagonist pyrilamine maleate. Treatment with 5 and 10 µg of histamine dichloride increased light phase water intake ($p < 0.05$). Treatment with 300 µg pyrilamine maleate decreased water intake in response to 24 h water deprivation ($p < 0.05$). Importantly, drug doses that influenced water intake had no effect on locomotor activity, which was measured by lines crossed in a one-hour open field test. We next plan to determine if estradiol enhances the dipsogenic effect of histamine in OVX female rats.

C-7. “TARGETING HIPPOCAMPAL REGULATION OF DOPAMINE SYSTEM FUNCTION: A NOVEL THERAPEUTIC APPROACH IN THE VCD RAT MODEL OF PERIMENOPAUSE”

Kayla Lilly¹, Angela Boley¹, Daniel Lodge¹, Stephanie Perez¹

¹ University of Texas Health at San Antonio Pharmacology Department

Perimenopause, the transitional period leading to menopause, is a period of increased risk for developing a psychiatric disorder or experiencing exacerbated symptoms of a pre-existing disorder. Psychiatric symptoms are debilitating and significantly diminish the quality of life for women and their families. Thus, understanding the neurocircuitry contributing to these conditions is critical for the discovery of novel therapeutics. Of particular interest is the dopamine system, which has been highly implicated in the regulation of mood, addiction, psychosis, and cognitive function. Here we demonstrate aberrant dopamine system function in the 4-vinylcyclohexene diepoxide (VCD) model of perimenopause. This model has translational relevance, as daily administration of VCD induces a progressive loss of ovarian follicles and accompanying hormonal changes that closely resemble the hallmarks of perimenopause occurring in women. Further, the aberrant dopamine system function in this model appears secondary to hippocampal hyperactivity. We posit that MP-III-022, a positive allosteric modulator selective for $\alpha 5$ -GABAA receptors, will decrease hippocampal hyperactivity and normalize downstream dopamine signaling, leading to a reversal of deficits in related behaviors. Indeed, aberrant dopamine system function in VCD rats was normalized by the systemic administration of MP-III-022. VCD rats also displayed deficits in cognition, as measured by the novel

object recognition task, which was also reversed by MP-III-022. Taken together, these data suggest that targeting hippocampal regulation of dopamine signaling may be a potential novel therapeutic for the treatment of psychiatric alterations during perimenopause.

C-8. N/A

C-9. “Exploring the Effects of Tibolone and IGF-I on Astrocyte Reactivity and Autophagy on Males and Females mice: A Comprehensive In Vitro Investigation”

Manuela Faddetta¹, X. Ganchala², M.A. Arevalo³

¹ University of Limerick, Faculty of Science and Engineering. ² Cajal Institute, CSIC, Functional and Systems Neurobiology Department. ³ Cajal Institute, CSIC, Functional and Systems Neurobiology Department

Astrocytes are prevalent cells within the central nervous system (CNS) that provide critical support to neurons and are actively involved in the formation and pruning of synapses. Under pathological conditions, such as disease or brain injury, astrocytes can become reactive, resulting in the formation of two distinct subtypes: A1 and A2. A1 astrocytes, lose their capacity to support neuronal survival and synaptogenesis, promoting the death of neurons and oligodendrocytes. Conversely, A2 astrocytes facilitate CNS repair and recovery by upregulating neurotrophic factors. The conversion of A1 astrocytes to A2 could present a novel approach for targeting cognitive decline in aging-related diseases. Previous research indicated that lipopolysaccharide (LPS) stimulation enhances the phagocytosis of cellular debris in male astrocyte cultures, while inhibiting this process in female cells. These LPS-induced effects are differentially modulated by IGF-I and Tibolone, depending on sex. This study aims to elucidate the sex-specific differences in astrocyte autophagy induced by LPS, investigating also the effects of IGF-I and Tibolone. Tibolone, frequently utilized in the clinical management of menopause symptoms, generates metabolites that cross the blood-brain barrier, resulting in increased concentrations within the brain. Additionally, Tibolone raises serum IGF-I levels in women and IGF-IR levels in rats. This study seeks to determine whether IGF-I contributes to Tibolone actions. Findings indicate that IGF-I exhibits sex-dependent regulation of autophagy-related gene and protein expression, whereas Tibolone broadly enhances autophagy levels. However, the interaction between IGF-I and Tibolone remains inconclusive, underscoring the need for further research to delineate their combined effects on inflammation, and autophagy.

C-10. “Serotonin Disruption Leads to Memory Deficits in the Whip Spider *Phrynos Marginemaculatus*”

Sidney Ley¹, Nicholas Brown², Daniel Wiegmann¹, Verner Bingman²

¹ Department of Biological Sciences, Bowling Green State University, Bowling Green, OH, USA. ² Department of Psychology, Bowling Green State University, Bowling Green, OH, USA

Amblypygids, or whip spiders, are unusual arachnids that navigate primarily by utilizing their unusually keen olfactory abilities. Previous research has shown that the whip spider (*Phrynos marginemaculatus*) can undergo associative olfactory learning, as well as form short and long-term memories based on this learning. Still, little is known regarding the underlying neurophysiology that controls these processes. Comparative research indicates that the whip spider's olfactory memory may be dependent upon serotonin signaling in the mushroom bodies, brain structures involved in learning

and memory in invertebrates. While serotonin function within the whip spider remains completely unexplored, it can be hypothesized, then, that if serotonin activity is present within the whip spider and then pharmacologically disrupted using an anti-serotonergic drug, the whip spider's performance in an olfactory navigation assay would be significantly impaired in comparison to controls treated with saline. To this end, subjects were trained in an olfactory memory paradigm, injected with either 0.9% physiological saline or the serotonin antagonist methiothepin mesylate, and tested at 24, 48, 72, and 96 hours, and 21 days afterward for memory retention. There were significant differences in the performance between the groups at 24 hours ($p = 0.005$) and the controls performed above chance on all but the 96-hour timepoint, while the treated group consistently performed at chance in all timepoints. Additionally, there were no differences in locomotion between the groups found at any timepoint. Taken together, these preliminary data indicate that MET impairs memory consolidation in the whip spider by impairing serotonin signaling.

C-11. "The basolateral amygdala differentially encodes social and nonsocial reward in male mice"

Jarildy L. Javier¹, Jennifer Isaac¹, Hymavathy Balasubramanian¹, Malavika Murugan¹

¹ Dept of Biology, Emory University, Atlanta, GA

As animals navigate their environment, they must make decisions on whether to engage with different reinforcing stimuli. While several studies have explored the neural substrates that underlie decisions between positive and negative stimuli, less research has focused on how different brain regions encode choice and reward when given two different, appetitive stimuli. Recently, we explored how the mPFC encoded social and nonsocial reward using a novel two choice operant assay (Isaac et al, 2023). This study revealed largely non-overlapping representations of sucrose and social reward that were modulated by internal state and sex. However, it's unclear if social and nonsocial reward representations remain largely distinct in other nodes of the reward circuitry, specifically the basolateral amygdala (BLA). To address this question, I focused on the role of the BLA, a region known to encode positive and negative valence, in encoding social and nonsocial reward-related behaviors. Using in vivo cellular resolution calcium imaging while mice performed an operant assay in which they can choose between food and social rewards, I found that the BLA differentially encodes social and nonsocial reward choice and consumption ($n=7$ adult male mice). Additionally, the BLA representations are distinct from those found in the mPFC. The unique representations in these two connected nodes of the social reward circuit suggests a possible difference in the information encoded in either region. This adds valuable insight into how the brain may differentially weigh competing reward options in one setting and support decisions to interact with one over another.

C-12. "Gonadal Steroid Hormones Influence Perineuronal Net Development in an Age-dependent Manner in the Medial Prefrontal Cortex of C57 Mice"

Jessica Tremblay¹, Marcia Chavez¹, Kalynn Schulz¹

¹ University of Tennessee Knoxville, Department of Psychology

Adolescence is a key developmental period in which brain and behavioral changes coincide with pubertal onset and the secretion of gonadal steroid hormones. The brain's ability to reorganize and respond to stimuli, also known as neuroplasticity, is regulated by perineuronal nets (PNNs). PNN

density increases across adolescence in the medial prefrontal cortex (mPFC); however, it remains unclear whether secretions of gonadal steroid hormones during puberty influence PNN development in the mPFC. The current study investigates if PNN density is regulated by hormones, age, or an interaction between these factors. To distinguish between these possibilities, male and female C57 mice were gonadectomized prior to the onset of puberty. After gonadectomy, males were administered testosterone and females were administered estradiol either prepubertally (P23), during adolescence (P40), or in adulthood (P63). Brains were collected after one week of steroidal hormone treatment. The A32 (prelimbic) and A25 (infralimbic) subregions of the mPFC were imaged on an immunofluorescent Keyence microscope, and the number of PNNs were counted. In the A32 subregion, both male and female hormone-treated animals displayed an increase in PNN density only during adolescence and adulthood. These data suggest that gonadal steroid hormones regulate PNN development in a time-dependent manner. In contrast, no effects of hormone or age were observed in the A25 subregion. These data may have implications for the increased mental health risks associated with early or late pubertal timing in humans.

C-13. “Valproic Acid Alters Sociosexual Signaling in Male Naked Mole-Rats”

Sama Al-Shammari^{1,2}, Madison Mickalakos^{2,3}, Joaquin San Agustin³, Melissa M. Holmes³⁻⁵

¹ Department of Forensic Science, University of Toronto Mississauga, ² Department of Biology, University of Toronto Mississauga, ³ Department of Psychology, University of Toronto Mississauga, ⁴ Department of Cell & Systems Biology, University of Toronto, ⁵ Department of Ecology and Evolutionary Biology, University of Toronto

Naked mole-rats are eusocial rodents that live in large colonies with strict social and reproductive hierarchies. Valproic acid (VPA) is a histone deacetylase inhibitor that alters social behavior in various species, including increasing social defeat responses in hamsters. Faykoo-Martinez et al. (2021) discovered that male naked mole-rats injected with VPA twice daily for seven days did not display any behavioural changes, whereas the female conspecifics they were paired with exhibited increased genital sniffing. These changes were linked to increased H3K18 and H3K27 acetylation in VPA-treated mole-rat livers. Here, we conducted a replication experiment to confirm the previous findings and to investigate the role of pheromones primarily found in male urine, called major urinary proteins (MUPs), in modulating the sociosexual behaviour of the females paired with VPA-treated males. While epigenetic analysis to investigate histone acetylation and mass spectrometry for urine analysis has not yet been conducted, the results of the replication experiment confirm that while VPA treatment did not alter the behaviour of males, females investigated VPA-treated males more than they did saline-treated males. Consistent with prior findings, this was specific to genital investigation as there was no significant difference in face and body sniffing by stimulus female mole-rats. Based on epigenetic, urine and preference test results, various potential implications, such as the role of MUP concentrations in male breeder selection, arise. Factors like individual differences in MUP and urinary testosterone concentration, as well as social rank, must also be considered.

C-14. “Effects of bacterial metabolites on neuronal cell death and microglia in neuroendocrine regions of the neonatal mouse hypothalamus”

Gabby West¹, Alexandra Castillo-Ruiz², Nancy G. Forger²

¹ Department of Biology, Georgia State University, Atlanta, GA 30302. ² Neuroscience Institute, Georgia State University, Atlanta, GA 30302

The microbiota consists of various microbial groups that inhabit our bodies' surfaces, with gut bacteria being the most populous. This microbiota communicates with the brain via several routes, including bacterial metabolites. We previously reported that the microbiota influences key neurodevelopmental events in neonatal mice, including neuronal cell death and microglia colonization in the hypothalamic paraventricular nucleus (PVN), a critical neuroendocrine region that coordinates the stress response. To probe for mechanism, here we tested whether bacterial metabolites are responsible (specifically short-chain fatty acids (SCFAs)). We also analyzed neighboring hypothalamic regions that regulate important metabolic functions (e.g., food intake, thermoregulation): the ventromedial (VMH) and dorsomedial (DMH) nuclei. Timed-pregnant mice received a SCFA cocktail: sodium acetate, sodium propionate, and sodium butyrate in their drinking water, while control dams received sodium-matched or untreated water. Treatments began on embryonic day 14, and offspring were euthanized on postnatal day 0 or 3. Brains were then processed by immunohistochemistry for quantification of cells displaying markers for cell death and microglia. SCFA supplementation reduced cell death in the PVN and VMH at P0. Interestingly, the extent of the effect in the VMH depended on the control group used for comparisons, with the largest effect seen when the SCFA group was compared to the water group. For microglia, we found no significant differences between groups for any brain region. Our results suggest that bacterial metabolites are crucial neurodevelopmental signaling molecules in regions with important neuroendocrine functions; they also highlight the importance of appropriate controls in SCFA studies.

C-15. “Maternal immune activation with lipopolysaccharide accelerates neonatal reflex acquisition and alters basal gene expression across early postnatal development in rats”

Mary Beth Hall^{1,2}, Elise Lemanski^{1,2}, Jaclyn M. Schwarz^{1,2}

¹ University of Delaware, Department of Psychological and Brain Sciences; ² University of Delaware, Interdisciplinary Neuroscience Program

Epidemiological evidence suggests that maternal infection during gestation increases risk for offspring to experience symptoms of neurodevelopmental disorders, of which males are more likely than females to be diagnosed. The goal of this project is to elucidate how maternal immune activation (MIA) with lipopolysaccharide (LPS) impacts brain and behavioral processes in male and female offspring. Sprague-Dawley dams were injected with LPS (50µg/kg, i.p.) or saline on embryonic day (E)15. From postnatal day (P)3-P21, the acquisition of neonatal reflex behaviors was observed in male and female offspring. mRNA expression of inflammatory and neurotrophic genes was also measured at P7 and P21. Results showed that E15 LPS offspring were able to forelimb grasp, cliff avoid, and right with a correct posture at an earlier age than E15 saline offspring. These results may be attributable to the stress acceleration hypothesis, whereby early-life adversity, such as MIA, may shift development to occur on a faster timeline. Moreover, MIA upregulated IL-1 β expression in medial prefrontal cortex (mPFC) of P7 male and female offspring and downregulated BDNF expression in dorsal hippocampus of P21 males only. These results are characteristic of human NDDs. However, MIA also downregulated IL-6 expression in mPFC of P21 male and female offspring. Together, these results suggest that MIA differentially affects offspring basal cytokine expression in an age-dependent manner. Overall, these findings provide us with a better understanding of how prenatal immune

activation affects early-life brain and behavioral processes in offspring, and how they may be differently dysregulated in males and females.

C-16. “Sexual Experience Modulates Morphine Sensitivity and Reward in Female Long-Evans Rats”

Hanni Zhu¹, Meg Frost¹, Colin James¹, Kathryn Kresse¹, Alexei Mendez¹, Sarah H. Meerts¹

¹ Carleton College Neuroscience Program

Mating behavior in female rats is a rewarding process that can be enhanced through experience. Sexually experienced female rats, compared to their naive counterparts, show shorter latencies to return to the male after receiving stimulations, spend more time with the male, and display increased proceptive behaviors during mating. Sexually experienced, but not naive, rats exhibit a conditioned preference for a single mating encounter. Because blocking opioid receptors prevents mating-induced reward in female rats, we investigated whether the enhancement of sexual performance following repeated mating experience is linked to differences in sensitivity to opioid reward. Ovariectomized female rats were treated with estradiol benzoate 48 hours, and progesterone 4-6 hours prior to testing to induce receptivity. Rats received either 4 paced mating tests (Experienced) or 4 exposures to an empty pacing chamber (Naive). All rats received saline before the CS- session. Rats were assigned to one of 4 morphine treatments within the Naive and Experienced conditions: saline, 0.5, 1, or 2 mg/kg morphine administered subcutaneously immediately before the CS+ session. A paired sample t-test revealed that naive female rats exhibited a conditioned preference for the CS+ after all doses of morphine, whereas experienced female rats exhibited a conditioned preference for the CS+ only after receiving 2 mg/kg morphine. This study suggests that sexual experience in female rats may reduce their sensitivity to morphine, potentially by modulating opioid receptors.

C-17. “Does knocking out the V1a2 vasopressin receptor impair dominance behavior in a highly social cichlid fish?”

Landon B. Porter¹, Cheng-Yu Li², Scott A. Juntti², Hans A. Hofmann^{1,3}

¹ Department of Integrative Biology, The University of Texas at Austin. ² Department of Biology, University of Maryland College Park. ³ Institute for Neuroscience, The University of Texas at Austin.

Animals of most species successfully navigate dynamic social environments to ensure survival and maximize their fitness. Context-appropriate decision-making depends on the successful integration of social stimuli with internal information about an individual's condition, its prior experience, and its current behavioral state. The neuromolecular mechanisms underlying these processes have been extensively investigated across vertebrate taxa, revealing that the nonapeptide arginine vasopressin (AVP) and its receptors regulate diverse social behaviors, including aggression and affiliation; however, a general framework delineating AVP's role in social decision-making across vertebrates remains elusive. In teleost fish, the V1a2 receptor subtype is widely expressed throughout the evolutionarily conserved Social Decision-Making Network (SDMN), suggesting a central role of this pathway in regulating social behavior. In fact, AVP V1a2 receptor subtype has been implicated in converting socially salient experiences into long-lasting changes in brain function and behavior. Burton's Mouthbrooder cichlid fish, *Astatotilapia burtoni*, naturally organize into dominance hierarchies with socially dominant (DOM) and socially subordinate (SUB) males. AVP is differentially regulated in

parvo- and gigantocellular preoptic neurons of DOMs and SUBs of this species and has been suggested to facilitate social ascent in SUB males. Here, we used immunohistochemistry to test the hypothesis that V1a2 expression throughout the SDMN is dependent on social status. We next employed CRISPR-Cas9 mediated knock-out of the V1a2 gene to test the hypothesis that V1a2 mediates social dominance behavior. By examining the role of specific receptors within these ancient nonapeptide systems, this work increases our understanding of how social behavior is modulated within the brain.

C-18. “Effects of constitutive CX3CR1-Cre expression on microglia and mouse behavior”

Jonila Shehu¹, Fadya Mroue-Ruiz¹, Bhoomi Desai¹, Madison Garvin¹, Faith Kamau¹, Urjoshi Kar¹, and Jessica Bolton¹

¹ Georgia State University, Neuroscience Institute

The Cre-loxP system is a widely used gene editing tool. Recently, Sahasrabuddhe et al. (2022) described the adverse effects of inducible CX3CR1-Cre expression on microglia in the early postnatal period. These included lower microglia density, a reactive phenotype, increased phagocytic function, and DNA damage, as well as an increase in anxiety-like behavior in adulthood. However, the effects of constitutive expression of CX3CR1-Cre on microglia remain unknown. We hypothesized that constitutive CX3CR1-Cre expression would also lead to similar adverse effects. To test this, we immunolabelled microglia in the parietal cortex of CX3CR1-Cre^{+/+} and -Cre^{-/-} littermates during the early postnatal period (P8 ± 1 day) and adulthood (P60-90). We analyzed microglial density and volume using ImageJ and Imaris software, respectively. We examined microglial proliferation and DNA damage by looking at the density of Ki67 and γ H2AX markers in colocalization with microglia. Additionally, we performed the looming-shadow threat task, elevated-plus maze, and open-field test to assess adult behavior. We found a significant decrease in Iba1+ microglia density and volume in Cre^{+/+} females at P8, although this difference disappeared by adulthood. Sholl analysis is ongoing to further define changes in microglial morphology caused by Cre expression. No significant differences were observed in the density of Ki67 and γ H2AX in any of the groups. Functionally, no differences were found in any of the behavioral outcomes. Overall, our findings suggest that constitutive CX3CR1-Cre expression may be less detrimental than inducible CX3CR1-Cre expression and highlight the need to perform correct control experiments when using these genetic tools.

C-19. “A REV-ERB agonist ameliorates neuroinflammation in a sex-specific manner in mice after spinal cord injury.”

Kalina J. Dusenbery^{1,2}, John C. Aldrich^{1,2}, Fiona C. Bremner^{1,2}, Alma Perez¹, Laura K Fonken³, Andrew D. Gaudet^{1,2}

¹ Department of Psychology, College of Liberal Arts, The University of Texas at Austin. ² Department of Neurology, Dell Medical School, The University of Texas at Austin. ³ Division of Pharmacology and Toxicology, College of Pharmacy, The University of Texas at Austin

Spinal cord injury (SCI) results in an immediate and persistent inflammatory state, leading to secondary damage and exacerbated chronic pathology. Neuroinflammation is driven by microglia and macrophages; these cells contain intrinsic circadian clocks that modulate their behavior and reactivity, making the circadian system a promising therapeutic target after SCI. We hypothesized that treating

mice with a pharmacologic agonist (SR9009) targeting REV-ERB –a key repressor of the molecular clock– would reduce pro-inflammatory gene expression after SCI. Mice received SCI (a moderate contusion at T9), then were treated with SR9009 or vehicle three hours post-injury and daily thereafter. Tissue was collected three hours after final drug delivery for analysis of injury-induced inflammatory mRNA profile. REV-ERB agonist administration after SCI led to a dose-dependent decrease in the expression of potent pro-inflammatory genes (e.g., Il1b and Tnf); this effect was driven by a robust decrease specifically in female mice. Indeed, when treated with SR9009, female mice had significantly lowered pro-inflammatory gene expression compared to agonist-treated males and vehicle-treated females. Overall, we find that pharmacologic enhancement of REV-ERB ameliorates the acute inflammatory response in female mice after SCI, suggesting it is a promising approach for reducing secondary damage and improving long-term neurologic recovery. Future studies should explore the molecular mechanisms underlying the observed sex difference to identify appropriate immunotherapies to combat secondary pathology after SCI.

C-20. “Long-Term Effects of Postnatal Clomipramine Exposure on Aminergic Systems in a Postpartum *Rattus norvegicus* Model of Obsessive-Compulsive Disorder”

Harika Kosaraju¹, Mia Bechara¹, Christina M. Ragan¹

¹ Molecular Mechanisms of Mothering and Anxiety (MoMMA) Lab, Georgia Institute of Technology

Obsessive-compulsive disorder (OCD), an anxiety disorder characterized by difficulty controlling thoughts and impulses, disproportionately affects postpartum women. Low central serotonin (5-HT) and dopamine (DA) levels are associated with OCD, as both neurotransmitters are important for mood stabilization and cognitive ability. Current treatment for OCD in adult humans includes clomipramine, a tricyclic antidepressant that targets serotonin and norepinephrine reuptake. However, male rat models of postnatal clomipramine administration demonstrate development of OCD-like behavior and increased central 5-HT and DA receptor expression in adulthood. OCD is also associated with decreased hippocampal volume in humans, indicative of impaired hippocampal function. Although OCD-like behavior and neurotransmitter levels due to clomipramine exposure have been analyzed in male rats, this model has not been examined in the hippocampus or in parous/nulliparous female rats. Based on previous studies, we hypothesized that administering clomipramine to female rats during their early postnatal period causes decreased adult postpartum 5-HT and DA levels in the hippocampus compared to controls. To test this hypothesis, 30 Sprague-Dawley (*Rattus norvegicus*) rats received intraperitoneal injections of either 0.9% saline (control) or 15 mg/kg clomipramine (experimental) twice daily during postnatal days 9-16. High-performance liquid chromatography was used to assess levels of 5-HT and DA in the hippocampus. Dams exposed to early-life clomipramine exhibited a 51.07% decrease in 5-HT and a 61.17% decrease in DA in their hippocampal regions compared to saline-exposed dams. These decreased adult amine levels suggest that drug exposure during a sensitive developmental period affects later postpartum aminergic systems.

C-21. “Correspondence Between Cortisol and Brain Activity During Social Motivation in Humans”

Alex Ogden¹, Cameron Hicks¹, Sally Farah¹, Dale Cohen², Kathleen Casto¹

¹ Kent State University Psychological Sciences, ² The University of North Carolina Wilmington Psychology

Task based cortisol response reflects the mobilization of resources necessary to meet the motivational demands of a stressor. Yet, there is limited evidence for the real-time coordination of endogenous cortisol change and patterns of neural activation associated with motivation. The purpose of this study is to provide initial evidence to establish this connection. In 33 human subjects, we assessed cortisol response and task performance in a computerized competitive persistence task, under three conditions of reward: 1) Monetary, 2) Positive Social Evaluation, and 3) Negative Social Evaluation. Using functional near infrared spectroscopy (fNIRS), we then tested cortical patterns of activation associated with performance motivation in each condition. Results showed that areas of the ventrolateral and orbitofrontal prefrontal cortex (PFC) were more active for those who showed higher task motivation. Exploratory analysis between reward conditions revealed that the highest activity was seen in the left ventromedial PFC for those in the negative social evaluation condition. Contrary to predictions, cortisol generally decreased during the task. Comparing neural activity to individual differences in cortisol area under the curve (AUC) across all trials, we explored candidate regions of interest for cortisol action in the PFC. This approach provides new evidence for the role of the PFC in competitive motivation, the effects of social evaluation, and the potential role of rapid cortisol response in facilitating (or recovering from) goal-directed motivation. These advancements will serve as the basis for testing endocrine-brain dysregulation in mood disorders and other conditions marked by motivational deficits.

C-22. “Adolescent Short Binge Ethanol Exposure and Drinking Duration Sex-Specifically After Ethanol Drinking”

Dashear Hill, B.S.¹, Ashud Kanu, B.S.², Chyanne Terry³, Antoniette M. Maldonado-Devincci, Ph.D.⁴

¹ Department of Family and Consumer Sciences, College of Agriculture and Environmental Sciences, North Carolina A&T State University. ² Department of Environmental Sciences, College of Agriculture and Environmental Sciences, North Carolina A&T State University. ³ Department of Psychology, College of Health and Human Sciences, North Carolina A&T State University

Alcohol exposure in adolescents disrupts behavior, emotions and cognitive developmental stages. Alcohol is a central nervous system depressant that can create dependency and adolescents tend to drink in a binge-like pattern. The objective of this set of studies was to determine if a short-binge exposure during adolescence and drinking duration alters sex differences in voluntary alcohol drinking patterns. The mice were exposed to 2 hours of ethanol or air vapor inhalation each day followed by 2 hour withdrawal in the drinking cage. Voluntary alcohol drinking sessions were conducted for 2 hours or 16 hours (overnight) with a choice between ethanol (12% v/v) and water over 5 consecutive days each week. This cycle was repeated five times. Drinking volumes were recorded each hour during the 2 hour session and the following morning for the 16 hour session to measure the preference and quantity of ethanol and water amongst male and female adolescent mice. After detailed analysis the influence of alcohol resulted in a pattern, female ethanol-exposed mice consumed higher amounts of ethanol compared to female air-exposed mice during 2hr voluntary drinking, whereas males did not show differences in drinking. During the 16 hour overnight-period, male ethanol-exposed mice showed increased ethanol drinking in later cycles compared to their air-exposed controls. Together, these data indicate that drinking duration may reveal nuances sex-specific differences in voluntary ethanol drinking following a short binge ethanol exposure.

D-1. “Chronic variable social stress exacerbates airway inflammation and glucocorticoid reactivity, but ameliorates anxiety-like behavior, glucocorticoid recovery, and prefrontal and hypothalamic MR and FKBP5 expression in a developmental BALB/cj female and male mouse model of allergic asthma”

Jasmine I. Caulfield, Ph.D.^{1,2}, Erin M. Cover², **Sanjana M. Velu**², Helen M. Kamens, Ph.D.², William J. Horton, Ph.D.², Sonia A. Cavigelli, Ph.D.^{1,2}

¹ Pennsylvania State University, Huck Institute for Life Sciences, 101 Life Sciences Building, University Park, PA 16802, USA. ² Pennsylvania State University, Department of Biobehavioral Health, 219 Biobehavioral Health Building, University Park, PA 16802, USA

Allergic asthma is the most common chronic health issue among youth in the U.S., occurring during a critical period of neurobiological development when stress can impact long-term mental health. Previous research indicates that youth with asthma are at higher risk for anxiety, similar to those facing social challenges. To investigate whether chronic social challenges during asthma development worsen immune function, anxiety, or stress physiology, we used a mouse model to study the effects of allergen-induced airway inflammation combined with social instability. From postnatal days 7-59, BALB/cJ mice were exposed to house dust mite extract (HDM) to induce airway inflammation and underwent repeated social isolation and reorganization (chronic variable social stress, CVSS) to simulate social instability. In adulthood, we assessed asthma-related cytokines and gene expression in lung tissue, as well as circulating corticosterone levels, anxiety-like behavior, and HPA axis-related gene expression in the prefrontal cortex, ventral hippocampus, and hypothalamus. Social instability increased HDM-induced cytokine levels and lung inflammation in females and heightened their corticosterone response to acute stress. However, it reduced HDM-induced anxiety-like behavior and affected the recovery speed of corticosterone and gene expression related to anxiety in the prefrontal cortex and hypothalamus in females. These findings confirm that chronic social stress exacerbates allergen-induced inflammation but does not increase anxiety symptoms; rather, it appears to buffer against allergen-induced anxiety and HPA dysregulation in females.

D-2. “Validating a Developmental Reprimo Knockout in the Female Mouse Hypothalamus via In-Situ Hybridization Techniques”

Rosalizbeth M. Martinez^{1,2}, Norma P. Sandoval^{1,2}, Elizabeth A. Dilday¹, Jae W. Park¹, Maria F. Cassol¹, J. Edward van Veen^{1,3}, & Stephanie M. Correa^{1,3}

¹ University of California, Los Angeles Department of Integrative Biology & Physiology. ² Co-first authors. ³ Co-senior authors

Previously, the Correa lab found that reprimo-expressing (Rprm) neurons within the ventromedial nucleus of the hypothalamus (VMH) are sexually dimorphic and estrogen-responsive. They discovered that transiently silencing Rprm with short interfering RNAs increases body temperature in female mice. We are now investigating the long-term impact on body temperature after permanently knocking out the Rprm gene in female mice. To study this, we developed mouse models using Flp-FRT recombination, which is specific to glutamatergic cells. I propose that Rprm KO should only occur in glutamatergic VMH neurons, but not the gabaergic cells in the arcuate nucleus (ARC). We used RNAscope technology to probe for Rprm RNA to validate the efficacy of this knockout model and visualize changes in expression. We found a significant decrease in Rprm expression in the VMH of

Rprm KO mice when compared to littermate controls, but no change in the ARC. Given that Rprm-expressing neurons are estrogen-sensitive, we wanted to determine whether knocking out Rprm in the VMH alters Esr1 expression levels, the gene encoding for estrogen-receptor alpha. We found no differences in Esr1 expression levels between Rprm KO mice and littermate controls. With the Rprm KO model validated, we analyzed the effects on body temperature. Preliminary results show that Rprm KO mice have lower body temperature than their littermate controls. This experiment aids in distinguishing the effects of transient versus long-term genetic manipulations, and may point to the permanent deletion of Rprm altering the ability of these mice to thermoregulate, which our lab is investigating further.

D-3. “Leptin Activated Hypothalamic BNC2 Neurons Acutely Suppress Food Intake in Mice”

Han L. Tan¹, Luping Yin², Yuqi Tan³, Jessica Ivanov¹, Kaja Plucinska⁴, Anoj Ilanges¹, Brian R. Herb⁵, Putianqi Wang¹, Christin Kosse¹, Paul Cohen⁴, Dayu Lin², Jeffrey M. Friedman¹

¹ Laboratory of Molecular Genetics, Howard Hughes Medical Institute, The Rockefeller University, New York, NY, USA. ²

Department of Psychiatry, Neuroscience Institute, New York University Langone Medical Center, New York, NY, USA. ³

Department of Microbiology and Immunology, Stanford University School of Medicine, Stanford, CA, USA. ⁴ Laboratory of

Molecular Metabolism, The Rockefeller University, New York, NY, USA. ⁵ Institute for Genome Sciences, Department of

Pharmacology, University of Maryland School of Medicine, Baltimore, MD, USA

The balance between hunger and satiety is crucial for maintaining energy balance, largely regulated by the hormone leptin. Leptin, produced by adipose, targets the hypothalamus's arcuate nucleus (ARC) to control food intake. Two neuron types in this system, orexigenic AGRP and anorexigenic POMC, express leptin receptors (LepR), and are central to hunger regulation, representing a 'yin-yang' model. Leptin promotes satiety by inhibiting AGRP and activating POMC neurons. While AGRP neurons regulate feeding on a rapid time scale, acute activation of POMC neurons has a minimal effect, suggesting that there is a heretofore unidentified leptin-regulated population that acutely suppresses appetite. Through single nuclei RNA-seq, we identified a novel population of leptin-target neurons expressing basophilin 2 (BNC2). Functional studies revealed BNC2 neurons suppress food intake with kinetics similar to the swift induction of feeding after AGRP neuron activation by monosynaptically inhibiting AGRP neurons. Furthermore, activating BNC2 neurons induces positive valence, suggesting that it suppresses the negative emotional effects of AGRP activation. Acute activation of BNC2 neurons also regulated peripheral glucose metabolism by decreasing glucose levels and increasing insulin sensitivity, an effect opposite to that of AGRP neuron activation. Finally, deleting LepR in BNC2 neurons of adult mice leads to marked hyperphagia and obesity, similar to the effects observed in LepR knockouts in AGRP neurons. These findings reveal that BNC2-expressing neurons are a crucial missing component in the neural circuit that maintains the balance between hunger and satiety, providing the 'yang' to the AGRP neurons' 'yin' in the regulation of feeding.

D-4. “Alterations of the Oxytocin system in Gaming Disorder: insights from a rat model study involving both sexes”

Antonino Casile^{1,2}, Brigitta Bonaldo⁴, Alice Fallaha², Martina Bettarelli², Maria Vittoria Micioni Di Bonaventura², Stefano Gotti^{2,3}, and Carlo Cifani¹

¹ University of Camerino, School of Pharmacy, Camerino (MC) Italy, ² Neuroscience Institute Cavalieri Ottolenghi (NICO)

University of Turin, Italy, ³ Department of Neuroscience “Rita Levi-Montalcini”, Turin (TO), Italy, ⁴ Department of Health

International Classification of Diseases (ICD-11) classified Gaming Disorder (GD) as a mental disorder. GD mainly occurs among adolescents, who after developing addiction, show psychopathological traits, such as social anxiety, depressive disorder, and social isolation. However, the different studies conducted in humans so far show several limitations, including exposure period, duration, and sex. Trying to address the lack of an experimental model for such a disorder, in the present work we proposed a new GD rat model to investigate some peculiar tracts of the disorder. GD-rats showed a significant increase in frequency and duration of play, and time spent in front of the screen compared to controls (CON). In addition, GD-females showed greater interaction and duration of play, which was maintained even in presence of sexual or social stimuli compared to GD-males, and reduced interaction time to CON-females. Quantitative analysis of immunoreactivity of Oxytocin in the Paraventricular nucleus showed a significative reduction in GD-rats compared to CON-groups. The loss of control over play, anxiety, and hyperactivity induced by the protocol confirms the translational validity of model. The preference of play over social interaction supports the hypothesis of the involvement of Oxytocinergic system in this mental disorder. Oxytocin is a potent neuroendocrine hormone involved in numerous pathologies, such as mental illness and addiction. Its alteration leads to the development of compulsive and maladaptive behaviors, such as those of addiction. It is therefore possible that alteration of this system may predispose or be one of the causes of onset of this mental disorder.

D-5. “Periadolescent Blue Light Exposure and Brain Development in the Rat Model”

Dominic Meszaros¹, Victoria Riesgo¹, Jari Willing¹

¹ Bowling Green State University

The introduction and proliferation of blue light emitting electronic devices to pre-adolescent humans has raised concern regarding its developmental impacts. Due to sampling and ethical concerns, animal models present an avenue to explore such effects. Current research indicates the prolonged BL exposure to adolescent and adult rats is harmful for proper sexual development as well as learning and memory respectively. However, an exploration of the behavioral effects of BL exposure in adolescents or pre-adolescents has not been conducted, such as spatial memory and anxiety. Both males and females (n=53) were assigned to six-hour blue light exposure (P25-55) or control. A behavioral battery of tests were run: open field testing, elevated plus maze, and novel object recognition (NOR). Anxiety, learning and memory were measured respectively. Results showed no precocious puberty with exposure in males or females. No anxiety-like impairment was seen. However, learning and memory tasks exhibited “impairment-like” behavior. Using Nissl and MAP-2 stained brain tissue, we are currently examining cortical thickness, volume of the prefrontal cortex, neuron number in the PFC and dendritic complexity in both the PFC and Hippocampus. These tests should be sufficient to describe structural differences in brain composition across groups. Results are pending. Comparative analyses could then be made between rats and humans in a proper, limited capacity using established literature.

D-6. “Insulin-like growth factor (IGF)-1 acts through Kiss1 neurons in mice to influence metabolism and reproduction”

Mengjie Wang^{1,2}, Muhammad Naveed¹, Yong Xu², Jennifer W. Hill¹

¹ Center for Diabetes and Endocrine Research, University of Toledo College of Medicine, Toledo, Ohio, USA. ² Department of Molecular and Cellular Biology, Baylor College of Medicine, Houston, TX, USA.

Kisspeptin, encoded by the Kiss1 gene, ties puberty and fertility to energy status; however, the metabolic factors that control Kiss1 neurons need to be clarified. To evaluate the impact of IGF-1 on the metabolic and reproductive functions of kisspeptin circuits, we created mice lacking IGF-1 receptors in Kiss1 neurons (IGF1RKiss1 mice). Previous studies have shown that IGF-1 and insulin can bind to each other's receptors, permitting IGF-1 signaling in the absence of IGF1R. Therefore, we also generated mice with simultaneous deletion of the IGF1R and insulin receptor (IR) in Kiss1 neurons (IGF1R/IRKiss1 mice). Female IGF1RKiss1 mice had a "metabolically healthy" profile with lower body weight and food intake, plus higher energy expenditure and physical activity. This phenotype was linked to higher pro-opiomelanocortin (POMC) expression and heightened brown adipose tissue (BAT) thermogenesis. Notably, IGF1RKiss1 mice also experienced delayed puberty and adult reproductive deficits. Male IGF1RKiss1 mice had lower gonadotropin and testosterone levels and impaired spermatogenesis. Thus, IGF-1 signaling in Kiss1 neurons impacts metabolism and reproduction in a sex-specific manner. IGF1R/IRKiss1 mice had higher fat mass and glucose intolerance, suggesting IGF1R and IR in Kiss1 neurons together regulate body composition and glucose homeostasis. Overall, our study shows that Kiss1 IGF1R and IR have cooperative roles in body weight, energy balance, glucose homeostasis, puberty, and fertility.

D-7. “The effect of age on pigeon (*Columbia livia*) decision-making in a variable reward, spatial memory task”

Timothy M. Burns¹, Verner P. Bingman¹

¹ Bowling Green State University Psychology Department

Recent studies on the avian hippocampus suggest that pigeons experience changes in hippocampal function as they age. The present research compared the behavior of young and old homing pigeons in a spatial memory task. Twelve (12) unsexed homing pigeons (*Columbia livia*) were divided into two experimental groups consisting of a younger group aged less than 7 years and an older group aged greater than eleven years. Subjects were trained to locate baited food bowls in an open floor arena with scattered landmark cues. Two food locations were never baited with food, while two others were baited with either a small, constant reward (2 food pellets per trial) or a large, variable reward (5 food pellets intermittently available). First order results suggest that the older pigeons do not suffer from spatial memory decline: there was no significant difference between the two experimental groups with respect to error rates (choosing an empty food bowl location). Further analysis revealed that older pigeons preferred the small, constant reward location in both the low and high variable conditions, whereas young pigeons only preferred the small, constant reward location in the low variable condition. In the high variable condition, younger, but not older, pigeons preferred the large, variable reward location. We propose that the avian hippocampus undergoes functional changes because of aging. Further research will be necessary to probe how the avian hippocampus and related structures evaluate risk and reward, and how that evaluation may change as a function of age.

D-8. “Effects of Social Stress on Corticotropin Releasing Hormone Activity in Female *Peromyscus californicus*”

Sinéad Archdeacon¹, Alexandra Serna Godoy¹, Alison V. Ramirez¹, Jessie “Jie” Li², Brian C. Trainor, PhD³

¹ College of Biological Sciences, University of California, Davis. ² UC Davis Genome Center ³ Department of Psychology, University of California, Davis

Anxiety disorders are a major public health concern, and developing more effective treatments requires a better understanding of the biological basis of stress responses. One major hormone involved in this response is corticotropin releasing hormone (CRH), which controls cortisol release via the hypothalamic-pituitary-adrenal (HPA) axis. Previous studies in *Peromyscus californicus* mice found that HPA activation after social stress is more pronounced in females than males. CRH has important effects on anxiety-related behavior by acting in the brain, but little is known about how stress alters the molecular biology of CRH-producing cells. To determine how stress affects transcription in Crh neurons, female *Peromyscus californicus* mice were randomly assigned to control or social stress conditions. Nuclei from four control and four stressed mice were sequenced using the 10x Genomics platform and Crh neurons were identified. After controlling for the false discovery rate, 51 transcripts were found to be upregulated by stress while 53 transcripts were downregulated. Transcript function was determined using Gene Ontology enrichment analysis. Changes in Crh neurons were also compared with stress-induced expression changes in oxytocin and vasopressin neurons. Results suggest that Crh neurons show a unique stress response profile compared to other peptidergic neurons, and indicate synaptic plasticity as an important mechanism of stress response in this cell type.

D-9. “Adolescent social isolation induces microglial phagocytosis and astrocyte atrophy in the nucleus accumbens and promotes adult social behavior dysfunction in rats.”

Jonathan W VanRyzin¹, Kathryn J Reissner¹

¹ Department of Psychology and Neuroscience, University of North Carolina- Chapel Hill, Chapel Hill, NC, USA

Adolescence is a dynamic developmental period marked by brain circuit refinement and social stress during this time can influence the development of maladaptive behaviors and increase the risk of later-life psychiatric disorders. However, the mechanisms by which this happens remain unknown. Astrocytes and microglia both promote circuit refinement during development and regulate neural function at maturity and are subject to dysregulation by stress. Thus, we aimed to test the hypothesis that adolescent social isolation affects later-life behavior by impairing glial function within the nucleus accumbens (NAc), a brain region responsible for integrating social, reward, and stress information. We isolated or group-housed male and female Long Evans rats from weaning until adulthood (P21-P56). From P57-P58, rats underwent social behavior testing with a novel age- and sex-matched conspecific in social interaction test and 3-chamber preference test. Following behavior, brains were collected for immunohistological analysis of astrocytes and microglia using GFAP and Iba1, respectively. Socially-isolated rats spent more time interacting with a novel conspecific compared to group-housed controls in the social interaction test, while there were no differences in social preference index between groups. We found GFAP+ astrocytes within the NAc of socially-isolated rats had decreased

structural complexity and a corresponding increase in the percentage of phagocytic Iba1+/CD68+ microglia. Phagocytic microglia had significantly more inclusions that co-labeled with the astrocyte marker GLT-1. Our results demonstrate that adolescent social isolation stress induces aberrant phagocytosis of astrocytes within the NAc as a putative mechanism by which isolation stress drives astrocyte atrophy and behavioral dysregulation.

D-10. “Cortical astrocyte regulation of glutamate synapses decreases in a sex-dependent manner following ethanol drinking in adolescence and adulthood in male and female mice.”

Andi Liss¹, Florence P. Varodayan¹

¹ Behavioral Neuroscience Program, Department of Psychology, Binghamton University-SUNY, Binghamton, NY, USA
Prefrontal cortex (PFC) pathology is associated with the cognitive symptoms of alcohol use disorder (AUD); however, the underlying mechanisms remain understudied. Preclinical studies in male rodents have shown that ethanol exposure in adolescence or adulthood alters glutamatergic transmission in the medial PFC (mPFC), and that astrocytes can also influence glutamatergic transmission by buffering calcium signaling and recycling glutamate. Following adolescent ethanol drinking, cortical astrocytes in adult male mice show reduced surface area to volume ratios and synaptic contacts, suggesting a decreased ability to regulate synapses. To date, no studies have examined the role of astrocytes in ethanol's synaptic actions in the mPFC of either sex, or if this change in astrocyte morphology following adolescent drinking produces long-term functional changes at glutamate synapses. Here, we assessed the role of astrocytes in ethanol's regulation of glutamate transmission in prelimbic (PL) mPFC layers 2/3 and 5 pyramidal cells in adult male and female C57BL/6J mice using ex vivo electrophysiology following chronic ethanol drinking in adolescence or adulthood. We found that acute ethanol increased glutamate release in PL layer 2/3 in water-drinking male adults and layer 5 in water-drinking female adults. Pharmacological reduction of astrocyte activity blocked these sex- and layer-specific effects. After chronic ethanol drinking in adolescence or adulthood, acute ethanol synaptic regulation in adulthood was no longer observed. Collectively, our findings demonstrate that mPFC astrocytes play critical roles in the cellular and behavioral actions of ethanol in both male and female mice. Ongoing molecular studies are examining the specific astrocyte mechanisms involved.

D-11. “Local synthesis of estrogens in the brain is required for social recognition in male and female Syrian hamsters”

Aspesi D.¹, Albers H.E.¹

¹ Neuroscience Institute and Center for Behavioral Neuroscience, Georgia State University, Atlanta GA USA.
Social recognition (SR) is an essential social behavioral skill for identifying previously encountered conspecifics and emitting appropriate behaviors. Gonadal steroid hormones, such as testosterone (T) and 17 β -estradiol (E2), influence SR. The mechanisms by which T affects SR, either through androgenic or estrogenic pathways via aromatase conversion into E2, remains unclear. Local brain synthesis of steroids may be critical for rapid social adaptation, making it essential to explore the role of neuro-derived steroids in SR. In this study, gonadally intact male and female Syrian hamsters received an intracerebroventricular (icv) infusion of letrozole, an aromatase inhibitor, 15 minutes before an SR test. The test included a 3-minute sample phase with two conspecific odors, followed by a 20-minute break and a 3-minute test phase with a new and a familiar odor. Letrozole impaired SR in

both sexes, suggesting a role for neuro-derived estrogens. In gonadectomized males and females, icv infusion of either T or E2 10 minutes before the test facilitated SR. A modified SR test with a 10-minute break, which did not impair SR in castrated males, was also used to evaluate letrozole's effects. Letrozole blocked SR in gonadectomized males and females, and only E2 infusion reversed this impairment, emphasizing the rapid modulation of SR by locally synthesized E2. These findings highlight aromatase's role in converting T to E2 in the Syrian hamster brain, crucial for regulating social behaviors. Understanding these rapid steroid hormone mechanisms is essential for elucidating the neurocircuitry involved in social behavior modulation.

D-12. N/A

D-13. “Hypothalamic Estrogen Signaling Coordinates Maternal Temperature Regulation in Mice”

Adriana R Vree^{1,2}, Laura R. Cortes^{1,2}, Mia Hansen¹, Sakina Rashid¹, Edward Van Veen^{1,3}, & Stephanie M. Correa^{1,3}

¹ University of California, Los Angeles Department of Integrative Biology & Physiology ² Co-first authors ³ Co-senior authors

High body temperature is a teratogen for developing fetuses; therefore, maintaining optimal temperature is critical during pregnancy. Core temperature (T_c) decreases in late pregnancy, possibly to protect and/or divert metabolic resources to the fetus. Estrogen signaling via estrogen receptor (ER) α in the medial preoptic area (MPO) modulates a variety of functions including thermoregulation and energy homeostasis. Previous work demonstrated that activating ER α neurons in the MPO of non-pregnant female mice decreases T_c, tying estrogen-sensitive cells to thermoregulation. We aim to dissect how hypothalamic estrogen signaling orchestrates thermoregulatory adaptations during pregnancy. Because circulating estrogen levels increase during pregnancy, we hypothesize that pregnancy is accompanied by upregulation of estrogen-signaling in the MPO. Stereotaxic delivery of a novel reporter, NeuroSeeER, to the MPO allows for the visualization of cells with high levels of estrogen-dependent gene expression (i.e., estrogen signaling) via expression of green fluorescent protein (GFP). We find the number of GFP+ cells was significantly greater in late pregnant mice relative to non-pregnant controls. This increase may be related to changes in the expression of estrogen receptors. To answer this, we used RNAscope to compare the number of MPO neurons expressing ER α (Esr1) and ER β (Esr2) in non-pregnant and pregnant mice. Despite greater estrogen signaling at late pregnancy, we found no difference in Esr1 and decreased expression of Esr2 in pregnant mice relative to non-pregnant. In summary, pregnancy requires altered regulation of temperature and metabolism, and the reported changes in hypothalamic estrogen signaling late in pregnancy may facilitate maternal homeostasis.

D-14. “Neural correlates of vocal communication during pair bond expression in prairie voles”

Dami Solaja¹, Rodrigo Muñoz-Casteñada^{3,4}, Pavel Osten³, Steven M. Phelps^{2,5}, Morgan L. Gustison^{1,2}

¹ Department of Psychology, Western University, London, Canada; ² Department of Integrative Biology, The University of Texas at Austin, Austin, United States; ³ Cold Spring Harbor Laboratory, Cold Spring Harbor, United States; ⁴ Appel Alzheimer's Disease Research Institute, Feil Family Brain and Mind Research Institute, Weill Cornell Medicine, New York, United States; ⁵ Institute for Neuroscience, The University of Texas at Austin, Austin, United States

The establishment of social bonds is crucial to the health of individuals. Maintenance of social bonds requires the integration of various sensory modalities such as auditory cues. Although research has established that communication is important for social bonding, the convergence of these neural circuits is poorly understood. To investigate this, female and male prairie voles (*Microtus ochrogaster*), a socially monogamous rodent, were paired and exposed to one of four social contexts. These contexts differed based on whether individuals were isolated or exposed to an opposite-sex partner and/or stranger. Ultrasonic vocalizations (USVs) were recorded during these behavioral observations. Brain tissue was analyzed for immediate early gene c-Fos expression in over 800 regions of interest (ROIs) using a whole-brain iDISCO imaging pipeline. It was hypothesized that USV rates would be associated with c-Fos expression in the ROIs that varied with social context. Twenty-five ROIs (e.g., subparafascicular nucleus) showed varying c-Fos expression across contexts. Seven of these ROIs were positively correlated to USV rates (e.g., basolateral amygdalar nucleus), while four ROIs were negatively correlated to USV rates (e.g., anterodorsal nucleus). A unique set of regions were inversely correlated with USV rates during female trials compared to males (e.g., nucleus circularis), but there was no significant difference in USV rates between female and male trials. Together, these findings suggest that neural circuits involved in vocalization are sex dependent, with underlying neuroendocrine differences that result in diverging neural patterns. Further research is needed to understand the specific functions of these ROIs during vocal communication.

D-15. “Effect of sex and estrous cycle on BNSTdl-CRF neuron activity and their role in defensive behaviors in male and female rats”

Rachel Chudoba¹, Joanna Dabrowska¹

¹ Rosalind Franklin University

Corticotropin-releasing factor (CRF) is a neuropeptide produced in the dorsolateral bed nucleus of the stria terminalis (BNSTdl), a region that mediates behavioral responses to stress. BNSTdl neurons can be classified as Type I-III based on electrophysiological properties. Using slice electrophysiology, we found that BNSTdl-CRF neurons in CRF-Cre transgenic male (n=24) and female rats (n=39) have hyperpolarized resting membrane potential relative to Type I/II neurons, delayed spikes, and prominent inward rectification, classifying them as Type III. Females also have lower rheobase than males (p=0.03, unpaired t-test) and lower spontaneous excitatory post-synaptic current amplitude (p=0.0002, one-way ANOVA) than males, particularly during proestrus (p=0.0002) and diestrus (p=0.01, Tukey's). In CRF-Cre rats expressing Cre-dependent inhibitory designer receptors, we assessed the effect of CRF neuron silencing with clozapine-N-oxide (CNO) on fear recall following unpredictable fear conditioning. Saline-treated males showed heightened post-shock startle compared to CNO-treated males during recall test 1 (p=0.0052, unpaired t-test). CNO-treated females showed heightened startle between cue presentations (non-cued fear) compared to saline-treated females during test 3 (p=0.0077, unpaired t-test), particularly during diestrus (p=0.03). Overall, BNSTdl-CRF neurons are more excitable in females and have reduced postsynaptic glutamate sensitivity during proestrus and diestrus compared to males. Following fear conditioning, BNSTdl-CRF neurons promote heightened post-shock startle in males and non-cued fear extinction in diestrous females (low estrogen and progesterone). All in all, BNSTdl-CRF neurons promote threat-sensitive changes in defensive strategy in a sex- and estrous phase- dependent manner.

D-16. N/A

D-17. “Sex differences in memory retention and social behaviors after social isolation in Alzheimer’s disease mice”

L. Toennies¹, K. Kaplan^{1,3}, N. Ferrara^{2,3}, H.C. Hunsberger^{1,3}

¹ Center for Neurodegenerative Diseases & Therapeutics, Rosalind Franklin University of Medicine and Science, ² Center for Neurobiology of Stress Resilience & Psychiatric Disorders, Rosalind Franklin University of Medicine and Science, ³ The Chicago Medical School

Alzheimer's disease (AD), the leading cause of dementia, disproportionately impacts women compared to men. Early detection and intervention strategies are needed to reduce future disease risk. Loneliness and isolation represent significant risks in dementia transition. Maintaining strong social connections is associated with reduced mortality rates and a lower risk of AD. An increasing prevalence of loneliness and AD after the COVID-19 pandemic proposes an urgent need to better understand the connection between isolation and AD progression within the context of sex differences. Following isolation or group housing of AD and control mice at 2-3 months of age, we measured social interaction and fear memory. Interestingly, we observed increased memory retention in isolated AD female mice compared to group-housed female and male mice. Our results provide the first evidence of potential memory enhancement after acute isolation. Previous studies were only performed in male rodent models and have not investigated an earlier time point before AD pathology. Using immunohistochemistry targeting c-Fos, PV interneurons, and inflammatory markers, we aim to understand the brain circuitry involved, how aging and long-term isolation impact these results, and whether this memory enhancement remains intact in other memory tasks.

D-18. “The central amygdala corticotropin releasing factor system in anxiety-like behavior in rats: Impact of age and sex”

Kelcie Schatz¹, Margaret Agajanov¹, Elena Varlinskaya¹, Marvin Diaz¹

¹ Psychology Department- Behavioral Neuroscience, Binghamton University, NY

The corticotropin-releasing factor (CRF) system is an important mediator of stress and anxiety-related behaviors. In accordance with the canonical anxiogenic role of CRF, blockade of CRF1 receptors (CRF1Rs) in the medial subnucleus of the central amygdala (CeM) of adult male rats attenuates anxiety-like behavior, suggesting that endogenous CeM CRF1R activation facilitates anxiety-like behavior. However, the role of CeM CRF1R activation in anxiety-like behavior across development and in both sexes has been overlooked. Our lab has demonstrated that the physiological consequences of CeM CRF1R activation are age and sex dependent. In adult males, CeM CRF1R activation increases inhibitory neurotransmission, whereas in adolescent males, CeM CRF1R activation decreases inhibitory neurotransmission. However, this developmental switch is absent in females, as CeM CRF1R activation decreases inhibitory neurotransmission in both adolescent and adult females. Taken together, this evidence highlights the importance of investigating the role of CeM CRF1R activation across development in males and females. To explore this, experiments are being conducted to measure anxiety-like behavior in the light-dark box test in juvenile (~postnatal day P 25), adolescent (~P45), or adult (~P80) male and female rats following an intra-CeM infusion of Stressin-1, a CRF1R

agonist, or vehicle. Effects of Stressin-1 were sex-dependent, with CeM CRF1R activation decreasing anxiety-like behavior in adult females. No effect of CeM CRF1R activation was detected in males of any age. A better understanding of the developmental trajectory of the CeM CRF system could help inform pharmacological treatments for anxiety-related disorders.

D-19. “Impact of light on female mouse reproductive development and function”

Kayla Miguel¹, Marcos Aranda¹, Eve Gold¹, Leandra Hawkins¹, Lily Ren¹, Aaniyah Jarrette¹, Anika Schipma¹, Christian Hernandez¹, Farners Amargant Riera², Francesca Duncan², Tiffany Schmidt¹

¹ Neurobiology Department, Northwestern University, ² Department of Obstetrics and Gynecology, Feinberg School of Medicine, Northwestern University

In mammals, light regulates many aspects of physiology and behavior. Light input from the retina is conveyed by retinal ganglion cells (RGCs) to over 40 different brain regions, including multiple hypothalamic nuclei involved in hormone release and sex-specific behaviors. However, the role of light in modulating many of these hypothalamic functions is unknown. One well-established role of light is the photoentrainment of the hypothalamic suprachiasmatic nucleus (SCN), the central circadian pacemaker. The SCN is involved in the timing of hormone release and ovulation, and aberrant lighting conditions that disrupt circadian rhythms can lead to worse reproductive outcomes. The main retinal input to the SCN is through the melanopsin-expressing intrinsically photosensitive retinal ganglion cells (ipRGCs). While the role of melanopsin has been studied in photoentrainment, it has not been studied in the context of reproductive measures. Here, we examine the roles of light and melanopsin on puberty onset, estrus cycle length, and ovarian reserve. To test the role of light, female mice were placed from birth in light-tight boxes in either LD (12 hours light, 12 hours dark) or DD (constant dark). Mice reared in DD had delayed puberty onset, shorter estrus cycles, and higher ovarian reserve than mice reared in LD. We next repeated these experiments in melanopsin null animals and found that these light-dependent differences did not require melanopsin. Overall, light regulates these reproductive measures and could likely play a role in other hypothalamic sexual functions and behaviors.

D-20. “Investigating sex differences in hippocampal neurogenesis in rats during first week of life in response to postnatal THC exposure”

Christie V. Dionisos¹, Jonathan VanRyzin², Margaret M. McCarthy²

¹ University of Maryland School of Medicine Program in Neuroscience, ² University of Maryland Medicine Institute for Neuroscience Discovery (UM-MIND)

Decriminalization of cannabis containing products and positive perceptions of cannabis use in the United States have resulted in increased use among pregnant people to alleviate nausea and pain, which may have adverse effects on their offspring. Various studies have looked at adverse changes in response to Δ^9 -tetrahydrocannabinol (THC), the primary psychoactive component of cannabis, either prenatally or in adult rodents, but few have investigated THC's effects in early postnatal hippocampal development. There is a well characterized and developmentally restricted sex difference in neurogenesis from birth through the first postnatal week in the rodent hippocampus in which males have close to twice as much neurogenesis than females. Our previous research has shown that in the

developing amygdala, postnatal THC exposure increases endocannabinoid tone (higher in males than females as it is testosterone driven) which results in fewer newborn cells in females only, effectively masculinizing newborn cell number and social play behavior in females while leaving male numbers unchanged. Here we investigate if postnatal THC exposure would similarly alter neurogenesis in a sex specific manner in the developing hippocampus. We hypothesize that intraperitoneal postnatal THC administration on postnatal days 0-3 increases neurogenesis in females only, masculinizing newborn cell number and effectively abrogating the naturally occurring sex difference. We will then explore the interactions of hormone receptors and endocannabinoid receptors in the developing hippocampus as well as its reciprocal connections to the developing amygdala, to better understand sex specific and region specific proliferation rates and subsequent altered social behavior.

D-21. “Pause for thought: How menopause and obesity affect a mouse model of Alzheimer’s disease.”

Charly Abi-Ghanem¹, Caitlyn Valerian¹, Aaron S. Paul¹, Abigail E. Salinero¹, Rachel M. Smith¹, Richard D. Kelly¹, Riane N. Richard¹, Ava A. Herzog¹, Christina A. Thrasher¹, Emily Groom¹, Matthew Wang¹, Kasey M. Belanger¹, Heddwen L. Brooks³, Damian G. Zuloaga², and Kristen L. Zuloaga¹

¹ Department of Neuroscience & Experimental Therapeutics, Albany Medical College, 47 New Scotland Avenue; MC-136, Albany, NY 12208, USA. ² Department of Psychology and Center for Neuroscience Research, State University of New York at Albany, 1400 Washington Ave, Biology 325, Albany, NY 12222, USA. ³ Department of Physiology, Tulane University School of Medicine, New Orleans, LA 70112, USA.

Menopausal women (60+yo) constitute about two-thirds of those with Alzheimer’s disease (AD). Menopause increases dementia risk by heightening the likelihood of metabolic, cardiovascular, and cerebrovascular diseases. Mid-life metabolic disease (e.g. obesity, diabetes, or prediabetes) is a well-known risk factor for dementia. A high fat diet can lead to poor metabolic health in both humans and rodents. This study aimed to determine the effects of menopause and high fat diet on metabolic, cognitive, and pathological outcomes in the AppNL F knock-in mouse model of AD. We used an accelerated ovarian failure model (4-vinylcyclohexene diepoxide, VCD) to simulate menopause. This ovary-intact model is more clinically relevant than ovariectomy, as mice undergo a perimenopausal period. At 3 months of age, AppNL F mice received VCD or vehicle (oil) and were then placed on either a control diet (10% fat) or a high-fat diet (HF; 60% fat) until 10 months of age. Menopause led to weight gain, glucose intolerance, and exacerbated obesity in response to a high-fat diet. An interaction between HF diet and menopause impaired episodic-like memory. Despite having no effects on amyloid pathology, menopause induced alterations in microglial response, myelin, and hippocampal neurogenesis. This work highlights the need to model endocrine aging in animal models of dementia and will contribute to further understanding the interaction between menopause and metabolic health in the context of AD.

D-22. “The role of the microglial phagocytic receptor, MerTK, in early-life adversity-induced changes in behavior and synapse development”

Urjoshi Kar¹, Madison Garvin¹, Jessica Bolton¹

¹ Georgia State University Neuroscience Institute

Early-life adversity (ELA) can profoundly impact an individual's risk for emotional disorders, likely by modulating the maturation of stress-related brain circuits. Our lab has shown that ELA causes an increase in the number of excitatory synapses onto corticotropin-releasing hormone (CRH)-expressing neurons in the paraventricular nucleus of the hypothalamus (PVN). ELA inhibits microglial excitatory synapse engulfment in the PVN during development to result in this synapse excess, but the mechanism by which this occurs remains unknown. We hypothesized that ELA induces microglial dysfunction via inhibition of the microglial phagocytic receptor, MerTK, resulting in changes to synapses and behavior. Thus, we generated a novel microglia-specific (m)MerTK deletion-containing mouse line, and exposed litters to control or ELA conditions (modeled using the limited bedding and nesting paradigm) from postnatal days (P)2-9. Immunolabeled excitatory synapses in the PVN at P10 were imaged via confocal microscopy and quantified in wild-type and mMerTK-KO offspring. In adulthood, a separate cohort was tested in a behavioral battery to assess threat-response and anxiety-like behaviors. We found that ELA increased the number of excitatory synapses in the P10 PVN of males, but not females, which was mimicked by the mMerTK-KO in controls. Similarly, both ELA and the mMerTK-KO in control males provoked an increase in escape behavior in males in the looming-shadow threat task. Our results demonstrate that the mMerTK-KO mimics the synaptic and behavioral effects of ELA in control males, while MerTK deletion in ELA males had no additional effect, indicating the MerTK pathway may already be inhibited by ELA.

2025 SBN MEETING IN VANCOUVER !
JULY 13-16, 2025



HOPE TO SEE YOU IN VANCOUVER IN 2025

A promotional banner for the OSSD 19th Annual Meeting. The left side of the banner features a photograph of several colorful hot air balloons floating in a clear blue sky. The balloons have various patterns and colors, including red, orange, yellow, and white. The right side of the banner has a solid green background with a dark blue diagonal stripe. The text "OSSD 19TH ANNUAL MEETING" is written in large, white, sans-serif capital letters. Below this, in smaller white text, are the details: "Hotel Albuquerque", "Albuquerque, NM", "June 1-5, 2025", and the website "www.ossdweb.org". There are also some faint, stylized geometric shapes in the bottom right corner.