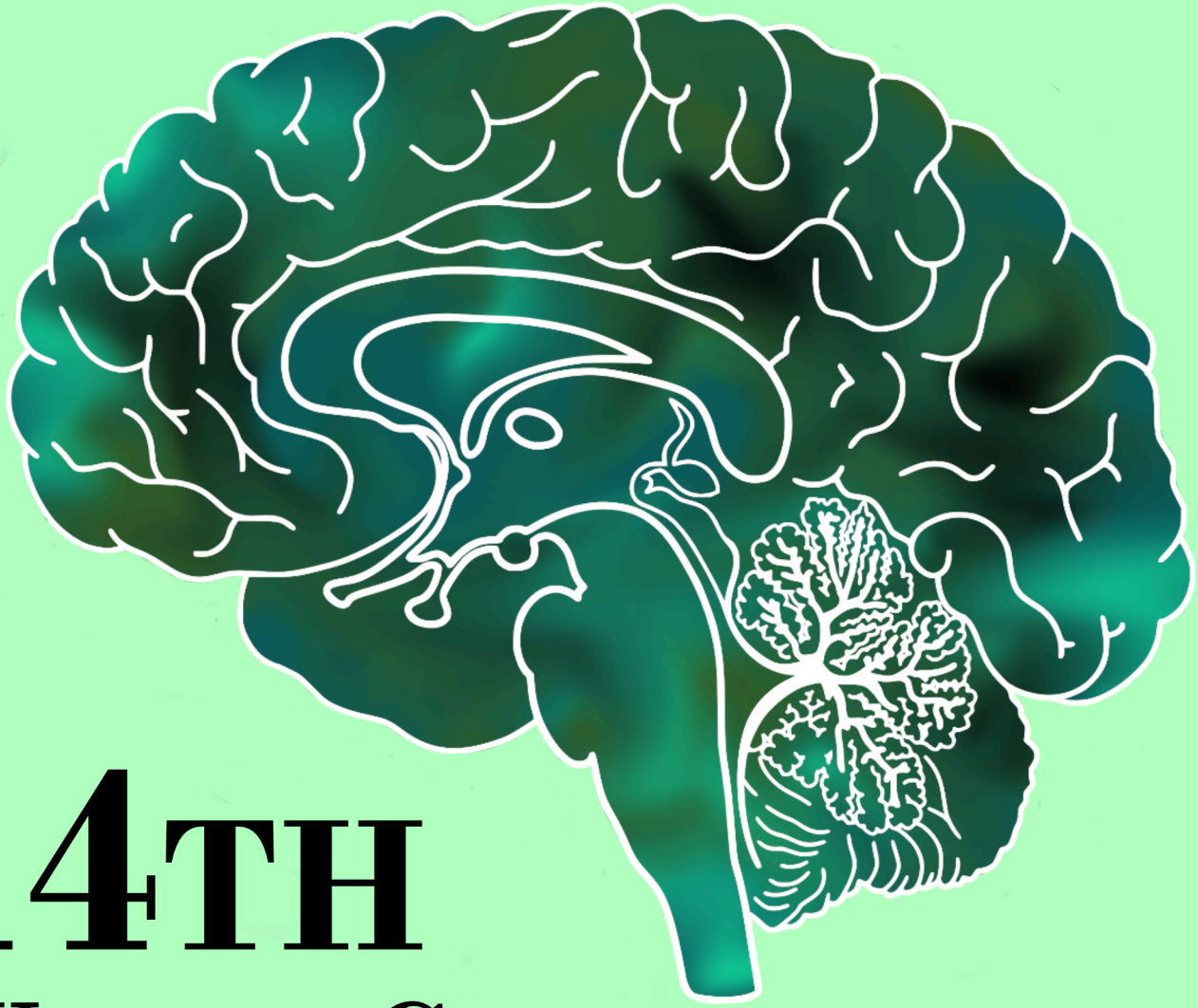
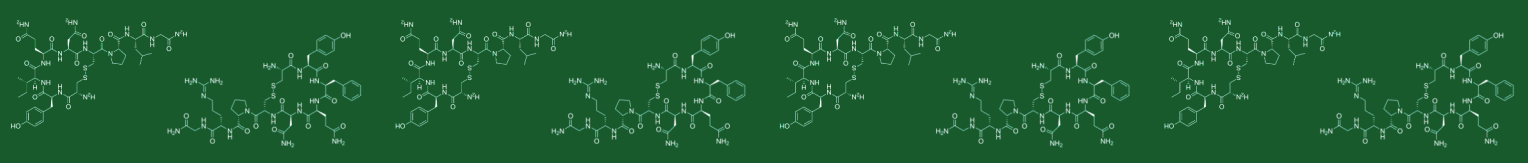


14th World Congress on Neurohypophysial Hormones

May 16-19, 2024 Atlanta, Ga



Contents

Welcome Message

Welcome to the 14th WCNH 2024 meeting!

The WCNH meeting continues to be the ultimate venue for young and established investigators engaged in comprehensive oxytocin and vasopressin research including developmental, physiological, behavioral, and clinical aspects of these major neuropeptides. Beyond its commitment to scientific excellence, the WCNH community is also dedicated to promoting diversity and equity in our field.

For the 14th WCNH meeting in Atlanta, we have designed an exciting scientific program showcasing a range of expertise and cutting-edge research in the oxytocin and vasopressin research field. Be sure to join us at the closing banquet, where we will celebrate our WCNH awardees, including recipients of the Glenn Hatton Travel Award and Best Poster Awards.

Alongside the exciting sessions programmed, we encourage you to explore Midtown Atlanta's excellent restaurants, parks, and museums, conveniently located within walking distance from the Historic Academy of Medicine.

We hope your experience at the WCNH in Atlanta is both enriching and enjoyable!

Javier Stern, M.D., Ph.D.
Chair, Local Organizing Committee
Director, Center for Neuroinflammation and Cardiometabolic Diseases
Distinguished Professor,
Neuroscience Institute
Georgia State University
Atlanta GA USA

Geert J. de Vries, Ph.D.
Local Organizing Committee
Regents' Professor and Chair
Department of Biology
Georgia State University

H. Elliott Albers, Ph.D.
Local Organizing Committee
Regents Professor of Neuroscience
Director, Center for Behavioral Neuroscience
Neuroscience Institute
Georgia State University

Larry Young, Ph.D.
Local Organizing Committee
William P. Timmie Professor of Psychiatry and Behavioral Sciences
Director, Center for Translational Social Neuroscience
Emory University
Director, Division of Behavioral Neuroscience and Psychiatric Disorders
Emory National Primate Research Center

Acknowledgments

Veronica Wright
Conference Administrative and Organizational Coordinator at Large, website management and design

Elba Lira Campos
Website/social media management and design

Morgan Gomez
Banners, Program, and Abstract Book Design

Manuel Bita Ongolo
Undergraduate volunteer team leader

Arkar Zaw, Sheryl Varghese, TyAzhia Goodwin, Varshini Kakarla, and Autuum Key
Undergraduate Volunteer team

Gary Brenneman, Ava Paine, Mark Jackson
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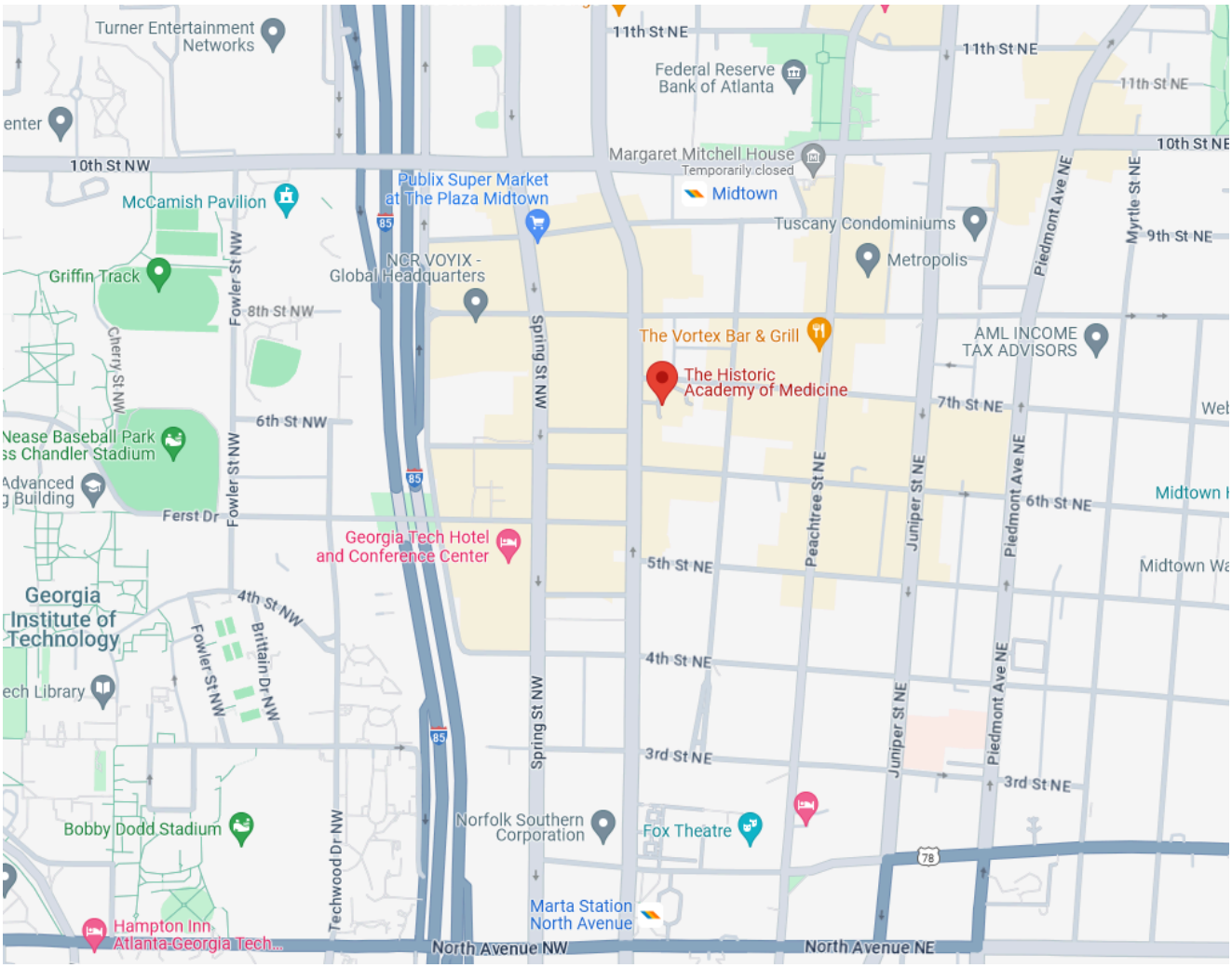
BSN
British Society for Neuroendocrinology

JNE Journal of Neuroendocrinology

INF

Midtown Atlanta



MARTA TRAIN SYSTEM

MAPS AND GETTING AROUND

THE MARTA

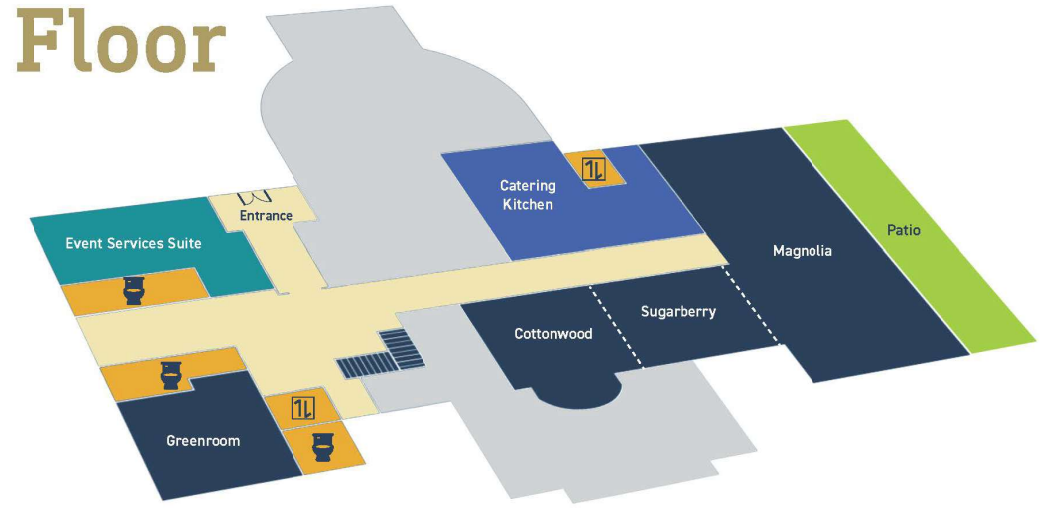
From Hartsfield Jackson International Airport:

Board either the Red (North Spring) or Gold (Doraville) Line trains and exit the train at Midtown Station (N4).
Exit Midtown station to 10th Street. Walk 1 ½ blocks east toward Peachtree Street. Turn left on Peachtree Street and walk one block north. Loews is located at the corner of Peachtree Street and 11th Street.

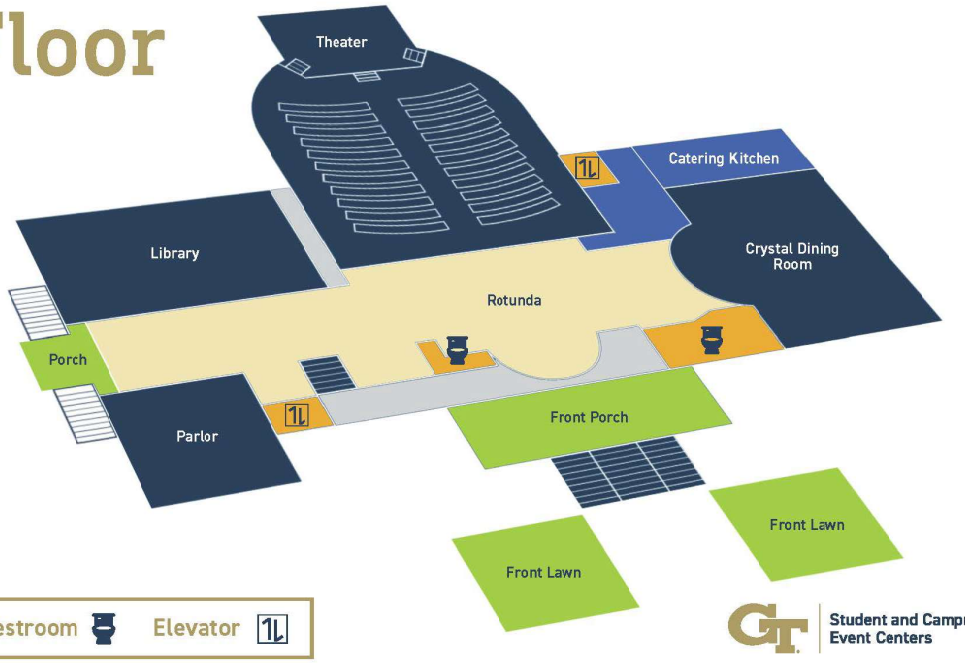
The Historic Academy of Medicine

The Historic Academy of Medicine

First Floor



Second Floor



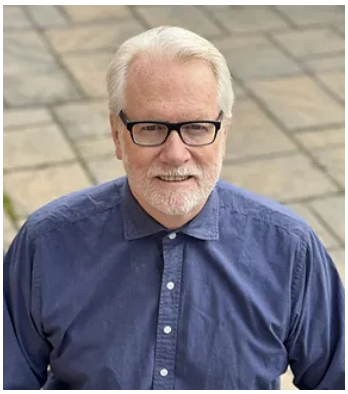
Restroom Elevator

Student and Campus Event Centers

Schedule at a Glance

	Thursday May 16th	Friday May 17th	Saturday May 18th	Sunday May 19th
8:00 AM				
		Plenary 2	Plenary 3	Plenary 4
9:00 AM		Coffee Break		
		Symposium 1	Symposium 5	Symposium 8
10:00 AM				Special Talk: Larry Young Scientific Contributions
11:00 AM		Coffee Break		
		Symposium 2	Symposium 6	Symposium 9
12:00 PM				
1:00 PM	Registration	Lunch	Lunch	Lunch
		Data Blitz	Poster 1	Poster 2
2:00 PM				
3:00 PM		Symposium 3	Symposium 7	Symposium 10
	Opening Session			
4:00 PM	Plenary 1		Journal of Neuroendocrinology: Brief Report & Breaking News	
		Coffee Break		Coffee Break
5:00 PM	Welcome Reception	Symposium 4	Free Time/ ATL Activities	Symposium 11
6:00 PM				
7:00 PM				Award Ceremony & Closing Banquet

Plenary Speakers



Charles Bourque - Mortyn Jones Lecture

McGill University, Canada

Bourque obtained a B.Sc.in Biology from the University of Ottawa and a Ph.D. in physiology under the supervision of Leo Renaud at McGill University (Montreal). Following Postdoctoral work with David A. Brown (University of London) he was recruited by the Department of Neurology-Neurosurgery at McGill University (currently Distinguished James McGill Professor) and as a Medical Scientist with the Research Institute of the McGill University Health Centre.

Robert Froemke

New York University, USA

Dr. Froemke is the Skirball Foundation Professor of Genetics in the Neuroscience Institute and Departments of Otolaryngology and Neuroscience/Physiology at NYU Grossman School of Medicine. The Froemke lab studies how sounds acquire meaning by relating synaptic plasticity to changes in behavior, such as the adaptations in the maternal brain to recognize infant cries or how cochlear implant stimulation leads to auditory perception.

Tatsushi Onaka

Jichi Medical University, Japan

Tatsushi Onaka is a Professor in the Department of Physiology at Jichi Medical University and has been the department head since 2011. He also serves as the Vice President of the Japan Neuroendocrine Society. Dr Onaka's research primarily focuses on the function of oxytocin neurons in regulating energy consumption, food intake, and stress coping behaviors.

Rae Silver

Columbia University, USA

Dr. Silver is a professor of Psychology at Columbia University and the Helene L. and Mark N. Kaplan Professor of Natural & Physical Sciences in the Neuroscience Department at Barnard College. She is the director of The Silver Neurobiology lab at Columbia, which studies the brain clock located in the suprachiasmatic nucleus (SCN) of the hypothalamus.

Thursday May 16th

1:00 - 4:00pm **Registration**

3:45 - 4:00pm **Opening Session**

Theater

4:00 - 5:00pm **Plenary Lecture 1**

Theater

Chair: Dr. Mike Ludwig - University of Edinburgh, Scotland

Rae Silver - Columbia University, USA
Portal pathways in the brain: Their potential for transporting neurovascular peptidergic signals

5:00 - 7:00pm **Welcome Reception**

Rotunda and Crystal Dining Room

Friday May 17th

8:30 - 9:15am **Plenary Session: Mortyn Jones Lecture**

Theater

Chair: Colin Brown - University of Otago, New Zealand

Charles Bourque - McGill University, Canada
Osmotic Control of Vasopressin: What we have learned since Verney and Andersson?

9:15 - 9:30am **Coffee Break**

Rotunda and Crystal Dining Room

9:30 - 11:10am **Symposium 1: Central Mechanisms in AVP and OXT information processing in health and disease**

Theater

Chair: Jeff Tasker - Tulane University, USA

9:30 - 9:55am **Alexandre Charlet - Centre National de la Recherche Scientifique, France**
Astrocytes in Mice Central Amygdala Mediates Oxytocin-dependent Behavioral Adaptation

9:55 - 10:20am **Tom Cunningham - UNT Health Science Center, USA**
Sex-based Diferences in Control of Neurohypophysial in a Model of Hyponatremia

10:20 - 10:45am **Zhihua Gao - Zhejiang University, China**
The Coordinative Role of Oxytocin Endocrine Neurons in Peripheral and Central Regulation

Friday May 17th

10:45 - 11:10am **Mike Ludwig - University of Edinburgh, Scotland**
Salt-loading reduces central osmoresponsiveness in magnocellular supraoptic neurones In-vivo

11:10 - 11:30am **Coffee Break**
Rotunda and Crystal Dining Room

11:30 - 1:10pm **Symposium 2: New Models and Approaches in AVP and OXT Research**
Theater

Chair: Yoichi Ueta - University of Occupational and Environmental Health, Japan

11:30 - 11:55am **Alec Davidson - Morehouse School of Medicine, USA**
Longitudinal Imaging of AVP Neuronal Behavior In-Vivo

11:55 - 12:20pm **Lang Geng - Beijing University, China**
Development and Optimization of Genetically Encoded Sensors for Oxytocin and Arginine Vasopressin

12:20 - 12:45pm **Quirin Krabichler - Heidelberg University, Germany**
Novel Transgenic Rat Models to Study the Functional Organization and Behavioral Roles of the Brain's Arginine Vasopressin System

12:45 - 1:10pm **Arthur Lefevre - University of California San Diego, USA**
Marmoset Monkeys as a Model of OT Action in Primates

1:10 - 3:00pm **Lunch**
Rotunda and Crystal Dining Room

2:00 - 3:00pm **Data Blitz**
Theater

3:00 - 4:40pm **Symposium 3: Coordinated Central and Periferal Actions of AVP and OXT**
Theater

Chair: Joe Verbalis - Georgetown University, USA

3:00 - 3:25pm **Atila Cihan - University of Basel, Switzerland**
Are Patients with Vasopressin Deficiency also deficient in Oxytocin?

3:25 - 3:50pm **Annette de Kloet - Georgia State University, USA**
Exploring Signaling Amongst Neurohypophyseal Hormones: A Complex Discourse that'll Elevate Your Blood Pressure

3:50 - 4:15pm **David Mendelowitz - George Washington University, USA**
Oxytocin Receptor Co-Localization in Brainstem Parasympathetic Cardiac Vagal Neurons

4:15 - 4:40pm **Takumi Oti - Okayama University, Japan**
Oxytocinergic Control Circuits in the Spinal Cord for Male Sexual Behavior

Friday May 17th

4:40 - 5:15pm **Coffee Break**

Rotunda and Crystal Dining Room

5:15 - 6:55pm **Symposium 4: Comparative Neuroendocrinology of AVP and OXT Systems**

Theater

Chair: Margarita Curras-Collazo - University of California, Riverside, USA;
Rui Oliveira - ISPA Instituto Universitario, Portugal

5:15 - 5:40pm **Christian Gruber - Medical University of Vienna, Austria**
Biological Function and Pharmacological Potential of Oxytocin Signaling in Ants

5:40 - 6:05pm **Rui Oliveira - ISPA Instituto Universitario, Portugal**
Evolutionarily Conserved Mechanism of Oxytocin in the Regulation of Social Behavior in Zebrafish

6:05 - 6:30pm **Allison Perkeybile - University of Virginia, USA**
Making Mothers: Pregnancy, Birth, and Epigenetic Regulation of the maternal Oxytocin Receptor Gene

6:30 - 6:55pm **Hirotaka Sakamoto - Okayama University, Japan**
Vasopressin/Oxytocin Peptide-signaling in Marine Planarians Functions as an Antidiuretic before Vascular System Acquisition and Synapse Evolution

Saturday May 18th

8:30 - 9:15am **Plenary Lecture**

Theater

Chair: Sue Carter - Indiana University, USA

Tatsushi Onaka - Jichi Medical University, Japan
Metabolic and Stress-coping Actions of Oxytocin

9:15 - 9:30am **Coffee Break**

Rotunda and Crystal Dining Room

9:30 - 11:10am **Symposium 5: Young Investigators in AVP and OXT Research**

Theater

Chair: Ryoichi Teruyama - Louisiana State University, USA

9:30 - 9:50am **Shelling Buffington - Baylor College of Medicine, USA**
Microbial Modulation of the Oxytocin-mesocorticolimbic Dopaminergic Pathway in Mouse for Autism

9:50 - 10:10am **Alex Castillo-Ruiz - Georgia State University, USA**
Long-term Effects of Cesarean Birth on Vasopressin and Oxytocin Neurons

Saturday May 18th

10:10 - 10:30am **Tim Gruber - Van Andel Institute, USA**
High-calorie Diets Uncouple Hypothalamic Oxytocin Neurons from a Gut-to-Brain Satiation Pathway Via K-opiod signaling

10:30 - 10:50am **Matt Kirchner - Georgia State University, USA**
Changes in Neuropeptide Large Dense Core Vesicle Trafficking Dynamics Contribute towards Adaptive Responses to a Systemic Homeostatic Challenge

10:50 - 11:10am **Elena Kozlova - University of California Riverside, USA**
Thyroid Dependent Disruption of Oxytocin and Gut Microbiome in an Enviromental Autism Mouse Model

11:10 - 11:30am **Coffee Break**
Rotunda and Crystal Dining Room

11:30 - 1:10pm **Symposium 6: The Dr. Larry J. Young Memorial Symposium: AVP and OXT in Neuropsychiatric Disorders**
Theater

Chair: Dev Manoli - University of California San Francisco (UCSF) & Karen Parker - Stanford University , USA

11:30 - 11:55am **Katrina Choe - McMaster University, Canada**
Investigating the Link Between ASD-risk Genes, oxytocin, and Social Behavior

11:55 - 12:20pm **Karen Parker - Stanford University, USA**
Vasopressin: A Trans-primate Biomarker of Social Impairment and Promising Treatment for Autism

12:20 - 12:45pm **Yannis Paloyelis - King's College London, UK**
Unravelling the Pharmacodynamics of Oxytocin Using Functional Neuroimaging

12:45 - 1:10pm **Julia Winter - University of Pennsylvania, USA**
Acute Versus Chronic Matters: Differential Behavioral and Molecular Effects of Oxytocin

1:10 - 3:00pm **Lunch**
Rotunda and Crystal Dining Room

1:10 - 3:00pm **Poster Session 1**
Magnolia / Sugarberry / Cottonwood

3:00 - 4:40pm **Symposium 7: Developmental Roles of OXT and AVP**
Theater

Chair: Elizabeth Hammock - Florida State University, USA

3:00 - 3:25pm **Bice Chini - Milan Center for Neuroscience, Italy**
Neonatal Oxytocin Administration in Mouse Models of Neurodevelpmental Disorders: Long Lasting Rescue Effects

Saturday May 18th

3:25 - 3:50pm **Heather Caldwell - Kent State University, USA**
Consequences of Altered Oxytocin and Vasopressin Signaling During Embryonic Development

3:50 - 4:15pm **Bruce S. Cushing - UT El Paso, USA**
Neonatal Organizational Effects of Oxytocin and Subsequent Behavioral Expression in Prairie Voles (Microtus ochrogaster)

4:15 - 4:40pm **William Kenkel - University of Delaware, USA**
The Role of Oxytocin in the Metabolic Consequences of Delivery by Cesarean section

4:40 - 5:00pm **Journal of Neuroendrocrinilogy: Brief report and Breaking News**
Theater

Presenter: Michael N. Lehman, Editor in Chief, Fundamental and Mechanistic Neuroendrocrinology

5:00pm **Free Time / ATL Activities**

Sunday May 19th

8:30 - 9:15am **Plenary Lecture**
Theater

Chair: Gil Levkowitz - The Weizmann Institute, Israel

Rob Froemke - New York University, USA
Love, Death and Oxytocin

9:15 - 9:30am **Coffee Break**
Rotunda and Crystal Dining Room

9:30 - 11:10am **Symposium 8: Emerging Roles of AVP and OXT on the Neurovascular Unit and Brain Microvessels**
Theater

Chair: Maurice Manning - University of Toledo, USA &
Bice Chini - Milan Center for Neuroscience, Italy

9:30 - 9:55am **Marta Busnelli - Consiglio Nazionale delle Reserche, Italy**
The Oxytocin System Plays a Key Role in Brain Microvascular Development

9:55 - 10:20am **Gil Levkowitz - The Weizmann Institute, Israel**
Oxytocin May Facilitate its Own Peripheral Uptake by Regulating Blood Flow Dynamics

10:20 - 10:45am **Ranjan Roy - Georgia State University, USA**
Vasopressin-mediated Neurovascular Coupling in Health and Disease States

Sunday May 19th

10:45 - 11:10am **Special Talk: Larry Young's Scientific Contributions**

Theater

Arjen Boender - Emory University, USA
Natural Variation in Oxytocin Receptor Signalling Causes Widespread Changes in Neural Gene Expression: A Link to the Natural Killer Gene Complex

11:10 - 11:30am **Coffee Break**

Rotunda and Crystal Dining Room

11:30 - 12:45pm **Symposium 9: Neurohypophysial Hormones and Sensory Processing**

Theater

Chair: Quentin Pittman - University of Calgary, Canada

11:30 - 11:55am **Elizabeth Hammock - Florida State University, USA**
Oxytocin in Sensory-Dependent Social Development

11:55 - 12:20pm **Eric Krause - Georgia State University, USA**
Studying Mechanosensitive Vagal Afferents that Express Oxytocin Receptors: Gut Feelings are Also Matters of the Hear

12:20 - 12:45pm **Michael Perkinson - Otago University, New Zealand**
Unveiling the Dynamics of Oxytocin Activity and Somatodendritic Release in Freely Behaving Rodents

12:45 - 1:30pm **Lunch**

Rotunda and Crystal Dining Room

1:00 - 3:00pm **Poster Session 2**

Magnolia / Sugarberry / Cottonwood

3:00 - 4:40pm **Symposium 10: The Hal Gainer Memorial Symposium: Emerging Areas in the Neurohypophysial Hormones Field**

Theater

Chair: Masha Prager-Khoutorsky - McGill University, Canada

3:00 - 3:25pm **James Blevins - University of Washington and VA Puget Sound Health care System, USA**
Efficacy of Oxytocin as a Monotherapy and Combination Therapy to Treat Obesity

3:25 - 3:50pm **Michael Greenwood - University of Bristol, UK**
Using Quantitative Phosphoproteomics to Explore Hypothalamo-neurohypophysial System Cellular Signalling

Sunday May 19th

3:50 - 4:15pm **Andre Mecawi - Federal University of Sao Paulo, Brasil**
Single-cell Transcriptomics of Hypothalamic Magnocellular Neurons: Unraveling Cellular Diversity, Activity-Associated Genes, and Interspecies Integration

4:15 - 4:40pm **Tian Xue - University of Science and Technology of China, China**
Light Promoted Brain Development: ipRGC, Oxytocin and Synaptogenesis

4:40 - 5:15pm **Coffee Break**
Rotunda and Crystal Dining Room

5:15 - 6:55pm **Symposium 11: Neurohypophysial Hormones Control Social and Defensive Behaviors in a Sex-, Age-, and Receptor-specific Manner**
Theater

Chair: Hala Harony-Nicholas - Icahn School of Medicine, USA &
Joanna Dabrowska - Rosalind Franklin University of Medicine and Science, USA

5:15 - 5:35pm **Alice Sanson - University of Regensburg, Germany**
Neuropeptides Trigger Maternal Care and Aggression in Lactating Rats: Influence of the Stress System

5:35 - 5:55pm **Joanna Dabrowska - Rosalind Franklin University of Medicine and Science, USA**
The Integration of Interoceptive Signals and Defensive Behaviors via Neurohypophysial Hormones in the Bed Nucleus of the Stria Terminalis (BNST)

5:55 - 6:15pm **Aras Petrulis - Georgia State University, USA**
Sex-specific Regulation of Social Motivation by Extrahypothalamic Vasopressin

6:15 - 6:35pm **Brian Trainor - University of California, Davis, USA**
Transcriptional Effects of Social Stress on Oxytocin Neurons in Female California Mice

6:35 - 6:55pm **Samantha Bowden - Michigan State University, USA**
Regulation of Juvenile Social Behaviors by Oxytocin and Vasopressin Systems in the Brain

7:00 - 9:00pm **Award Ceremony and Closing Banquet**
Magnolia

9:00 - 11:00pm **Post-Meeting Party**

Data Blitz

2:00 - 3:00 pm Friday May 17th

1. Revealing the linkage between dehydration and fertility using an RNAseq approach

Sarah Couture - University of New Hampshire, USA

2. Investigating the oxytocin and vasopressin systems in the biological basis of canid pair bonding: establishing coyotes as a model organism

Sara Freeman - Utah State University, USA

3. Variability in expression patterns of the oxytocin receptor in the cerebellum

Ayumu Inutsuka - Jichi Medical University, Japan

4. What causes social fears in autism spectrum disorders?

Freddy Jeanneteau - Inserim IGF, France

5. Maternal *L. reuteri* rescues offspring ASD-like behavior and thyroid hormone target transcripts in oxytocin neurons in an environmental mouse model of autism

Elena Kozlova - University of California Riverside, USA

6. Oxytocin receptor signaling inventromedial hypothalamic (VMH) neurons governs glucose homeostasis via controlling endogenous glucose production by the liver

Franziska Lecchner - Helmholtz Zentrum Munchen, Germany

7. Characterization of oxytocin receptor expressing neurons in the endopiriform nucleus: a novel area for shifting gears from surveillance to novelty

Steffy Manjila - Penn State University, USA

8. Neural circuit remodeling into oxytocin neurons facilitates pregnancy-associated hyperphagia in female mice

Kazunari Miyamichi - RIKEN Center for Biosystems Dynamics Research, Japan

9. Central mechanisms underlying traumatic brain injury-induced hyponatremia in mice

Julie O'Reilly - McGill University Health Centre, Canada

10. V1b vasopressin receptor antagonism ameliorates cardiovascular parameters in ovariectomized female rats subjected to chronic mild unpredictable stress

Noreen Rossi - Wayne State University, USA

11. In vivo determination of the direction of blood flowin the newly discovered SCN-OVLT vascular portal systemin rat

Ranjan Roy - Georgia State University, USA

12. Investigating the role of local acidosis in hypothalamic vasopressin cell excitation in the context of salt-evoked inverse neurovascular coupling responses

Levi Shook - Georgia State University, USA

Data Blitz

2:00 - 3:00 pm Friday May 17th

13. Stress-induced anxiety precipitated by genetic disruption of TSC in oxytocin-responsive cells

Prerana Shrestha - Stony Brook University, USA

14. Chemogenetic activation of oxytocin neurons improves pain in areserpine-induced fibromyalgia rat model

Yoichi Ueta - University of Occupational and Environmental Health, Japan

15. Transcriptional diversity of the oxytocin receptor in the prairie vole

Emma Whelan - University of Virginia, USA

16. Dynamic modulation of pulsatile activities of oxytocin neurons during lactation

Kasane Yaguchi - RIKEN Center for Biosystems Dynamics Research, Japan

17. Dual actions of oxytocin receptor-expressing neurons in the ventromedial hypothalamus in social coping behaviors

Masahide Yoshida - Jichi Medical University, Japan

Poster Session 1

1:10 - 3:00 pm Saturday May 18th

1.1 Optical Control of Oxytocin Signalling

Ahmed I.[1-4], Liu J.4, Gieniec K.[5], Winokur S.[1-4], Bair-Marshall C.[1-4], Adewakun A.[1-4], Stephens J.[1-4], Hetzler B.[6], Arp C.[6], Khatri L.[1], Vanwalleghem vG.[7,8], Seidenberg A.[1-4], Cowin P.[1,9], Trauner D.[6,10-11], Chao M.[1-4,12], Davis F.[5,13-14], Tsien R.[4], Froemke R.[1-4,12]

[1]Department of Cell Biology, New York University Grossman School of Medicine, New York NY USA; [2]Neuroscience Institute, New York University Grossman School of Medicine, New York NY USA; [3]Department of Otolaryngology, New York University Grossman School of Medicine, New York NY USA; [4]Department of Neuroscience and Physiology, New York University Grossman School of Medicine, New York NY USA; [5]EMBL Australia Node in Single Molecule Science, School of Medical Sciences, University of New South Wales Sydney Australia; [6]Department of Chemistry, New York University, New York NY USA; [7]Danish Research Institute of Translational Neuroscience -DANDRITE, Nordic-EMBL Partnership for Molecular Medicine, Aarhus University, Aarhus Denmark; [8]Department of Molecular Biology and Genetics, Aarhus University, Aarhus Denmark; [9]Department of Dermatology, New York University Grossman School of Medicine, New York NY USA; [10]Department of Chemistry, University of Pennsylvania, Philadelphia PA USA; [11]Department of Systems Pharmacology and Translational Therapeutics, Perelman School of Medicine, University of Pennsylvania, Philadelphia PA USA; [12]Center for Neural Science, New York University, New York NY USA; [13]Department of Biomedicine, Aarhus University Aarhus Denmark; [14]Danish Research Institute of Translational Neuroscience, Aarhus University, Aarhus Denmark.

1.2 Deletion of the arginine-vasopressin 1a receptor impairs sexual and maternal behavior in female syrian hamsters

Aspesi D., Stoehr, M., Taylor, J., Greib, Z.A, Huhman, K.L. Albers H.E.

Neuroscience Institute and Center for Behavioral Neuroscience, Georgia State University, Atlanta GA USA.

1.3 Oxytocin, not Vasopressin, mediates High Incidence of MDMA-Induced Hyponatremia - A Complication Preventable by Fluid Restriction

Cihan Atila (MD, PhD)[1,2], Isabelle Straumann (MSc) [2,3], Patrick Vizeli (PhD) [2,3], Julia Beck (MD)[1,2], Sophie Monnerat (MD, PhD)[1,2], Friederike Holze (PhD)[2,3], Prof Matthias E Liechti (MD)[2,3] and Prof Mirjam Christ-Crain (MD, PhD)[1,2]

[1]Department of Endocrinology, Diabetology and Metabolism, University Hospital Basel, Basel, Switzerland; [2]Department of Clinical Research, University of Basel, University Hospital Basel, Basel, Switzerland; [3]Division of Clinical Pharmacology and Toxicology, University Hospital Basel, Basel, Switzerland

1.4 Oxytocin receptor null mutation delays peer social bonding and impacts oxytocin release dynamics in female prairie voles

Black A.[1], Komatsu N.[2], Manoli D.[3], Landry M.[2], Beery AK.[1]

[1]Department of Integrative Biology, University of California Berkeley, Berkeley CA USA, [2]Department of Chemical and Biomolecular Engineering, University of California Berkeley, Berkeley CA USA, [3]Department of Psychiatry and Behavioral Sciences, University of California San Francisco, San Francisco CA USA.

1.5 Consoling disrupted by selective inhibition of oxytocin receptor expressing neurons in the anterior cingulate cortex of prairie voles

Blumenthal S.[1], Horie K.[2], Young L. [1,3]

[1]Center for Translational Social Neuroscience, Emory University, Atlanta GA USA, [2]Department of Molecular and Cell Biology, Graduate School of Agricultural Science, Tohoku University, Sendai, Miyagi, Japan, [3]Department of Psychiatry, Emory University, Atlanta GA USA.

1.6 Impaired oxytocin signaling in the central amygdala in rats with chronic heart failure

Elba Campos Lira[1] Ferdinand Althammer[1,2], Ranjan K. Roy[1,3], Matthew K. Kirchner[1,3] Stephanie Schimmer[4], Alexandre Charlet[5], Valery Grinevich[4], Javier E. Stern[1,3]

[1]Center for Neuroinflammation and Cardiometabolic Diseases, Georgia State University, Atlanta, GA, USA; [2]Institute of Human Genetics, Heidelberg University, Heidelberg; [3] Neuroscience Institute, Georgia State University, Atlanta, GA, USA; [4]Department of Neuropeptide Research in Psychiatry, Central Institute of Mental Health, Heidelberg University, Mannheim, Germany; [5]Centre National de la Recherche Scientifique and University of Strasbourg, Institute of Cellular and Integrative Neuroscience, Strasbourg, France

1.7 Revealing the linkage between dehydration and fertility using an RNAseq approach

Couture, S.[1], Alim Djazouli, FZ.[1&2], Nicholls, S.[1], Hegde, D.[1], O'Brien, C.[1], MacManes, M.[1]

[1]Department of Molecular, Cellular, and Biomedical Sciences, University of New Hampshire, Durham NH USA; [2]Department of Biotechnology and Agroecology, Laboratory of Animal Research and Biobank Sciences, University of Blida1, Blida Algeria.

1.8 Single nucleus rna sequencing reveals heterogeneity of magnocellular neurons in the rat supraoptic nucleus and their response to water deprivation

Victor J. Duque[1], Tays A. Camilo[1], Benjamin Gillard[2], Audrys G. Pauza[2], Michael P. Greenwood[2], David Murphy[2], André S. Mecawi[1]

[1]Laboratory of Molecular Neuroendocrinology, Department of Biophysics, Paulista School of Medicine, Federal University of São Paulo, São Paulo, Brazil; [2]Molecular Neuroendocrinology Research Group, Bristol Medical School: Translational Health Sciences, Dorothy Hodgkin Building, University of Bristol, Bristol, England.

1.9 Crosstalk amongst oxytocin and vasopressin synthesizing neurons of the paraventricular nucleus of the hypothalamus increases blood pressure in mice

Khalid Elsaafien[1,2], Matthew K. Kirchner[1,2], Caitlin Baumer-Harrison[3], Yalun Tan[4], Karen A. Scott[1,2], Javier E. Stern[1,2], Annette D. de Kloet[1,2], Eric G. Krause[1,2]

[1] Neuroscience Institute, [2] Center for Neuroinflammation and Cardiometabolic Diseases, Georgia State University, [3] Department of Psychiatry, University of Pennsylvania, [4]Department of Pharmacodynamics, University of Florida

Poster Session 1

1:10 - 3:00 pm Saturday May 18th

1.10 Spatial analysis of oxytocin receptor mrna expression in the human brain in psychiatric conditions: association with autism and schizophrenia

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1.11 Investigating the oxytocin and vasopressin systems in the biological basis of canid pair bonding: establishing coyotes as a model organism

Freeman S.M.[1], Turano A.[1,2], Young J.K.[3], Catrow J.L.[4]

[1]Department of Biology, Utah State University, Logan UT USA, [2]National Wildlife Research Center, Predator Research Facility, USDA, Logan, UT USA, [3]Department of Wildland Resources and Ecology Center, Utah State University, Logan UT USA, [4]Metabolomics, Proteomics, and Mass Spectrometry Cores, University of Utah, Salt Lake City, UT USA

1.12 The effects of sex and social experience on vasopressin 1a receptor distribution in the social brain of mice

Friesen C.N.[1], Selke A.[1], de Vries G.J.[2], Petrulis A.[1]

[1]Neuroscience Institute, Georgia State University, Atlanta GA USA, [2]Department of Biology, Georgia State University, Atlanta GA USA.

1.13 Sex-dependent increases in peripheral oxytocin levels in response to intravenous kisspeptin in healthy humans

Galbiati F[1,2,3], Aulinas Maso A[2], Plessow F[1], Plummer L[4], Campbell MB[4], Nazarloo H[5], Carter S[5], Carroll RS[3], Kim HK[3], Pereira SA[3], Paulis D[3], Davis J[6], Kaiser UB[3], Seminara S[4], Lawson EA[1]

[1,5] Neuroendocrine Unit, Massachusetts General Hospital and Harvard Medical School, Boston, MA, USA; [2] Department of Endocrinology and Nutrition, Hospital de la Santa Creu i Sant Pau, IR-Sant Pau, CIBERER-Unit 747 (ISCIII), Barcelona, Spain; [3] Division of Endocrinology, Diabetes, and Hypertension, Brigham & Women's Hospital and Harvard Medical School, Boston, MA, USA; [4] Reproductive Endocrine Unit, Massachusetts General Hospital and Harvard Medical School, Boston, MA, USA; [5] Kinsey Institute, Indiana University, Bloomington, IN, USA; [6] Department of Psychiatry, University of Illinois at Chicago, Chicago, IL, USA

1.14 A randomized controlled trial of intranasal oxytocin for obesity

Franziska Plessow, Ph.D.[1,], Liya Kerem, M.D.[1,2,*], Marie-Louis Wronski, M.S.[1,3], Elisa Asanza, M.S.N., M.P.H.[1], Michelle L. O'Donoghue, M.D.[4], Fatima C. Stanford, M.D.[1,5], Kamryn T. Eddy, Ph.D.[6], Tara M. Holmes, M.S., R.D.[7], Madhusmita Misra, M.D., M.P.H.[1,5], Jennifer J. Thomas, Ph.D.[6], Francesca Galbiati, M.D.[1], Maged Muhammed, M.D.[1], Aluma Chovel Sella, M.D.[1,8], Kristine Hauser, C.N.P.[1], Katherine Holman, B.S.[1], Julia Gydus, B.S.[1], Anna Aulinas, M.D., Ph.D.[1,9], Mark Vangel, Ph.D.[10], Brian Healy, Ph.D.[10], Arvin Kheterpal, M.D.[11], Martin Torriani, M.D.[11], Laura M. Holsen, Ph.D.[12], Miriam A. Bredella, M.D.[11], and Elizabeth A. Lawson, M.D., M.M.Sc.[1]*

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Hospital de la Santa Creu i Sant Pau, IIB-Sant Pau, Barcelona, Spain; [10]Biostatistics Center, Massachusetts General Hospital and Harvard Medical School, Boston, MA; [11]Division of Musculoskeletal Imaging and Intervention, Department of Radiology, Massachusetts General Hospital and Harvard Medical School, Boston, MA; [12]Division of Women's Health, Department of Medicine and Department of Psychiatry, Brigham and Women's Hospital and Harvard Medical School, Boston, MA

**Drs. Plessow and Kerem contributed equally.*

1.15 Robust grk2/3/6-dependent desensitization and internalization of oxytocin receptor in neurons

George K.[1], Hoang H.[1], Tibbs T.[1], Nagaraja R.Y.[1], Troyano-Rodriguez E.[1], and Ahmad M.[1]

[1]Department of Cell Biology, University of Oklahoma Health Sciences Center, Oklahoma City, Oklahoma 73104

1.16 Amplifying vasopressin in a sexually differentiated circuit leads to alteration in social and emotional behavior

Sara Guedez Suarez[1], Caitlin Friesin[1], Geert J de Vries[2], and Aras Petrulis[1]

[1]Neuroscience Institute, Georgia State University, Atlanta, GA; [2]Department of Biology, Georgia State University, Atlanta, GA

1.17 Oh-synap! The role of synaptotagmin 11 in osmoregulation

Haan KD.[1], Fisher TE[1]

[1]University of Saskatchewan, Saskatoon, SK Canada

1.18 Behavioral and structural phenotypes of novel vasopressin 1a receptor cre-driver and conditional knockout mouse strains

Hartswick, D.R.[1], Freisen, C.N.[1], Selke, A.[1], Kindler, P.[1], Schappaugh, N[1], Rigney, N.[1], de Vries, G.J.[2], Petrulis, A.[1]

[1]Neuroscience Institute, Georgia State University, Atlanta, GA, [2]Department of Biology, Georgia State University, Atlanta, GA

1.19 Juvenile pleasant sensations facilitate attachment formation with human hands via oxytocin receptors in the rat hypothalamus

Himeka Hayashi[1,2] and Hirotaka Sakamoto[1,2]

[1]Department of Biology, Faculty of Environmental, Life, Natural Science and Technology, Okayama University, Okayama Japan, [2]Ushimado Marine Institute (UMI), Faculty of Environmental, Life, Natural Science and Technology, Okayama University, Okayama Japan.

1.20 Generational influence of parenting

Hinton TD.[1], Maheshwari, T[1]., Perkeybile AM.[1], Connelly JJ.[1]

[1]Department of Psychology, Program of Fundamental Neuroscience, University of Virginia, Charlottesville VA USA

1.21 Exploring plasma copeptin levels in patients with mood disorders with and without suicidal history

Hu, H.[1] Ballard, E.[1] Yuan, P.[1] Johnston, J.[1] Bornhorst, J.[2] Creo, A.[3] Zarate, C.[1] Verbalis, J.[4]

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Poster Session 1

1:10 - 3:00 pm Saturday May 18th

1.22 Variability in expression patterns of the oxytocin receptor in the cerebellum

Inutsuka A.[1], Hattori A.[1], Yoshida M.[1], Takayanagi Y.[1], Onaka T.[1]

[1]Department of Physiology, Jichi Medical University, Shimotsuke JAPAN.

1.23 What causes social fears in autism spectrum disorders?

Dromard Y.[1], Borie A.[1], Chakraborty P.[1], Guillon G.[1], Muscatelli F.[2], Desarmenien M.[1], Jeanneteau F.[1]

[1]Intitut de Génomique fonctionnelle, Department of Neuroscience, University of Montpellier, CNRS, INSERM, France; [2]Intitut des neurosciences de la Méditerranée, University of Aix-Marseille, INSERM, France

1.24 Oxytocin receptor expressing neurons utilize piezo2 to mediate the baroreflex in female mice

Dominique N. Johnson[1,2], Khalid Elsaafien[1,2], Karen A. Scott[1,2], Annette D. de Kloet[1,2], Eric G. Krause[1,2]

[1]Neuroscience Institute, [2]Center for Neuroinflammation and Cardiometabolic Diseases, Georgia State University.

1.25 Vasopressin signaling impacts functional responses in human fungiform taste bud cells

Khosravinezhad D., Iyer S., Gangakhedkar R., Montmayeur J-P., Dotson C.D.

Neuroscience Institute, Georgia State University, Atlanta GA USA

1.26 Compartmentalized Ca^{2+} dynamics shape intrinsic excitability and somatodendritic release in vasopressin neurons

Kirchner M.[1], Stern J.[1]

[1]Center for Neuroinflammation and Cardiometabolic Diseases, Georgia State University, Atlanta GA USA.

1.27 Cytoskeletal regulation of vasopressin neurons in health and salt-dependent hypertension

Kobrinsky S[1], Li B[1], Gu N[1], Grinevich V[2], Prager-Khoutorsky M[1].

[1]Department of Physiology, McGill University, Montreal, QC; Canada, [2]Department of Neuropeptide Research in Psychiatry, University of Heidelberg, Mannheim, Germany.

1.28 Thyroid dependent disruption of oxytocin and gut microbiome in an environmental autism mouse model

Kozlova E. V.[1,2], Denys M. E.[1], Bishay A. E.[1], Campoy L.[1], Habbal A.[1], Luna C.[1], Lam A.[1], Korde Y.[1], Liu R.[3], Do E.[3], Hsiao A.[3] and Curras-Collazo M.C.

[1]University of California Riverside, Riverside, CA; [2]Neuroscience Graduate Program, Riverside, CA; [3]Microbiology & Plant Pathology, University of California Riverside

1.29 Oxytocin signalling in the ventromedial hypothalamus as mechanistic link between social isolation and glucose homeostasis

Lamont H.[1], Routh V.[1], Carcea I.[1]

[1]Rutgers Brain Health Institute, Rutgers University, Newark NJ USA.

1.30 ΔN-TRPV1 regulates cytoskeletal organization and cell morphology in magnocellular vasopressin neurons

Laporte C.[1], Prager-Khoutorsky, M.[1]

[1]Department of Physiology, McGill University, Montreal QC, CA

1.31 Oxytocin receptor signaling in ventromedial hypothalamic (vmh) neurons governs glucose homeostasis via controlling endogenous glucose production by the liver.

Lechner F[1,2,#], Gruber T[2,3,4,#], Wu M.[5], Murat CDB[1,2], Le Thuc O[1,2], Gonzalez-Garcia I[1,2,6], Backes H.[7], Hallschmid M[2,8,9], Wiedemann T.[5], Tschop MH[10], Pospisilik JA[3,4], Garcia-Caceres C[1,2,11]*.*

[1]Institute for Diabetes and Obesity, Helmholtz Diabetes Center, Munich, Germany; [2]German Center for Diabetes Research (DZD), Germany; [3]Department of Metabolism and Nutritional Programming, Van Andel Institute, Grand Rapids, MI, USA; [4]Department of Epigenetics, Van Andel Institute, Grand Rapids, MI, USA; [5]Institute for Diabetes and Cancer, Helmholtz Diabetes Center, Helmholtz Zentrum München, German Research Center for Environmental Health (GmbH), 85764 Neuherberg, Germany; [6]Department of Physiology, CiMUS, University of Santiago de Compostela, Santiago de Compostela, Spain; [7]Multimodal Imaging Group, Max Planck Institute for Metabolism Research, Cologne, Germany; [8]Institute of Medical Psychology and Behavioural Neurobiology, University of Tübingen, Tübingen, Germany; [9]Institute for Diabetes Research and Metabolic Diseases, Helmholtz Centre Munich at the University of Tübingen, Tübingen, Germany; [10]Division of Metabolic Diseases, Department of Medicine, Technische Universität, Munich, Germany; [11]Medizinische Klinik und Poliklinik IV, Klinikum der Universität, Ludwig-Maximilians-Universität München, 80336 Munich, Germany

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1.32 Variation in neuropeptide receptor density across dispersal and by sex in belding's ground squirrels

Lee N.S.[1,2,3], Beery A.K.[3,4], Nunes S.[5]

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1.33 Oxytocin may facilitate its own peripheral uptake by regulating blood flow dynamics

Rajamannar P.[1,2], Raz O.[3], Levkowitz G.[1,2]

Departments of Molecular Cell Biology[1], Molecular Neuroscience[2], and Physics of Complex Systems[3], Weizmann Institute of Science Rehovot, Israel

1.34 Salt-loading reduces central osmoresponsiveness in magnocellular supraoptic neurones *in vivo*

Mike Ludwig, Maja Lozic, Roongrit Klinjampa, Duncan MacGregor and Gareth Leng

Centre for Discovery Brain Sciences, University of Edinburgh, Edinburgh, EH8 9XD, UK

1.35 L-name model of preeclampsia in mice: neuroendocrine, metabolic and locomotor adaptations

Moreira G. M.[1], Marciano N. J. S.[1], Mecawi A. S.[1]

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Poster Session 2

1:00 - 3:00 pm Sunday May 19th

2.1 Maternal *L. reuteri* rescues offspring ASD-like behavior and thyroid hormone target transcripts in oxytocin neurons in an environmental mouse model of autism

Kozlova E. V.[1,2], Denys M. E.[1], Bishay A. E.[1], Piamthai V.[3], Do E.[3], Liu R.[3], Lam A.[1], Luna C.[1], Korde Y.[1], Hsiao A.[3] and Currás-Collazo M.C.

[1]University of California Riverside, Riverside, CA; [2]Neuroscience Graduate Program, Riverside, CA; [3]Microbiology & Plant Pathology, University of California Riverside

2.2 Characterization of oxytocin receptor expressing neurons in the endopiriform nucleus: a novel area for shifting gears from surveillance to novelty

Manjila S.B.[1], Son S.[1], Wu Y-T.[1], Parmaksiz D.[1], Kline H.[1], Betty R.[1], Pi H-J.[1], Minteer J.[1], Browning K.N.[1] and Kim, Y.[1]

[1] Neural and Behavioral Sciences, Penn State College of Medicine, Hershey, PA USA

2.3 Using multivariate classification methods to study the effect of intranasal oxytocin on salience processing of social and non-social stimuli

Mann A.[1], Yang K.[1], Tertikas G.[1], Paloyelis Y.[1], Brodmann K.[1*]*

[1]Institute of Psychiatry, Psychology and Neuroscience, King's College London, London, UK

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2.4 The oxytocin system in craniopharyngioma: a systematic review

Mann A.[1], Kalitsi J.[1,2], Jani K.[3], Martins D.[1], Kapoor RR,[3,4] Paloyelis Y.[1*]*

[1]Institute of Psychiatry, Psychology and Neuroscience, King's College London, London, UK, [2]Florence Nightingale Faculty of Nursing, Midwifery and Palliative Care, Child and Family Health Nursing, King's College London, London, UK,[3]Faculty of Life Sciences and Medicine, King's College London, London, UK, [4]Department of Paediatric Endocrinology, Variety Children's Hospital, King's College Hospital NHS Foundation Trust, London, UK.

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2.5 Effect of oxytocin on hyperalgesia in alcohol-dependent rats

J. Marendes Jr., M.A. Muench, A. Ghaly, B.J. Tunstall

University Of Tennessee Health Science Center (Uthsc), Department Of Pharmacology, Addiction Science, And Toxicology, Memphis, Tn, 38163

2.6 Dichotomic effects of intranasal oxytocin on amygdala's responses during anticipation and perception of pain in women

Martins, Daniel[1,2]; Avetta, Andrea[1]; Fotopoulou, Aikaterini[3]; Paloyelis, Yannis[1]**

[1]Department of Neuroimaging, Institute of Psychiatry, Psychology and Neuroscience (IoPPN) - King's College London (KCL), London, United Kingdom; [2]NIHR Maudsdley Biomedical Research Centre, South London and Maudsley NHS Trust, London, United Kingdom; [3]Department of Clinical, Educational and Health Psychology, Division of Psychology and Language Sciences, UCL.

** Shared joint authors*

Poster Session 2

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2.7 Convergent suppressive effects of intranasal oxytocin on hypothalamic responses during palatable tasting in women with and without compulsive eating

Martins, Daniel[1], Leslie, Monica[1,2]*, Dipasquale, Ottavia[1], Leppanen, Jenni[3], Treasure, Janet[2], Paloyelis, Yannis[1]*

[1]Department of Neuroimaging, Institute of Psychiatry, Psychology and Neuroscience (IoPPN) - King's College London (KCL), London, United Kingdom; [2]Department of Psychological Medicine, Institute of Psychiatry, Psychology and Neuroscience (IoPPN) - King's College London (KCL), London, United Kingdom; [3]School of Psychology, University of Chester, Parkgate Road, Chester, CH1 4BJ, United Kingdom

2.8 The role of oxytocin and vasopressin synthesizing neurons of the paraventricular nucleus of the hypothalamus in stress-induced hypertension

Sá J.[1,2], Eikenberry S.[3], Scott K.[1,2], Krause E.[1,2], de Kloet A.[1,2]

[1] Neuroscience Institute, [2] Center for Neuroinflammation and Cardiometabolic Diseases, Georgia State University, [3] Department of Physiology and Aging, University of Florida

2.9 Tickling of rats does not replicate juvenile social play with regards to the involvement of central oxytocin and vasopressin systems

Tivey E.K.L.[1], Martin J.E.[2], Brown S.M.[1], Bishop V.R.[1], Lawrence A.B.[3], Meddle S.L.[1]

[1]The Roslin Institute, The Royal (Dick) School of Veterinary Studies, The University of Edinburgh, Edinburgh, UK. [2]School of Natural and Environmental Sciences, Newcastle University, Newcastle upon Tyne, UK, [3]Animal and Veterinary Sciences, Scotland's Rural College (SRUC), Edinburgh, UK.

2.10 Neural circuit remodeling into oxytocin neurons facilitates pregnancy-associated hyperphagia in female mice

Kengo Inada[1], Hiroko Yukinaga[1], Mitsue Hagihara[1], and Kazunari Miyamichi[1]

[1] RIKEN Center for Biosystems Dynamics Research, Kobe, Japan

2.11 Central mechanisms underlying traumatic brain injury-induced hyponatremia in mice

O'Reilly J.[1], Bourque C.W.[1]

[1]Center for Research in Neuroscience, Research Institute of the McGill University Health Centre, Montreal QC Canada.

2.12 A dedicated oxytocin signaling in the hypothalamus modulates social avoidance learning in male and female mice

Osakada T.[1], Jiang Y.[1], Yan R.[1], Wei D.[1], Tabuchi R.[1], Dai B.[1], Wang X.[1], Liu J-J.[1], Tsien R. W.[1], Mar A. C.[1], Lin D.[1]

[1]Neuroscience Institute, New York University Langone Medical Center, New York NY USA.

2.13 Transcriptional diversity of the oxytocin receptor in the prairie vole

Page EA.[1], Danoff JS.[2], Perkeybile AM.[1], Connelly JJ.[1]

[1]Department of Psychology, Program of Fundamental Neuroscience, University of Virginia, Charlottesville VA USA; [2]Department of Molecular Biology and Biochemistry, Rutgers University, New Brunswick NJ USA.

2.14 Oxytocin promotes blood-brain barrier formation in a mouse model of autism spectrum disorder and schizophrenia

Paolini C. [1,2], Piacentini F.[2], Busnelli M.[2], Castellani G.[3], Benedetti A.[3], Papaleo F.[3] and Chini B.[1]

[1]CNR Neuroscience Institute, Vedano al Lambro (MB), Italy; [2]University of Milano Bicocca, Vedano al Lambro (MB), Italy; [3]Genetics of Cognition Laboratory, Istituto Italiano di Tecnologia, Genova, Italy.

Poster Session 2

1:00 - 3:00 pm Sunday May 19th

2.15 Quantification of oxytocin neurons in the rodent brain using shield tissue clearing and lightsheet microscopy

Peng S.[1], Patwardhan, A.[1], and Choe KY.[1]

[1]Department of Psychology, Neuroscience, and Behaviour, McMaster University, Hamilton ON CA.

2.16 Oxytocin neuron activity in freely behaving, lactating mice

Perkinson MR, Kim JS, Iremonger KJ, Brown RSE, Brown CH

Brain Health Research Centre, Centre for Neuroendocrinology and Department of Physiology, School of Biomedical Sciences, University of Otago, New Zealand

2.17 Cortical and subcortical oxytocinergic functions may be mediated by distinct release modes of the peptide

Ramos, E.N.[1], Jiron, G.M.[1], Perkeybile, A.M.[1], Carter, C.S.[1], Connelly, J.J.[1], Erisir, A.

[1] Department of Psychology, University of Virginia, Charlottesville VA USA

2.18 Neural connectivity of oxytocin receptor-expressing neurons in the nucleus accumbens and their role in social attachment in prairie voles

Rigney N.[1], Young L.[1]

[1]Department of Psychiatry, Emory University, Atlanta GA USA.

2.19 Grandmotherhood is associated with reduced *oxtr* dna methylation

Rilling J.K.[1,2,3,4,5], Lee M.[6], Zhou C.[6], Gonzalez A.[6], Lindo J.[6]

[1]Department of Psychology, Emory University, Atlanta, GA USA, [2] Department of Psychiatry and Behavioral Sciences, Emory University, Atlanta, GA USA, [3]Center for Behavioral Neuroscience, Emory University, Atlanta, GA USA, [4]Emory National Primate Research Center, Emory University, Atlanta, GA USA, [5]Center for Translational Social Neuroscience, Emory University, Atlanta, GA USA, [6]Department of Anthropology, Emory University, Atlanta GA USA.

2.20 V_{1B} vasopressin receptor antagonism ameliorates cardiovascular parameters in ovariectomized female rats subjected to chronic mild unpredictable stress

Noreen F. Rossi and Dragana Komnenov

Dept. of Physiology, Wayne State University School of Medicine, Detroit, MI USA

2.21 In vivo determination of the direction of blood flow in the newly discovered scn-ovlt vascular portal system in rat

Ranjan K Roy[1]; Yifan Yao[2]; Rae Silver[2,3,4,5]; Javier E. Stern[1]

[1]Neuroscience Institute, Georgia State University Atlanta GA 30303; [2]Department of Psychology, Columbia University; [3]Department of Neuroscience and Behavior, Barnard College; [4]Graduate Teaching Faculty, Department of Pathology and Cell Biology, Columbia University Irving Medical Center; [5]Zukerman Institute Affiliate, Columbia University

2.22 Oxytocin system activity in the nucleus accumbens shell drives maternal aggression and is altered by local crf infusion

Sanson A.[1], Demarchi L.[1], Rocaboy E.[1], Bosch O.J.[1]

[1] Department of Behavioural and Molecular Neurobiology, Regensburg Center of Neuroscience, University of Regensburg, Regensburg, Germany

Poster Session 2

1:00 - 3:00 pm Sunday May 19th

2.23 Properties of V1aR-expressing cells within the social decision-making neural network

Schappaugh N.[1], Zaw A.[1], de Vries G.[2], Petrulis A.[1]

[1]Neuroscience Institute, Georgia State University, Atlanta, GA, [2]Department of Biology, Georgia State University, Atlanta, GA

2.24 Chronic activation of vagal sensory neurons that express oxytocin receptors decreases food intake and body weight in male mice

Scott K.[1,2], Johnson D.[1,2], Elsaafien K.[1,2], de Sá, J.[1,2], de Lartigue G.[3], de Kloet A.[1,2], Krause E.[1,2]

[1]Neuroscience Institute, [2]Center for Neuroinflammation and Cardiometabolic Diseases, Georgia State University, Atlanta GA USA, [3]Monell Chemical Senses Center, Philadelphia PA USA

2.25 Reduced stimulation of oxytocin neurons in zebrafish following complete social isolation

Shams S.[1], Zentveld, L.[2], Westberg L.[2]

[1]Department of Biochemistry & Molecular Biology, Mayo Clinic, Rochester MN USA, [2]Department of Pharmacology, Institute of Neuroscience & Physiology, Gothenburg University, Gothenburg, Sweden

2.26 Effects of chronic intranasal oxytocin on visual attention to faces vs natural scenes in older adults

Shoenfelt A.[1], Pehlivanoglu D.[1], Lin T.[1], Ziaei M.[2,3], Feifel D.[4], Ebner N.C.[1,5,6]

[1]Department of Psychology, University of Florida, Gainesville, FL, USA; [2]Kavli Institute for Systems Neuroscience, Norwegian University of Science and Technology, 7030 Trondheim, Norway; [3]K.G. Jebsen Centre for Alzheimer's disease, Norwegian University of Science and Technology, 7030 Trondheim, Norway; [4]Department of Psychiatry, University of California, San Diego, CA, USA; [5]Institute on Aging, University of Florida, Gainesville, FL, USA ; [6]Center for Cognitive Aging and Memory, University of Florida, Gainesville, FL, USA

2.27 Investigating the role of local acidosis in hypothalamic vasopressin cell excitation in the context of salt-evoked inverse neurovascular coupling responses

Levi Shook[1,2], Javier E. Stern[1,2]

[1] Neuroscience Institute, Georgia State University, Atlanta GA; [2] Center for Neuroinflammation and Cardiometabolic Diseases, Georgia State University, Atlanta GA

2.28 Stress-induced anxiety precipitated by genetic disruption of tsc in oxytocin-responsive cells

Olivia Tabaka[1], Saheed Lawal[1], Alex Fraser[1], Rodrigo D Triana[2], Karen S Ruiz[2], Mian Hou[2], Matthew Dickinson[1], Maggie Marmarcz[2], Eric Klann[2], Prerana Shrestha[1]

[1]Stony Brook University, Neurobiology & Behavior, Stony Brook, NY; [2]New York University, Center for Neural Science, New York, NY

2.29 Chemogenetic activation of oxytocin neurons improves pain in a reserpine-induced fibromyalgia rat model

Ikeda N.[1], Kawasaki M.[1], Baba K.[1], Nishimura H.[1], Suzuki H.[1], Ohnishi H.[1], Maruyama T.[2], Sakai A.[1], Ueta Y.[2]

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Poster Session 2

1:00 - 3:00 pm Sunday May 19th

2.30 The role of oxytocin in the development of social preference in zebrafish

Westberg L., Nabinger D.D., Hassan N., Eken F., Blide M., Cronell P., Zentveld L., Shams S.

Department of Pharmacology, Institute of Neuroscience & Physiology, University of Gothenburg, Gothenburg, Sweden.

2.31 Unravelling the interplay between stress, inflammation, and methylation of the oxytocin receptor gene in aging

Wright K.[1], Polk R.[1], Lin T.[1], Kroll K.[2], Perkeybile A.[2], Connelly J.[2], & Ebner N. C.[1]

[1]Department of Psychology, University of Florida, Gainesville FL USA, [2]Department of Psychology University of Virginia, Charlottesville VA USA

2.32 Central mechanisms underlying MDMA induced hyponatremia in rats

Wyrosdic, J.C.[1], Thirouin, Z.S.[1], Bourque, C.W.[1]

[1]Brain Repair and Integrative Neuroscience Program, Research Institute of the McGill University Health Centre, Montreal General Hospital, 1650 Cedar Avenue, Montreal, QC H3G1A4, Canada.

2.33 Dynamic modulation of pulsatile activities of oxytocin neurons during lactation

Kasane Yaguchi[1,2], Gen-ichi Tasaka[1], and Kazunari Miyamichi[1]

[1] Laboratory for Comparative Connectomics, RIKEN Center for Biosystems Dynamics Research, Kobe Japan, [2] Graduate School of Biostudies, Kyoto University, Kyoto Japan

2.34 Dual actions of oxytocin receptor-expressing neurons in the ventromedial hypothalamus in social coping behaviors

Yoshida M., Nasanbuyan N., Inutsuka A., Takayanagi Y., Onaka T.

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2.35 Competing vasopressinergic projections to lateral hypothalamic hypocretin cells

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2.36 Oxytocin receptor genetic variation influences the social vigilance of female prairie voles

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Abstracts

Plenary Talks

Plenary 1

Portal pathways in the brain: Their potential for transporting neurovascular peptidergic signals

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Background: Brain regions communicate via both wiring (neural) and volume (diffusible) transmission. Portal systems, an example of volume transmission, are specialized vascular arrangements in which capillary beds of two distinct regions are joined via connecting veins (Dorland 2020 ISBN9781455756438) enabling transport of neurosecretions in high concentrations from one capillary bed to another without dilution in the systemic circulation. Almost 100 years after the discovery of the pituitary portal system (Popa & Fielding 1930 PMID17104309), we discovered a second portal system linking the suprachiasmatic nucleus (SCN) and the organum vasculosum lamina terminalis (OVLT), a sensory circumventricular organ (CVO) (Yao et al. 2021 PMID34561434). **Questions addressed**: As a first step in exploring the direction of signaling and function(s) of this system, we use in vivo two-photon microscopy. We also explore the role of the SCN and circadian timing by examining hemodynamics of the SCN-OVLT portal pathway. To assess how arginine vasopressin, a diffusible output signal of the SCN, reaches its targets, we performed detailed analyses of SCN capillary vasculature (Yao et al., 2023 PMID 37553858) and its innervation. Next we address the broader question of whether the capillary beds of the remaining sensory CVOs, namely the subfornical organ and area postrema, are directly connected to the capillary beds of the neural tissue to which they are attached. To preserve the integrity of the vasculature under study, we performed iDISCO clearing, lightsheet microscopy and novel brain registering tools for volumetric images of blood vessels in CVOs and adjacent neural tissues. **Implications of the findings**: As did the discovery of the pituitary portal system, our findings are poised to revise the calculus on how small populations of neurons relay their secretions to local targets in the brain. We surmise that the existence of new portal pathways in the brain promises to expand the broad field of Neuroendocrinology by the finding of new, extrasynaptic routes for neural secretions to reach their local parenchymal targets.

Plenary 2 - Mortyn Jones Lecture

Osmotic control of vasopressin: What have we learned since Verney and Andersson?

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The pioneering works of EB Verney and B Andersson provided early indication that secretion of vasopressin (VP) in mammals wasunder the control of osmoreceptors and sodium detectors located in the anterior hypothalamus. In the decades that followedmultiple groups greatly advanced our understanding of the anatomical location, molecular identitiesand functional rolesof these mysterious interoceptive sensors. We now know that a significant amount of redundancy exists in the body's ability to measure ambientlevels of extracellular sodium and total solute concentration (osmolality), both in terms of cellular/molecular mechanisms and anatomical distribution. We also know that the body can anticipatefutureperturbations in sodium/osmolality based on inputs from the circadian clock and inputs from the upper alimentary tracts which inform the brain about changes related to the time of day and ingestive behavior.Over 40 years ago, studies performed by Bill Mason, Gareth Leng and their colleagues provided evidencethat the magnocellular neurosecretory cells (MNCs) that secrete vasopressin and oxytocin are intrinsically sensitive to osmotic perturbations and that this unusual phenotype might contribute to the regulation of these

neurons under certain conditions, a point that has been debated to a significant extent at early editions of the Word Congress on Neurohypophysial Hormones (WCNH). In this lecture, Dr. Bourque will provide an update on what is known about the cell-autonomous sensory properties of MNCs and describe recent evidence showing that these intrinsic properties can play a functional role under physiological conditions in vivo.

Plenary 3

Metabolic and stress-coping actions of oxytocin

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Oxytocin has multiple functions including metabolic and stress-coping actions.

When foods are consumed or CCK is administered, oxytocin neurons in the hypothalamus are activated in a manner dependent on prolactin releasing peptide neurons in the medulla oblongata via afferent signals of the vagus nerve. Endogenous oxytocin has been suggested to induce satiety. Oxytocin receptor-deficient mice showed increased meal size. Oxytocin has also been found to regulate energy consumption. Oxytocin increases body temperature through serotonin neurons, which activate the sympathetic nervous system. Oxytocin receptor-deficient mice exhibited a decrease in oxygen consumption during stressful situations, which led to an increase in body weight. Furthermore, oxytocin plays a role in macronutrient preference including the preference for sucrose. The intake of sucrose leads to the release of FGF21, a hepatocytokine, from the liver. The administration of FGF21 activated oxytocin neurons in the hypothalamus, which in turn reduced sucrose intake. These findings suggest that oxytocin systems control energy homeostasis by reducing food intake and increasing energy consumption, particularly in stressful situations.

Various stressful stimuli, as well as food intake, activate oxytocin neurons. Studies with oxytocin receptor-deficient mice showed that they exhibit deficits in stress-coping behavior during social stress. These animals exhibited a decrease in allo-grooming behavior towards their defeated cage-mates and displayed a reduced social defeat posture towards dominant conspecifics to indicate their inferiority. These stress-coping behaviors have been found to be induced by distinct oxytocin-oxytocin receptor systems. On the other hand, overactivation of the neurons expressing the oxytocin receptor in the ventromedial hypothalamus induced the generalization of social fear memory, resulting in general social avoidance. This suggests the importance of an appropriate degree of activation of these neurons. Furthermore, the activation of oxytocin systems during development affected energy metabolism and social behavior in adulthood, suggesting developmental actions of oxytocin.

Plenary 4

Love, Death, And Oxytocin

Froemke, R.C.

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The neuropeptide oxytocin is important for maternal physiology and social behavior. In this talk, I will discuss new and unpublished data from our lab on when, where, and how oxytocin is released from hypothalamic neurons to enable maternal behavior in new mother mice. I will focus on maternal responses to infant distress calls, and how oxytocin enables rapid neurobehavioral changes for dams and alloparents to recognize the meaning of these calls. We have built a new system combining 24/7 continuous video monitoring with neural recordings from the auditory cortex and oxytocin neurons of the hypothalamus in vivo. With this documentary approach, we have identified behaviors of experienced and naïve adults learning to co-parent together which also activate oxytocin neurons. I will discuss circuits routing sensory information to oxytocin neurons leading to oxytocin release in target areas important for maternal motivation. Finally, I will discuss longer-term behavioral monitoring over months, examining how single mothers build nests to help ensure pup survival or how this sometimes goes awry.

Symposium 1 Abstracts

Astrocytes in mice central amygdala mediates oxytocin-dependent behavioral adaptation

Alexandre Charlet

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Our daily life is a succession of cognitive actions influenced by our emotions. Emotions, often referred to as feelings, are necessary to maintain a balance throughout the life of an individual, if not his survival: love, anger, pain and fear are the most common examples. While neuronal networks sustaining emotions are well studied, a tremendous question persists: how does our brain cellular networks supports the enduring effects of emotions?

Considerable evidence support a role for neuropeptides in the control of emotions in mammals. Oxytocin, a nonapeptide mainly synthesized in the hypothalamus, recently became a centre of attention for the regulation of affective behaviours. Particularly, oxytocin-induced fear and anxiety regulations take place within the amygdala, a key nucleus in processing of emotions in both physiological and pathological conditions. Despite these advances, we still lack evidence on how neuropeptidergic systems support the plasticity of emotions processing throughout a lifetime. Interestingly, alterations in glial cells might be linked to emotional impairments and adaptation, particularly within the amygdala brain region. While major studies demonstrated over the last years a role for astrocytes in functional cellular networks modulation, illustrated in memory and anxiety processing, the contribution of astrocytes in the neuromodulatory effects of a neuropeptide has rarely been explored. We recently demonstrated that a subpopulation of the central amygdala astrocytes express the G-protein coupled oxytocin receptor, and thus that oxytocin can tune astro-neuronal networks activity to control amygdala-related behaviour.

This work paves the way to our ongoing projects, for which I hypothesize that astrocytes and neurons fulfill synergistic functions achieved at differential time points to support behavioral adaptation.

Sex-based differences in control of neurohypophysial hormones in a model of hyponatremia

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Hyponatremia, the dilution of plasma sodium by water retention due to increased vasopressin (AVP) release, increases the mortality of patients with chronic diseases such as congestive heart failure and cirrhosis . However, the cause of increased AVP in these diseases is unknown. Our lab has used bile duct ligation (BDL) to induce cirrhosis and study neural mechanisms contributing to increased AVP release and decreased plasma osmolality. Our studies indicate that the activation of projections from the A1 and A2 regions of the brainstem to the supraoptic nucleus (SON) contributes to the increase in plasma AVP in male BDL rats. Changes in chloride transporter activity that reduce or reverse the inhibitory effects of GABA are also observed in AVP neurons from male BDL rats. Female BDL rats, in contrast, do not demonstrate increased plasma AVP or significant changes in plasma osmolality. Instead, females show increased circulating oxytocin (OXY). Preliminary studies suggest that this sex difference may be estrogen-dependent. Spatial transcriptomics (Visium, 10x genomics) has also been used to explore possible sex-based differences in the SON that may contribute to the response to BDL. A DESeq2 analysis of SON transcripts comparing male BDL rats to controls identified 195 differentially expressed genes (DEGs) that gene ontology analysis indicated are related to membrane transport and synapse function. A similar analysis comparing the SON of BDL females to control females identified 139 DEGs related to microglia proliferation and vesicle membrane regulation. These data suggest the central plasticity contributing to the increased release of AVP and hyponatremia in male rats in response to BDL appears absent in female rats, and this may be related to the differential regulation of OXY, microglia, and vesicle regulation.

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2.Schrier RW. Water and sodium retention in edematous disorders: role of vasopressin and aldosterone. Am J Med. 2006;119:S47-53. doi: 10.1016/j.amjmed.2006.05.007

Symposium 1 Abstracts

Exploring the connections between oxytocin-mediated lactation and emotion control using deep learning-based tools

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Sucking-induced milk ejection is a classical neuroendocrine reflex, which relies on the burst firing of hypothalamic oxytocin neurons and pulsatile release of oxytocin. It is well-known that breastfeedinginduces a number of beneficial psychological effects to the mother, and lactationis readily affected by the emotional states of the mother. However, these studies were primarily based on epidemiological analyses in human with correlative connections. Lactation-related animal studies were predominantly carried out in anesthetic states, precluding the accurate assessment of emotional changes. By carefully analysing mother-pup interactions, recording hypothalamic oxytocin neuronal activity of awake lactating rats, and real-time monitoring intramammary pressure, we extracted milk ejection-related characteristic behavioral patternsof mother and pups. Using these features, we developed a deep learning framework to quantify milk ejections based on videos of lactating rats in natural environment. We further leveraged the framework to discern the emotional states when mother rats were under different stress conditions, which revealed important phenotypic and functional connections between milk ejections and emotions.

Salt-loading reduces central osmoresponsiveness in magnocellular supraoptic neurones in vivo

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Magnocellular vasopressin and oxytocin neurones in the supraoptic nucleus (SON) are directly osmosensitive and are modulated by osmotic pressure. High salt intake increases the spontaneous firing rate of vasopressin neurones in vitro and in vivo. Here we investigate the neural plasticity of vasopressin and oxytocin neurones in the SON after salt-loading, in response to additional acute osmotic and hypovolemic challenges, using extracellular single cell recordings in urethane-anaesthetised adult male Sprague-Dawley rats.

The results show that prolonged salt-loading (drinking of 2% NaCl solution for 7 days) leads to the sustained activation of the vasopressin and oxytocin neurones in the SON by increasing their basal firing activity. Also, a proportion of vasopressin neurones switch from a continuous to a phasic firing pattern in the salt-loaded group. Pituitary vasopressin stores were significantly reduced, and plasma sodium levels were significantly higher in salt-loaded than in euhydrated rats.

Moreover, the response to an additional acute hyperosmotic challenge was significantly reduced in salt-loaded rats (1.89 spikes/s) compared to euhydrated rats (2.96 spikes/s). Under salt-loading conditions in vivo, the GABA-mediated inhibitory signal to vasopressin neurones arising from the brainstem is maintained, with no significant differences in the response to intravenous injection of cholecystokinin (CCK) or. the α-1 adrenergic agonist phenylephrine.

Salt-loading induced hypovolaemia, and to investigate whether hypovolaemia reduces the response to acute osmotic stimulation, we induced an acute hypovolaemic condition by the intraperitoneal injection of polyethylene glycol (PEG) solution. The electrophysiology recordings showed that injection of PEG leads to the sustained activation of the vasopressin and oxytocin neurones, gradually increasing their electrical activity over a 75-min period after administration, but there were no significant differences between the responses of euhydrated, salt-loaded and rehydrated rats.

These results indicate that salt-loading induces significant changes in the neural properties of SON neurones and reduces their responsiveness to acute osmotic challenges. Since maximal antidiuretic effects of vasopressin are achieved at a low circulating concentration of about 10 pg/ml, it seems likely that once a level of electrical activity is reached that achieves this level of secretion, any further increase would give no benefit for water retention but would hasten depletion of the neurohypophysial stores.

Symposium 2 Abstracts

Longitudinal imaging of AVP neuronal behavior *in vivo*

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A comprehensive understanding of neural circuits requires cell-resolved and cell-type-specific observation of networks and their components within the context of receiving inputs and generating relevant outputs. Here we describe in vivo, longitudinal deep-brain Ca2+ imaging of large groups of AVP+ and NMS+ neurons in awake behaving mice. We observe single-cell Ca2+ waves indicative of burst firing across the day and night, stochastic circadian rhythms in single-cell parameters, rhythms in network dynamics, and, with some cell types, robust and diverse responses to sensory input. We demonstrate that such recordings are achievable in deep brain regions including suprachiasmatic and supraoptic nuclei, and may provide new insights into context-specific and longitudinal behavior of neurohormone-producing brain cells and circuits.

Development and optimization of genetically encoded sensors for oxytocin and arginine vasopressin detection

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Oxytocin (OT) and Arginine vasopressin (AVP) are two neuropeptides that play important roles in both the peripheral system and the central nervous system. To fully understand their functions, it is critical to be able to monitor OT and AVP dynamics in vivo with high selectivity, sensitivity and good spatiotemporal resolution. To achieve this, we have developed genetically encoded sensors, $GRAB_{OT1.0}$ and $GRAB_{AVP0.2}$, for OT and AVP respectively based on the GRAB (G protein-coupled receptor activation–based) strategy. The sensors can couple the conformational change of GPCR upon OT/AVP binding to the fluorescence increase of the fluorescence module, cpEGFP.

Further optimization of the sensors' cpEGFP, GPCR backbone and linker peptide resulted in a new generation of OT and AVP sensors with higher signal-to-noise ratios (SNR). Moreover, the new OT sensor shows higher selectivity for OT compared to AVP despite their similar structures. These sensors can detect the respect peptides with fiber photometry recording upon hypertonic stimulation in freely moving mice with good temporal resolution. Additionally, we have developed and optimized red-shifted OT and AVP sensors to enable multiplex-imaging. These tools will greatly contribute to our understanding of OT/AVP functions in both physiological and pathophysiological conditions.

A novel transgenic rat to tackle the behavioural roles of magnocellular vasopressin neurons

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Arginine vasopressin (AVP) is a neuropeptide widely expressed in the brain, but its largest extent produced by magnocellular neurons of the hypothalamus. AVP originating from magnocellular neurons exerts well-known hormonal roles such as water retention in the kidney, but in the brain it promotes a variety of behaviours including anxyogenic and aggressive behaviours. However, it remains unclear whether magnocellular AVP neurons of the hypothalamus are involved in driving these or other behaviours. To tackle this question, we generated a novel transgenic AVP-IRES2-Cre knockin Sprague-Dawley rat, using CRISPR/Cas9-mediated targeted insertion of IRES2-Cre into the 3' untranslated region of the AVP gene. This rat allows us to dissect, manipulate, and image specific AVP circuits in the brain using viral vectors, primary focusing on magnocellular AVP neurons of the

hypothalamus. Up to now we mapped input and output connectivity of magnocellular AVP neurons, performed their ex vivo patch-clamp electrophysiology as well as monitored their activity during osmotic challenge. Our ongoing studies are focused on functional validation of selected extrahypothalamic projections of magnocellular AVP neurons aiming to reveal their involvement in the modulation of socio-emotional behaviours.

Marmoset monkeys as a model of OT action in primates

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Marmosets have emerged as a powerful neurobiological model of primate brain function, particularly for facets of social brain functions. This New World primate exhibits pair-bonding, alloparenting, and engages in a suite of prosocial behaviors including food-sharing, imitation, and social gaze. Moreover, these monkeys are highly voluble, frequently engaging in bouts of vocal exchanges. Altogether, this makes them an optimal model to study the effects of OT on primates' social behaviors. This axis of research is important because a lot of findings from rodent literature have failed to translate to humans.

After presenting the advantages and disadvantages of marmoset monkeys for OT research, I will show the results of a first study in which I mapped OT fibers throughout their brains using immunohistochemistry. Critically, the OT connectome in marmosets closely mirrors that of rodents. I will also describe the development of an adeno-associated virus (AAV) vector for the activation or inhibition of OT neurons in primates. Finally, behavioral and electrophysiological data obtained from freely moving marmosets within the colony room will be presented, showcasing the utility of a semi-naturalistic paradigm for elucidating the neural underpinnings of complex social behaviors.

Combined, these findings and tools will help to compare the effects of OT on rodents and primates more directly. Furthermore, my preliminary results demonstrate that the rich vocal behavior of marmosets make them an ideal model to study the neural bases of acoustic communication and its modulation by OT.

Symposium 3 Abstracts

Are patients with vasopressin deficiency also deficient in oxytocin?

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Arginine vasopressin deficiency (AVP-D), known as central diabetes insipidus (cDI), is a rare neuroendocrine condition clinically characterized by polyuria and polydipsia. Despite treatment with desmopressin (vasopressin receptor 2 agonist), patients often report residual psychological symptoms such as heightened anxiety levels, difficulties describing or expressing emotions, and depressed mood. Due to the anatomical proximity, disruptions leading to AVP-D could also affect oxytocin-producing neurons. The central oxytocinergic system promotes pro-social effects such as in-group favoritism and protection against social threats, trust, attachment, and empathy, and is involved in emotion recognition. An additional oxytocin deficiency in patients with AVP-D could explain – at least partially – the observed socio-emotional changes. For other pituitary hormones, a provocation test to stimulate the respective hormone is often applied in case of a suspected deficiency. Still, no standard provocation test for oxytocin has been established, and testing attempts or physiological stimuli (e.g., exercise) have failed to reveal a consistently strong increase in oxytocin levels.

3,4-Methylenedioxymethamphetamine (MDMA, 'ecstasy') is used recreationally for its effects on empathic feelings and sociability. Several studies have documented marked increases in circulating oxytocin levels in response to MDMA. Importantly, recent data from animal and human studies demonstrated a selective effect on oxytocin without changing AVP (or copeptin) levels in response to

MDMA. By stimulating oxytocinergic neurons, MDMA induces not only an increase in peripheral levels but also central OXT-mediated behavioral effects. The prosocial effects of MDMA on emotion processing and social interaction, such as increased trust, closeness to others, identification of facial emotions, and fear extinction, are mediated partly by a strong oxytocin release.

Using MDMA ('ecstasy') as a psychoactive and biochemical provocation test, our findings indicate a clinically relevant oxytocin deficiency in patients with AVP-D. In healthy controls, we observed the expected increase in plasma oxytocin in response to MDMA stimulation with typical pro-social, empathic, and anxiolytic effects. In contrast, in patients with AVP-D, we found no notable increase in plasma oxytocin and, in agreement, lower MDMA-induced subjective effects, reflecting the lack of activation of central key regions important for socio-emotional processing. Furthermore, these findings contribute to deepening our understanding of oxytocin as a key hormone in centrally generated socio-emotional effects reflected by reduced pro-social, empathic, and anxiolytic effects in patients with an oxytocin deficiency.

Exploring signaling amongst neurohypophyseal hormones: a complex discourse that'll elevate your blood pressure.

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The paraventricular nucleus of the hypothalamus (PVH) is a major integrative brain center that contains several structurally and functionally distinct populations of neurons. Many of these PVH neurons are involved in blood pressure regulation, and in order to coordinate their actions, they interact with each other, at least in part, by way of paracrine (neuropeptide-mediated) signaling. Here, we test the hypothesis that PVH neurons that synthesize oxytocin, in particular, influence the neuroendocrine and/or autonomic regulation of blood pressure by way of such a paracrine signal. Experiments combine the Cre-LoxP system in mice, in vivo and in vitro optogenetics, Ca^{2+} imaging, pharmacology and various neuroanatomical and whole animal physiological techniques to test this hypothesis. The collective overall implications of the findings that will be presented are that robust firing of oxytocinergic PVH neurons spurs local release of oxytocin; and that this local oxytocin acts as a paracrine signal to increase blood pressure by way of elevating circulating neurohypophyseal hormones and activating vasopressin 1a receptors. Thus, these studies unravel a novel mechanism by which the oxytocinergic PVH neurons influence blood pressure.

Oxytocin receptor co-localization in brainstem parasympathetic cardiac vagal neurons

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Surprisingly little is known about the expression of oxytocin (OXT) receptors in the nucleus ambiguus (NA) and dorsal motor nucleus of the vagus (DMNX), and in particular whether some, all, or none of the parasympathetic cardiac vagal neurons (CVNs) within these different nuclei express OXT receptors. In this study we characterized the co-localization of OXT receptors in CVNs, and non-CVN cholinergic neurons located in the NA and DMNX nuclei. The transgenic oxytocin receptor (OXTR)-Cre mouse JAX #031303 and the Cre dependent floxed ChR2-eYFP mouse JAX #012569 were crossbred for this study. Alexa Fluor 555 conjugates of cholera toxin subunit B was injected into the pericardial sac to retrogradely label CVNs. Colocalization analysis was performed using an Imaris algorithm for co-expression overlapped cells and the percentage of colocalization was calculated for each cell population. We found that over half of the CVNs in the DMNX co-localize with OXT receptor positive neurons. Surprisingly, CVNs in the NA, as well as the other ChAT neurons in the NA, have sparse co-localization with OXT receptor positive neurons. To selective excite OXTR+ CVNs in the DMNX we use a retrograde Cre dependent site-specific recombinase FlpO virus (addgene AAV 87306) that is injected into the pericardial sac. 4 weeks later a Flp-dependent DREADDs virus (Addgene AAV 154868 -pAAV-hSyn-fDIO-hM3D(Gq)-mCherry) is injected into the DMNX. Selective chemogenetic excitation of OXTR+ CVNs in the DMNX

significantly decrease heart rate. Future work is needed to test if selective activation of OXT receptor positive CVNs in the DMNX prevents or reverses autonomic dysfunction in cardiorespiratory diseases.

Oxytocinergic control circuits in the spinal cord for male sexual behavior

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The oxytocin (OXT)-ergic mechanisms controlling socio-sexual behaviors comprise intricate neuronal circuitry systems in widely distributed regions of the brain and spinal cord. At the organismal level, it is increasingly evident that hormonal regulations are pivotal alongside neuronal circuit activation. The gastrin-releasing peptide (GRP) system in the lumbosacral spinal cord is a crucial component of the neural circuits regulating penile reflexes in rats, commonly known as the "spinal ejaculation generator (SEG)" (1). OXT, conventionally recognized as a neurohypophysial hormone, is now known for its role in regulating socio-sexual behaviors in mammals, ranging from social bonding to empathy. Nonetheless, the functional interplay between GRP neurons and the hypothalamo-spinal oxytocin system remains unclear. OXT is primarily synthesized in hypothalamic neurons and released from the posterior pituitary into circulation. Additionally, it is released from neuronal dendrites into the hypothalamus, where it exerts crucial functions in social behaviors through non-synaptic volume transmission. Although OXT is well-known functions primarily revolve around regulating female reproductive functions such as parturition, milk ejection, and maternal behavior, evidence suggests that a subset of parvocellular OXT neurons project to the lower spinal cord, influencing male sexual function in rats. Recently, we have shown the functional interaction between GRP neurons and the hypothalamo-spinal oxytocin system, as well as the effects of these neuropeptides on male sexual behavior (2). The spinal GRP neurons expressed OXT receptors and were activated by OXT during male sexual behavior. Further, intrathecal administration of OXT receptor antagonist not only attenuated ejaculation but also affected pre-ejaculatory behavior during normal sexual activity. These results indicated that OXT influences male sexual activity via the spinal GRP neurons. Furthermore, we demonstrate the recently identified localized volume transmission role of OXT in the spinal cord. Insights from our studies suggest that the newly discovered "OXT-mediated spinal control of male sexual function" may be useful in the treatment of erectile and ejaculatory dysfunction (3).

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Symposium 4 Abstracts

Biological function and pharmacological potential of oxytocin signalling in ants

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The oxytocin and vasopressin signalling system, with its origin dating back over 600 million years, has been identified in various invertebrate phyla, including molluscs, annelids, nematodes, and insects. Despite its widespread presence across insect orders, these peptides and their receptors remain elusive in common insect models like flies and bees. Consequently, our understanding of the biological roles of this G protein-coupled receptor (GPCR)-mediated signalling system in insects, the largest animal group, is limited.

This study focuses on elucidating the biological role of oxytocin-like neuropeptides in insects, with a specific emphasis on ants. Utilizing a diverse set of methods such as transcriptome mining, pharmacological and genetic manipulations, in situ imaging, and behavioural observations, we present

a comprehensive overview into the proposed biological functions of the oxytocin-like signalling system in ants. Moreover, this approach involves the development of a workflow combining high-resolution mass spectrometry imaging and microtomography for multiplexed mapping of neuropeptides in ant brains.

Intriguingly, chemical functionalization of ant peptides has led to the optimization of pharmacological probes for human oxytocin/vasopressin receptors. By exploring additional ant signalling components (G proteins and arrestins) and employing comparative pharmacology, we aim to gain new insights into the molecular pharmacological of oxytocin and vasopressin receptors.

In conclusion, ants emerge as a compelling model system for translational science in humans. Investigating the functional roles and spatial distribution of neuropeptides, like oxytocin not only contributes to our understanding of fundamental neurobiological processes but also guides the structure-function analysis of human GPCR signalling.

Evolutionary conserved role of oxytocin on the regulation of social behavior in zebrafish

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Social interactions play a major a major role in different functional domains relevant for Darwinian fitness, such as finding food, choosing mates, or avoiding predators. Therefore, at the proximate level social interactions are a key mortality risk factor with health implications and at the ultimate level, sociality impacts ecological and evolutionary processes. Our lab studies social behavior at both levels, combining the study of proximate causes (gene modules, hormones, neural circuits, cognitive processes) and ultimate effects (evolutionary consequences). For this purpose, we have been using zebrafish as a model organism. In this talk I will first show how oxytocin plays a critical role in the development of sociality in zebrafish and how it interacts with the developmental environment to shape the emergence of different components (social affiliation, social recognition) of adult social behavior. I will then, show how oxytocin is also necessary and sufficient for the expression of complex social behavior in adult zebrafish, including social contagion of fear and emotion recognition. Together these results support a deep homology role of oxytocin in the regulation of social behavior across vertebrates, despite the engagement of different neural circuits across taxa.

Making mothers: Pregnancy, birth, and epigenetic regulation of the maternal oxytocin receptor gene

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Nearly one in three births in the United States occurs after a labor induced with exogenous oxytocin, yet little is known about the long-term consequences of this birth intervention for maternal health. Research indicates a link between labor induction and increased risk for postpartum hemorrhage, the leading cause of maternal mortality worldwide. Notably, incidence of severe postpartum hemorrhage has risen over the past decade. Labor induction has also been linked to increased risk for postpartum depression, a serious mental health condition impacting nearly one in five mothers that can lead to decreased quality of life for mothers and disrupted mother-infant interactions and caregiving. Despite the rising rates of severe postpartum hemorrhage and the high prevalence of postpartum depression, little is known about factors that increase risk for these outcomes. For some women, the common link between labor induction, postpartum hemorrhage, and postpartum depression risk may well be altered functioning of the oxytocin system. Here we use the prairie vole to understand how pregnancy, natural birth, and labor induction with synthetic oxytocin impact epigenetic control of the oxytocin receptor gene, Oxtr, in the brain and the uterus of new mothers. Oxtr DNA methylation (5mC), DNA hydroxymethylation (5hmC), and gene expression were examined at time points throughout pregnancy, in the peripartum period, and postnatally. These same Oxtr markers were examined in a second cohort of term pregnant females after administration of exogenous oxytocin prior to labor, a model of labor induction. Results identify a switch in the

regulatory state of Oxtr from late pregnancy to early postpartum in unmanipulated birth that is driven by 5mC/5hmC turnover in the regulatory region of Oxtr. This turnover increases at term pregnancy, facilitating a rise in Oxtr transcript expression, and then returns to pre-pregnancy levels within one week postpartum. This shift in 5mC/5hmC turnover that occurs at term pregnancy can be accelerated by labor induction. Maternal treatment with exogenous oxytocin results in an epigenetic state of Oxtr that resembles what is seen postnatally in unmanipulated birth, but at a prenatal time point in both the maternal brain and uterus. These findings provide valuable insight into how natural birth and labor induction, a very common birth intervention, differentially shape development of the epigenetic state of Oxtr in the maternal uterus and brain and highlight an important avenue to explore when considering factors that increase risk for adverse maternal health outcomes in women.

Vasopressin/oxytocin peptide-signaling in marine planarians functions as an antidiuretic before vascular system acquisition and synapse evolution

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Vasopressin (VP) and oxytocin (OT) have been fundamental in regulating key physiological functions in mammals for over a century. VP, known as an antidiuretic hormone (ADH), is vital for mammalian osmoregulation, while OT stimulates uterine contractions during childbirth and milk ejection during breastfeeding. Recent studies have illuminated their roles beyond the endocrine system, demonstrating their involvement in emotion, cognition, learning, bonding, and various socio-sexual behaviors within the central nervous system. Orthologs of VP/OT are widely conserved among bilaterians, indicating an evolutionary origin tracing back to a common ancestor. Despite previous assumptions, investigations into basic animal genomes have identified an ancestral VP/OT-like neuropeptide system in intertidal planarians, termed the "platytocin" system (1). This discovery challenges the belief that such systems are absent in Platyhelminthes and suggests platytocin may be the earliest VP/OT-like molecule in animals (1). Experiments with euryhaline planarians, adapting to fluctuating salinities, reveal platytocin's role as an ADH, regulating body fluids in amphibious environments. Moreover, platytocin is implicated in sensory processing, learning, and reproduction, potentially serving as a precursor to vertebrate VP/OT systems. Platytocin is localized within neuronal processes, coexisting with dense-cored vesicles containing conventional neurotransmitters. However, definitive "chemical" synaptic structures are yet to be observed in planarians or non-bilaterians, suggesting a transitional phase in nervous system evolution from synaptic to neuroendocrine systems. The platytocin system in planarians presents an ideal model for studying the evolutionary development of synapses and neuroendocrine systems.

Platyhelminthes are one group of simple bilaterian invertebrates; they have a simple body system: no vascular-circulatory system, no body cavity, and a mouth and anus that are morphologically identical. In contrast, they do have an organized “brain” as a central nervous system (2). It is proposed that the central (neuro)endocrine system became indispensable with the evolution of centralized nervous systems in primitive bilaterians like Platyhelminthes.

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Microbial modulation of the oxytocinergic-mesocorticolimbic dopaminergic pathway in mouse models for autism

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Progress in understanding the neural basis of social behavior has yielded remarkable insight into how dysfunction within the social reward circuit and of the underlying molecular machinery – centered around the oxytocinergic system – contributes to the social deficit endophenotype characteristic of autism spectrum disorders (ASD). The genetic heterogeneity of ASD, however, has presented a serious challenge toward the development of broadly effective therapies for treating social dysfunction across the diverse autistic patient population, necessitating novel therapeutic approaches. Over the past decade, our work[1-4], and that of others, in preclinical genetic, environmental, and idiopathic mouse models for ASD has begun to reveal the complex interplay between host and microbial genetics – the metagenome – modulating brain development, function, and behavior, including social behavior. Simultaneously, it has unveiled the therapeutic potential for precision targeting of the gut microbiome to relieve and, in some cases, even prevent social deficits, which is now beginning to be realized in human patients[5]. Indeed, in a recent double-blind, randomized, placebo-controlled clinical trial, the commensal bacterial species we first found to rescue social dysfunction by restoring hypothalamic oxytocin and related deficits in synaptic plasticity within the dopaminergic reward system in male offspring born to high-fat diet-fed dams, Limosilactobacillus reuteri, was found to selectively reverse social deficits in children with ASD. In this talk, I will provide an overview of how a serendipitous discovery at the bench morphed into a promising, microbiome-targeted treatment for social dysfunction in ASD. I will also discuss ongoing, multi-OMICs-informed studies in the lab aimed at therapeutic targeting of the maternal gut microbiome to prevent social dysfunction in mouse models for ASD, and, ultimately, human patients.

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Long-term effects of Cesarean birth on vasopressin and oxytocin neurons

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Birth is an extraordinary event for placental mammals and occurs at a time when key developmental processes, such as neuronal cell death, are shaping the brain. Little is known about the contributions of birth to brain development and whether birth mode (vaginal vs. Cesarean) alters neurodevelopmental trajectories. To study these questions, we manipulated birth mode in mice (matched for gestation length and birth time) and found that, on the day of birth, Cesarean-born newborns had greater cell death across many brain regions, with the most dramatic effect observed in the paraventricular nucleus of the hypothalamus (PVN). Intriguingly, this effect was associated with reduced vasopressin (VP) neuron number at weaning and in adulthood, specifically in regions of the PVN enriched for magnocellular VP neurons. Cesarean birth also reduced VP soma size and VP efferent projections from the PVN. We also investigated whether the effect of birth mode has long-term effects on oxytocin (OT) neurons in the PVN. Although Cesarean birth also reduced the

number and size of OT neurons in the nucleus, these effects were subtler than for VP neurons. No effect of birth mode was found in other prominent VP and OT hypothalamic populations: the suprachiasmatic and supraoptic nuclei. Our findings are likely of functional relevance because we also found that Cesarean birth was associated with behavioral alterations (softer neonatal ultrasonic calls, increased nest-building behaviour), as well as increased body weight, which are functions linked to VP signaling. Thus, Cesarean delivery causes long-term effects on the VP and, to a lesser extent, OT systems in the PVN, suggesting that this region is particularly sensitive to the effects of birth mode. We are currently determining under both baseline and physiological stress conditions whether (i) the birth mode-dependent changes in VP neuron number are associated with differences in VP production and (ii), given that VP participates in glucocorticoid release, we are also investigating whether birth mode influences circulating levels of corticosterone. Our studies are of clinical relevance given the widespread practice of Cesarean delivery across the world.

High-calorie diets uncouple hypothalamic oxytocin neurons from a gut-to-brain satiation pathway via κ-opioid signaling

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Oxytocin-expressing paraventricular hypothalamic neurons (PVN^{OT} neurons) integrate afferent signals from the gut including cholecystokinin (CCK) to adjust whole-body energy homeostasis. However, the molecular underpinnings by which PVN^{OT} neurons orchestrate gut-to-brain feeding control remain unclear. Here, we show that mice undergoing selective ablation of PVN^{OT} neurons fail to reduce food intake in response to CCK and develop hyperphagic obesity on chow diet. Notably, exposing wildtype mice to a high-fat/high-sugar (HFHS) diet recapitulates this insensitivity towards CCK, which is linked to diet-induced transcriptional and electrophysiological aberrations specifically in PVN^{OT} neurons. Restoring OT pathways in diet-induced obese (DIO) mice via chemogenetics or polypharmacology sufficiently re-establishes CCK's anorexigenic effects. Lastly, by single-cell profiling we identify a specialized PVN^{OT} neuronal subpopulation with increased κ-opioid signaling under HFHS diet, which restrains their CCK-evoked activation. In sum, we document a (patho)mechanism by which PVN^{OT} signaling uncouples a gut-brain satiation pathway under obesogenic conditions.[1]

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Changes in neuropeptide large dense core vesicle trafficking dynamics contribute towards adaptive responses to a systemic homeostatic challenge

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Neuropeptides are packed into large dense core vesicles (LDCVs) that are transported from the soma out into their processes. Limited information exists regarding mechanisms regulating LDCV trafficking, particularly in vasopressin (VP) neurons during challenges to bodily homeostasis.

Addressing this gap, we used 2-photon imaging in an ex vivo preparation to study LDCVs trafficking dynamics in VP neurons, which traffic and release neuropeptide from their dendrites and axons. We report a dynamic bidirectional trafficking of VP-LDCVs with important differences in speed and directionality between axons and dendrites. Acute, short-lasting stimuli known to alter VP firing activity and axonal/dendritic release caused modest changes in VP-LDCVs trafficking dynamics. Conversely, chronic/sustained systemic osmotic challenges upregulated VP-LDCVs trafficking dynamics, with a larger effect in dendrites. These results support differential regulation of dendritic and axonal LDCV trafficking, and that changes in trafficking dynamics constitute a novel mechanism by which peptidergic neurons can efficiently adapt to conditions of increased hormonal demand.

Thyroid dependent disruption of oxytocin and gut microbiome in an environmental autism mouse model

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The incidence of neurodevelopmental disorders such as Autism Spectrum Disorder (ASD) is rising at an alarming rate. ASD etiology is thought to be multifactorial, resulting from genetic susceptibilities that may interact with environmental toxicant exposures. In humans, PBDEs are thyroid-disrupting chemicals associated with altered socioemotional behavior, decreased IQ and hyperactivity. We have previously demonstrated ASD-like effects after perinatal exposure to PBDEs[1]. Maternal thyroid hormone (TH) status affects fetal brain development and has been linked to ASD. Since THs regulate oxytocin (OXT) and/or vasopressin neuroendocrine systems critical for social behavior, PBDEs may produce ASD-like actions via disruption of the TH system. Therefore, we tested the hypothesis that developmental exposure to PBDEs produce neurobehavioral and hypothalamic OXT deficits in a TH-dependent manner. C57BL/6N dams were exposed to human-relevant doses of the penta-PBDE mixture, DE-71, at 0.1 mg/kg/d (L-DE-71), 0.4 mg/kg/d (H-DE-71) or with vehicle control (VEH/CON) for 10 wks perinatally. A subset of dams received supplementation with levothyroxine (mT4), a synthetic analogue of thyroxine (T4) (8 µg/100 g bw; GD 12-PND 21). Other dams were administered only the thyroid synthesis inhibitor, 6-propyl-2-thiouracil (PTU; 50 mg/L; GD 14-PND 21). Compared to VEH/CON, free T4 was attenuated in L-DE-71 dams. mT4 significantly elevated plasma TT4 in L-DE-71 and plasma OXT in L-DE-71 and VEH/CON groups. Plasma free T4 was elevated in L-DE71 (but not L-DE-71+mT4) female offspring at PND15. On a social novelty preference test adult male and female L-DE-71 offspring showed no preference for novel over familiar conspecific; mT4 rescued the preference for novel only in females. To determine if the social deficit is associated with neuroendocrine disruption of OXT, we performed stereological and densitometric analyses on OXT immunofluorescence in PND 30 hypothalamic sections. DE-71 reduced OXT immunofluorescence IOD and total number of OXT-ergic cells in the paraventricular nucleus (PVH) in male (Land H-DE-71) and female offspring (L-DE-71) and in supraoptic nucleus (SON) in male offspring (L-DE-71). mT4 supplementation normalized OXT content and cell number in L-DE-71 female and H-DE-71 male PVH; no rescue was observed in SON. Plasma OXT levels were not compromised by DE-71 but were upregulated in female VEH/CON+mT4 female and male L-DE-71+mT4 and H-DE-71+mT4. RT-qPCR showed that DE-71 downregulated hypothalamic Trh and cortical Oatp1c1 and Dio3 in females. Gene alterations were sex-dependent with males showing downregulated hypothalamic Oxt and upregulated hypothalamic Dio2, Esr1 and Esr2 and cortical Oatp1c1. PTU's actions did not exactly mimic those of DE-71. In ongoing in situ hybridization experiments we are testing the RNA expression of these deregulated thyroid responsive markers in PVH OXTergic neurons. Analysis of 16S rRNA NGS indicates significant thyroid dependent effects of L-DE-71 on gut microbiome. Our results suggest that PBDEs disrupt social behavior and central OXT in a TH-dependent and sex-dependent manner. We provide novel information about possible TH-responsive genes that may contribute to neurodevelopmental disorders such as ASD.

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Symposium 6 Abstracts

Investigating the link between ASD-risk genes, oxytocin, and social behaviour

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Difficulties with social interactions and communication are core symptoms of autism spectrum disorders (ASD). Although the neurobiological mechanisms of social difficulties in ASD remain poorly understood, recent evidence suggests that oxytocin, a neurohormone with an established role in social bonding and trust, could play an important role. Using genetically-engineered mouse models and integrating multiple approaches, my laboratory's research investigates the possibility that separate ASD-linked gene mutations (e.g. Cntnap2, Fmr1, Shank3) lead to a common phenotype of disrupted oxytocin signalling in the brain, as a potential convergent mechanism underlying the low sociability in ASD. In this talk, I will discuss previous and current evidence that supports this hypothesis.

Vasopressin: A trans-primate biomarker of social impairment and promising treatment for autism

Karen J. Parker

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Autism spectrum disorder (ASD) is currently diagnosed behaviorally because its pathophysiology remains poorly understood. Consequently, there are no laboratory-based diagnostic tests to detect ASD and no disease-modifying medications that effectively treat its core behavioral features. The capability of rapidly detecting ASD based on neurochemical markers, however, would revolutionize ASD detection, enable more timely behavioral intervention, and provide targets for pharmacological treatment. To address these urgent unmet clinical needs, we developed a translational ASD research program, spanning studies of naturally low-social rhesus monkeys to children with ASD. Converging evidence from this body of research indicates that the neuropeptide vasopressin plays a critical and conserved role in regulating social abilities, and that brain vasopressin (but not oxytocin) signaling is impaired in low-social monkeys, children with ASD, and newborn infants before the period when ASD first manifests. On the basis of this compelling evidence, we recently conducted a double-blind, randomized, placebo-controlled pilot trial. We found that intranasal vasopressin treatment is well tolerated and significantly improves social abilities in children with ASD. These findings suggest that a neurochemical marker of impaired social functioning may be present very early in life, before behavioral symptoms emerge, and that the vasopressin signaling pathway may hold diagnostic and therapeutic promise for ASD.

Symposium 6 Abstracts

Unravelling the pharmacodynamics of oxytocin using functional neuroimaging

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Intranasal oxytocin has attracted attention as a potential treatment for several neuropsychiatric and neurodevelopmental disorders due to promising preclinical results. However, a plethora of clinical trials and research studies in humans has yielded inconsistent results which, to a large extent, may have resulted from our lack of understanding of the pharmacodynamics of intranasal oxytocin. In this talk we will show results from a series of studies, in healthy humans, patients and mice that systematically capitalized on innovative combinations of pharmacological research, imaging transcriptomics, and recent progress in advanced neuroimaging to precisely characterize the pharmacodynamics and central mechanism of intranasal oxytocin.

First, we will discuss a set of studies in healthy volunteers where we characterised the spatiotemporal profile of changes in brain's physiology after administration of oxytocin, compared the effects across routes/methods of administration and investigated dose-response focusing on a key central target, the amygdala. Second, we will showcase how neuroimaging markers of functional response to intranasal oxytocin might provide a principled way of assessing disease-target engagement during early treatment development focusing on people at clinical high risk for psychosis, compulsive eating and antisocial personality disorder. Third, we will demonstrate the importance of gathering human-specific data when it comes to the effects of oxytocin on the brain by presenting some cross-species preliminary data on an innovative MRI biomarker of excitation:inhibition (E:I) balance, a key element of brain functioning known to be disrupted in many neuropsychiatric conditions.

Acute versus chronic matters: Differential behavioural and molecular effects of oxytocin

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Oxytocin (OXT) currently attracts enormous scientific attention due to its prominent prosocial, anxiolytic and anti-stress effects (1). The role of brain OXT in balancing socio-emotional behaviours has extensively been studied using physiological models of naturally occurring elevated brain OXT system activity, pharmacological, pharmacogenetics and optogenetics approaches. For example, brain OXT is essential for naturally occurring social preference behaviour, and the reversal of stress-induced social avoidance and social fear.

However, open questions regarding fundamental mechanisms of action remain, which need to be deciphered, before OXT can be considered a safe treatment option for psychopathologies associated with social and emotional dysfunctions. With respect to its therapeutic implications OXT effects were found to be dose- and duration-dependent, as opposite behavioural effects were found after acute versus chronic treatment. The underlying molecular mechanisms of the anxiogenic effects of chronic OXT will be described, which may challenge the concept of OXT as a chronic treatment option of psychiatric disorders (2). Moreover, non-synonymous mutations in the OXT receptor gene, which have been associated with psychopathologies, may alter the cellular response to synthetic OXT and, thus, treatment success in mutant carriers (3). Supported by DFG, BMBF and EU.

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Symposium 7 Abstracts

Neonatal oxytocin administration in mouse models of neurodevelopmental disorders: Long lasting rescue effects

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Oxytocin (OXT), a master regulator of the social brain in early infancy, adolescence and adult life, is under intense investigation as a potential therapeutic treatment in disorders characterized by social deficits. Abnormalities in OXT release and/or OXTR distribution have indeed been reported in several mice model of neurodevelopmental disorders, suggesting a crucial role of this system in the appearance and progression of symptoms. Here we will discuss the results obtained with a perinatal OXT supplementation in two models of neurodevelopmental disorders characterized by an impairment of the OXT system already at birth.

The Magel2-KO is a model of Prader-Willi and Schaaf Yang syndromes, two neuro-developmental diseases characterized by autism-related features, sensory impairments and feeding difficulties in early infancy. In Magel2-KO mice, an early postnatal OXT treatment was demonstrated to rescue neonatal lethality, to prevent the appearance of social and learning deficits as well as deficit in hippocampal circuitry and functions. Investigating OXTR developmental trajectories, we found that males and females Magel2-KO displayed a widespread, substantial, down-regulation of OXTR levels as compared to WT animals and that the post-natal OXT treatment restored OXTR levels in adult Magel2-KO in sex and region-specific ways.

The effects of OXT administration were also investigated in the LgDel/+ transgenic mice, which model the human deletion of 22q11.2 (22q11.2DS), also known as DiGeorge Syndrome, one of the most common genetic vulnerability factors for psychiatric disorders. The 22q11.2DS is associated with a spectrum of developmental social and intellectual disabilities, high prevalence of ADHD (~37%) and ASD during childhood, and schizophrenia (~41%) in adulthood. Notably, the altered behavioral, cortical and neuro-immunological postnatal developmental trajectories in 22q11.2DS mice were ameliorated by a perinatal treatment with intranasal OXT. In particular, OXT supplementation prevented the development of sensorimotor gating and social deficits and attenuated both peripheral and brain inflammatory phenotypes in LgDel/+ mice. This was related to a long-lasting effect of OXT in upregulating the expression of tight junction molecules Claudin-5 and Claudin-1, reducing the permeability of the blood-brain and the blood-cerebrospinal fluid barriers. Our hypothesis is that, in addition to its effects on neurons and glial cells, OXT regulation of the vascular compartment also contribute to the therapeutic potential of the neuropeptide during post-natal neurodevelopment.

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Consequences of altered oxytocin and vasopressin signaling during embryonic development

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The oxytocin (Oxt) and arginine vasopressin (Avp) systems, beyond being modulators of sex-specific social behaviors in juvenile and adult animals, are also important in development. In mice, Oxt (Oxtr) and vasopressin 1a receptors (Avpr1a) are present and functional in embryonic life, positioning them to directly influence aspects of brain development. Recent work from our lab has been exploring how perturbations in either Oxtr or Avpr1a signaling during embryonic development may affect brain development and behavior. After pharmacologically disrupting Oxtr or Avpr1a signaling on embryonic day (E)16.5, we have been measuring the effects on adolescent and adult behaviors. Our data suggest that disruption of either system results in measurable, but distinctive, effects on behavior. Thus, it appears both Oxtr and Avpr1a signaling during embryonic development may directly support the development of the neural substrates that regulate social behaviors.

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Neonatal organizational effects of oxytocin and subsequent behavioral expression in prairie voles (*Microtus ochrogaster*)

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The "neurohormone" oxytocin (OT) is critical role in the expression/modulation of adult sociosexual behavior. In polygynous mammals, where females often avoid males, OT is essential in reducing social avoidance and stress, facilitating the social contact required for mating, and establishing maternal/infant bonds. In socially monogamous species, such as prairie voles (Microtus ochrogaster), OT is involved in the formation of male/female partner preferences that are essential for the formation of long-term pair bonds and male parental behavior. While the adult effects are well document and often the focus of studies, OT also plays a major role in "establishing" the adult responses during early development through major organizational effects with the brain. This is important as the early social environment influences the production and release of OT, which then in turn can alter the expression of adult sociosexual behavior. The purpose of this presentation is discuss the organizational effects of neonatal OT manipulation on the expression of adult social behavior, focusing on the neural mechanisms that are known to be involved in the regulation of sociosexual behavior including the central production of OT, serotonin receptors and the expression of estrogen receptor alpha.

The role of oxytocin in the metabolic consequences of delivery by Cesarean section

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Delivery by caesarean section (CS) is associated with a >50% increase in the risk of developing obesity by 5 years of age. Importantly, the association between CS and subsequent obesity survives correction for: breastfeeding, antibiotic exposure, and maternal characteristics such as obesity. We hypothesize that this relationship is causal and driven by the abridged oxytocin signalling that neonates experience when delivered by CS. Compared to vaginally delivered counterparts, CS neonates experience lower levels of oxytocin and several other 'birth-signalling' hormones as measured in cord blood. Our previous work has shown that manipulations of oxytocin during the perinatal period can produce lifelong neurodevelopmental consequences. Here, we set about modelling CS in prairie voles (Microtus ochrogaster). Our first studies confirmed that indeed, offspring delivered by CS go on to show heavier body weights that persist into adulthood. Moreover, we can prevent this heavy phenotype by treating CS neonates with oxytocin (0.1 mg/kg, subcutaneous). Our preliminary findings suggest that CS offspring show reduced oxytocin immunoreactivity in the paraventricular nucleus of the hypothalamus as adults. We will present further findings on the metabolic profile of CS offspring as well as on oxytocin ligand and receptor densities in appetite and metabolism-regulating brain regions. Because delivery by CS now accounts for 32.1% of all births in the United States, we see this issue as having tremendous public health relevance. We hope that by normalizing the hormonal milieu at delivery, the negative neurodevelopmental consequences of CS delivery can be avoided while preserving the procedure as a medically necessary intervention.

Symposium 8 Abstracts

The oxytocin system plays a key role in brain microvascular development

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The interplay between vascular and neuronal systems is critical for the normal growth and function of neurons [1]. Indeed, brain development relies heavily on proper cerebrovasculature, that not only supports the proliferation, differentiation and migration of neural progenitors but also ensures brain homeostasis, and the supply of oxygen and nutrients for healthy neuronal functions [2]. Alterations of the brain microvascular network has already been reported in diverse neurological and psychiatric conditions like Alzheimer [3], Parkinson Diseases [4] and chronic pain [5] and very recently also in ASD [6, 7, 8].

The oxytocin (OXT) system, regulating postnatal neuron maturation is strongly implicated in pathological conditions affecting the social sphere, and can also have an effect on angiogenesis and vascular functions [9], an aspect generally overlooked.

We found that OXT receptors are expressed at the level of brain microvessels and, applying innovative technologies and tools for the study of brain anatomy, we observed in mouse models of neurodevelopmental disorders that OXT system alterations negatively impact brain vasculature development and functions.

We discovered an unexpected new important role of the OXT system in brain microvascular development and our data support the hypothesis that brain vascular deficiency can contribute to the etiopathogenesis of neurodevopmental disorders, opening the way to novel therapeutic strategies.

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Symposium 8 Abstracts

Oxytocin may facilitate its own peripheral uptake by regulating blood flow dynamics

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The hypothalamo-neurohypophyseal system is an important neuroendocrine brain-to-blood conduit through which the neurohormones oxytocin and arginine-vasopressin are released into the general circulation to affect peripheral physiological functions in response to various homeostatic challenges. These neuro-hormones are released from hypothalamic axonal termini into the perivascular space of the posterior pituitary, also known as the neurohypophysis. However, whether a simple diffusion force drives the uptake of oxytocin and vasopressin from the perivascular spaces into the blood circulation, or an active mechanism executes fast and efficient neurohormone release to the periphery remains unsolved.

Here we suggest that neuro-vascular coupled changes in blood flow dynamics facilitate uptake of neurohormones into the periphery. We have established a method to dynamically monitor activity-dependent capillary blood-flow and permeability by using live-imaging of a transgenic serum-labelled larval zebrafish. We show that following an osmotic physiological challenge, oxytocin neurons were activated concomitant with local oscillation of blood flow velocities of neurohypophyseal capillary. Genetic ablation of oxytocin neurons as well as treatment with oxytocin receptor antagonist attenuated stimulus-dependent effects on capillary blood flow. Conversely, optogenetic stimulation of oxytocin neurons resulted in increased blood flow velocities, which were inhibited in the presence of the oxytocin receptor antagonist. Lastly, osmotic challenge elicited a local increase in neurohypophyseal capillary permeability in an oxytocin signaling-dependent manner suggesting self-perpetuating mechanism of peripheral hormone secretion.

Our study demonstrates that physiologically-elicited changes in neurohypophyseal blood flow and permeability, are regulated by oxytocin. We propose that oxytocin-dependent neuro-vascular coupling facilitates its efficient uptake into the blood circulation to affect its peripheral target organs, rather than a diffusion-based hormone secretion.

Vasopressin-mediated neurovascular coupling in health and diseases states

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Neurovascular coupling (NVC), the process that links neuronal activity to cerebral blood flow changes, has been mainly studied in superficial brain areas, namely the neocortex. Still, whether the conventional, rapid and spatially restricted NVC response can be generalized to deeper, and functionally diverse brain regions, and whether this is altered in disease states, remains unknown.

We addressed this important gap in our knowledge by implementing a novel in vivo, real-time two-photon experimental approach using a transgenic eGFP VP rat. In a recent study, we demonstrated that a systemic salt load evoked a significant intraparenchymal arteriole (but not venule) vasoconstriction that was mediated by locally released VP within the SON. The salt-induced vasoconstriction resulted in a progressive decrease in SON pO2. In this study, we aimed to investigate whether this salt-evoked NVC response was altered in rats with chronic heart failure (HF). We found that under baseline conditions, SON pO2 levels were lower in HF compared to sham rats ($p<0.01$, $n=7$). Basal vascular diameter of the internal carotid artery (ICA) and intraparenchymal arteriole were however not significantly different between sham and HF rats (for ICA; $p=.1147$, intraparenchymal artery; $p=.8815$, $n=6$ each group). Despite this initial basal hypoxia, we found that the salt challenge induced intraparenchymal arteriolar vasodilatation ($p < 0.01$; $n = 6$) and a progressive increase in SON pO2 ($p < 0.001$; $n = 6$) in HF rats. Direct application of VP in the SON via a microdialysis probe still induced arteriolar vasoconstriction ($p<0.01$, $n=5$). We tested then the hypothesis that the salt-evoked vasodilation in HF rats is mediated by local release of adenosine. Intra SON delivery of an adenosine agonist (CGS21680) in control rats causes vasodilation of intraparenchymal arterioles in sham rats ($p < 0.0,1$ $n= 4$). Finally, we found that the salt-evoked vasodilation in HF rats was absent when an adenosine A2A receptor antagonist

(ZM241385) was locally delivered into the SON. In fact, a salt-dependent vasoconstriction was unmasked under this condition ($p < 0.05$; n=4), which was significantly higher compared to salt induced vasoconstriction in Sham rats ($p<0.01$, n=6 in sham and n=4 in HF). Together, our results support a change in the polarity of the salt-induced NVC in HF rats, from vasoconstriction (VP-mediated) to vasodilation (adenosine-mediated). The cellular source of adenosine, as well as the overall functional significance of the NVC switch during HF remains to be determined.

Special Talk: Larry Young's Scientific Contributions

Natural variation in oxytocin receptor signalling causes widespread changes in neural gene expression: a link to the natural killer gene complex

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Natural variation in oxytocin receptor (OXTR) levels increases diversity in social behaviors. In the socially monogamous prairie vole (Microtus ochrogaster), variation in Oxtr expression in the nucleus accumbens (NAC) is associated with variation in pair bonding, alloparental behavior and resilience to neonatal social neglect. Previously, we found a set of nine single nucleotide polymorphisms in and near the Oxtr gene that largely explain individual variation in Oxtr expression, specifically in the NAC ($R^2>0.7$). Our aim is to understand the molecular mechanism that links genetic variation in the Oxtr gene to social diversity. To this end, we performed bulk ATAC and RNA-seq, and single nucleus RNA-seq (snRNA-seq) on the two genotypes that either produce low (T/T) or high (C/C) NAC OXTR.

Firstly, we reveal wide-spread transcriptional changes between genotypes across brain areas and cell types. Interestingly, most differential expressed genes (DEGs) were found in a region that is devoid of OXTR protein (superior colliculus), and in cell types that express no or very little Oxtr mRNA, such as oligodendrocytes and astrocytes. Using a systemic OXTR knock-out prairie vole that was constructed on the high NAC OXTR genetic background (C/C), we show that the expression of the majority of DEGs (>75%) is affected by a lack of OXTR signalling.

Specifically, we find an enrichment for DEGs ($P<9.33e^{-6}$) and differentially accessible regions (DARs; $P=3.36e^{-39}$) in the natural killer gene complex (NKC), which locates to the Oxtr chromosome. This genetic region harbours a cluster of C-type lectin-like receptors (CTLRs), which function as receptor (CLEC subtype)-ligand (KLR subtype) pairs in the periphery. Two CTLR genes were found to be strongly DE between genotypes: Clec12a ($P_{adj}=8.5e^{-9}$) and Klrb1a ($P_{adj}=3.93e^{-40}$). Clec12a is restricted to microglia, and Klr1b1a expression is enriched in Oxtr expressing cells ($P=9.24e^{-16}$). This suggests that CLEC:KLR interactions are used centrally in the interaction between microglia and neurons to guide synaptic pruning. We are currently investigating this possibility.

Finally, we provide evidence that a link between CLEC:KLR and OXTR expression exists in humans as well. Interestingly, two human KLR genes (KLRC2 and KLRF1) have been associated with autism spectrum disorders. We find that the expression of these genes varies with Oxtr mRNA. Collectively, our findings show that OXTR signalling impacts brain function, possibly by controlling microglial activity. Thereby, they could explain how cumulative differences in oxytocin signalling (either through genetic background or experience) can have lasting impact on social behaviors.

Symposium 9 Abstracts

Oxytocin in sensory-dependent social development

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Oxytocin (OXT) regulates species-typical social behaviors and interacts with early life experience to shape adult responses. In mice, robust infant OXT production emerges postnatally and depends on sensory experience, with increased sensory experience leading to increased OXT production. We observed that OXT receptors (OXTR) are in the periphery of the neonate in sites receiving parental contact, such as the whisker pads, oronasal cavity and the anogenital area. OXT may modulate such social sensory contact, as we also observed OXTR expression in primary sensory neurons in the peripheral nervous system. Thus, we hypothesize that peripheral OXTR activity impacts sensory-dependent brain and behavior responses in developing and mature mice. Using anatomical, genetic, pharmacological, and behavioral methods in mice, we identified the primary sensory circuits expressing OXTR, the impact of loss of OXTR on OXT production and behavior, and the brain and behavioral response to exogenous application of OXT. We identified OXTR on predominantly mechanosensory neurons of the trigeminal nerve and dorsal root ganglia. Lack of OXTR in a congenital knock-out reduced the production of Oxt mRNA and neural activity in the paraventricular nucleus of the hypothalamus (PVN) during post-natal development. Application of OXT to the face increased activity in the PVN of pre-weanling male, but not female, mice. In contrast, OXT decreased the neural response to whisker stimulation in canonical whisker circuits. Mice with a conditional deletion of the Oxtr gene from cells of neural crest origin (e.g. peripheral sensory ganglia) demonstrated blunted sociability and increased aggression as adults. Combined, these data are consistent with a role for OXTR on peripheral sensory neurons to modulate sensory-dependent social interactions, beginning in development, with potential for sex-specific effects.

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Studying mechanosensitive vagal afferents that express oxytocin receptors: gut feelings are also matters of the heart

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Interoception broadly refers to awareness of one's internal milieu. Vagal sensory afferents monitor the internal milieu and maintain homeostasis by engaging brain circuits that alter physiology and behavior. While the importance of the body-to-brain communication that underlies interoception is implicit, the vagal afferents and corresponding brain circuits that shape perception of the viscera are largely unknown. Here, we use mice to parse neural circuits subserving interoception of the heart and gut. We determine vagal sensory afferents expressing the oxytocin receptor, hereafter referred to as NDGOxtr, send projections to the aoritc arch or stomach and duodenum with molecular and structural features indicative of mechanosensation. Chemogenetic excitation of NDGOxtr significantly decreases food and water consumption, and remarkably, produces torpor-like phenotype characterized by reduction in cardiac output, body temperature, and energy expenditure. Chemogenetic excitation of NDGOxtr also creates patterns of brain activity associated with augented hypothalamic-pituitary-adrenal axis activity and behavioral indices of vigilance. Recurrent excitation of NDGOxtr supresses food intake and lowers body mass, indicating that mechanosensation of the hear and gut can exert enduring effects on energy balance. These findings suggest that the sensation of vascular stretch and gastrointestinal distention may have profound effects on whole body metabolism and mental health.

Symposium 9 Abstracts

Unveiling the dynamics of oxytocin activity and somatodendritic release in freely behaving rodents

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The advent of new technologies in neuroscience research has provided the opportunity for a more comprehensive understanding of how the brain controls physiology and behaviour at the level of individual neurons, populations of neurons and networks. We are bringing these technologies to bear on the oxytocin system's role in the control of maternity in freely behaving mammals.

Initially, we used GCaMP photometry to confirm that the patterns of milk-ejection bursts in oxytocin neuron of mice are similar to those previously observed in rats, with the inter-burst interval principally being dependent on the number of pups suckling. We are now combining fibre photometry with the use of a novel oxytocin sensor (OT1.9; gifted by Prof Yulong Yi, Peking University) to monitor the contribution of somatodendritic oxytocin release to oxytocin neuron burst generation with a higher temporal resolution than could be achieved previously with microdialysis. These experiments have just started, and we hypothesize that somatodendritic oxytocin will progressively increase from the onset of suckling until a threshold is reached to trigger each burst until suckling ceases. In addition, we are using the oxytocin sensor to determine the pattern of synaptic oxytocin release in the nucleus accumbens, where oxytocin interacts with the dopaminergic system to modulate maternal behaviour.

With a view to investigating the spread of excitation across the oxytocin neuron population during bursts at a neuron-by-neuron level, we developed the use of Neuropixels for multi-single unit recording of oxytocin (and vasopressin) neuron activity. We have validated Neuropixels in urethane-anesthetized rats, finding a robust response to intravenous infusion of hypotonic saline. Our next goal is to use Neuropixels in freely behaving lactating rats to explore the coordination of oxytocin neuron bursts within and across the paraventricular and supraoptic nuclei. We aim to determine whether there are any "hot spots" where bursts are more likely to start and whether somatodendrtic oxytocin diffusion is required for the spread of excitation across the population.

Overall, these diverse technological approaches collectively promise to advance our understanding of oxytocin's multifaceted role in maternal physiology and behaviour, opening new pathways in neuroendocrine research.

Symposium 10 Abstracts

Efficacy of oxytocin as a monotherapy and combination therapy to treat obesity

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Recent studies indicate that oxytocin (OT) reduces body weight (BW) in high fat diet (HFD)-induced obese (DIO) rodents and non-human primates, in part by reducing energy intake and increasing energy expenditure (EE). However, OT has only been found to elicit modest weight loss (≈ 3.3-9.3%) in DIO rodents and non-human primates and in humans who are overweight or obese (1-3). Recent studies suggest that combination therapy (i.e. dual agonists) may be more optimal than

monotherapy for sustained weight loss (4). Given the well characterized effects of OT to reduce energy intake in DIO rodents and nonhuman primates and humans with obesity (1-2; 5), what remains unclear is whether OT can be used as an adjunct with a drug that directly targets beta-3 receptors in interscapular brown adipose tissue (IBAT) to promote BAT thermogenesis (surrogate measure of EE) to elicit more pronounced weight loss in DIO rats. We hypothesized that the combined treatment of OT and the beta-3 agonist, CL 316243, would produce an additive effect to decrease BW and adiposity in DIO rats by reducing energy intake and increasing BAT thermogenesis. We assessed the effects of subcutaneous infusions of OT (50 nmol/day) or vehicle (VEH) in combination with daily intraperitoneal injections of CL 316243 (0.5 mg/kg) or VEH on food intake, IBAT temperature (T_{IBAT}; functional measure of BAT thermogenesis), BW and body composition. OT and CL 316243 alone reduced BW by 8.0±0.9% ($P<0.05$) and 8.6±0.6% ($P<0.05$), respectively, but the combined treatment produced more pronounced weight loss (14.9±1.0%; $P<0.05$) than either treatment alone ($P<0.05$). These effects were associated with decreased energy intake, adiposity, adipocyte size ($0.05<P<0.1$) and plasma leptin and insulin ($P<0.05$). In addition, CL 316243 alone ($P<0.05$) and in combination with OT ($P<0.05$) elevated T_{IBAT} throughout the treatment period. These findings suggest that the effects of the combined treatment on weight loss are additive and appear to be driven, in part, by OT- and CL 316243-elicited changes in energy intake as well as CL 316243-elicited increases in BAT thermogenesis.

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Using quantitative phosphoproteomics to explore hypothalamo-neurohypophysial system cellular signalling

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The hypothalamo-neurohypophysial system comprises large magnocellular neurones in the paraventricular nucleus and supraoptic nucleus that make the hormones vasopressin and oxytocin and release them peripherally into the blood circulation from nerve terminals located in the posterior pituitary gland, and within the brain from dendrites and axon collaterals. By signalling at specific receptors in the periphery and centrally vasopressin and oxytocin can regulate physiological processes crucial for the maintenance of homeostasis. To investigate hypothalamo-neurohypophysial system

cellular signalling in response to water deprivation, feeding, and anaesthesia, we have adopted quantitative proteomic approaches to study this system in the rat (1, 2). One of the most striking findings has been changes to the supraoptic nucleus phosphoproteome elicited by anaesthesia. Although suppressed in most parts of the brain, neuronal activity, as measured by FOS activation, is increased in the hypothalamic supraoptic nucleus by general anaesthetics, and evidence points to this brain region being involved in the induction of general anaesthesia and natural sleep (3). We found many changes in the phosphoproteomes in the supraoptic nucleus and cingulate cortex after 15 minutes of isoflurane exposure. Pathway analysis indicated that proteins undergoing phosphorylation adaptations are involved in cytoskeleton remodelling and synaptic signalling events. These data suggest that rapid posttranslational modifications in proteins involved in cytoskeleton remodelling and synaptic signalling events might mediate the central mechanisms mediating general anaesthesia. These omic datasets offer new insight into hypothalamo-neurohypophysial system cell signalling.

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Single-cell transcriptomics of hypothalamic magnocellular neurons: unraveling cellular diversity, activity-associated genes, and interspecies integration

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The hypothalamic magnocellular neurons (MCNs) reside in the supraoptic and paraventricular nuclei, playing a crucial role in the synthesis and release of neuropeptides, namely vasopressin (AVP) and oxytocin (OXT). These neuropeptides are essential regulators of renal water reabsorption and female reproductive function, respectively. Beyond their classical endocrine role, it is now apparent that MCNs exhibit collateral axons reaching various brain regions, influencing a spectrum of bodily, emotional, and cognitive functions. Consequently, understanding the molecular regulation and transcriptional diversity of MCNs becomes imperative to unravel the intricate regulatory mechanisms they govern. Our recent single-nucleus RNA sequencing analysis of the rat supraoptic nucleus unveiled eight distinct subtypes of MCNs. Notably, one major oxytocin (OXT) and two major vasopressin (AVP)-producing clusters were identified, each exhibiting unique transcriptomic signatures. Employing Cell-Chat analyses shed light on the putative pathways through which MCNs communicate with other neuronal and glial cells. Pseudotrajectory analyses pinpointed genes associated with the transcriptional plasticity of MCNs. To delve deeper into MCN regulation, we utilized NEUROeSTIMator and scVelo to identify genes and pathways linked to neuronal activation and AVP RNA processing during water deprivation. By integrating our newly generated rat data with previously published single-nucleus/cell RNA sequencing data from mouse, marmoset, and human hypothalamic tissues, we conducted a comprehensive analysis of transcriptional conservation and divergence in OXT and AVP-producing MCNs across mammalian evolution. This collective data provides a thorough transcriptomic dissection of MCN diversity, unveils activity-associated responses, and delineates its evolutionary molecular convergences and divergences in mammals.

Symposium 10 Abstracts

Light promoted brain development: ipRGC, oxytocin and synaptogenesis

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After birth, sensory inputs including vision (light) are crucial for promoting cortical synaptogenesis, one of the hallmarks of brain development. Evidence suggests that the release of oxytocin to the cerebrospinal fluid, which is necessary for this experience-dependent synaptogenesis, can also be enhanced by light inputs. However, the neural mechanism regulating this light-promoted synaptogenesis and the lifelong effects on cognition and learning ability remains unknown.

Developmentally, melanopsin-expressing intrinsically photosensitive retinal ganglion cells (ipRGCs) respond to light much earlier than rods and cones in mammalian retina, suggesting that ipRGCs may play a role in light-promoting brain development. Here we found that ipRGC-mediated light sensation promotes synaptogenesis of pyramidal neurons in various cortices and the hippocampus. This phenomenon depends on activation of ipRGCs and is mediated by the release of oxytocin from the supraoptic nucleus (SON) and the paraventricular nucleus (PVN) into cerebral-spinal fluid. We further characterized a direct connection between ipRGCs and oxytocin neurons in the SON and mutual projections between oxytocin neurons in the SON and PVN.

More interesting and important, we found that the lack of ipRGC-mediated, light-promoted early cortical synaptogenesis compromised learning ability in adult mice. Overall, our findings highlight the importance of light sensation and oxytocin diffusion in early life for the development of learning ability, and therefore call attention to suitable light environment for infant care.

Symposium 11 Abstracts

Neuropeptides trigger maternal care and aggression in lactating rats: influence of the stress system

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The most important pro-social behavior in female mammals is certainly taking care of the offspring. In order for a virgin to become maternal, the mother's brain undergoes dramatic changes in the peripartum period, termed as matrescence, leading to the necessary adaptations both in behavior and physiology. Among those brain adaptations are changes in the activity of the "pro-maternal" oxytocin (OXT) and arginine vasopressin (AVP) systems as well as of the rather "anti-maternal" corticotropin-releasing factor (CRF) system. Indeed, a fine-tuned central activity of these systems is necessary to promote maternal responses towards the young. One of brain regions we have studied in more detail is the bed nucleus of the stria terminalis (BNST), which is crucially involved in both maternal behavior and emotionality. Here, locally altered neuropeptides' transmission is linked to impairments of different aspects of maternal behavior, from nursing to retrieving to defending the pups. Specifically, we will discuss the effects of altered OXT and AVP transmission in the BNST on maternal aggression during the maternal defense test, a psychosocial stressor. As maternal aggression is reduced following intra-BNST manipulations of OXT and AVP systems, we further investigated the involvement of the stress system on local OXT or AVP release.

Overall, we find that intra-BNST neuropeptides signaling plays a crucial role in modulating maternal aggression, and that their release is increased during stress exposure as well as under increased CRF system transmission.

Symposium 11 Abstracts

The integration of interoceptive signals and defensive behaviors via neurohypophysial hormones in the bed nucleus of the stria terminalis (BNST)

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Interoceptive signals dynamically interact with the environment to shape appropriate defensive behaviors. Neurons in the hypothalamus regulate internal states, such as thirst and circadian rhythmicity, and send projections to the bed nucleus of the stria terminalis (BNST) to influence defensive responses, including fear and anxiety-like behaviors. Hypothalamic hormone and neuromodulator, arginine-vasopressin (AVP) modulates water and electrolyte balance and is involved in circadian rhythmicity. The dorsolateral BNST ($BNST_{DL}$) expresses oxytocin (OTR) and vasopressin receptors and here we show that AVP directly excites $BNST_{DL}$ neurons via OTR. These excitatory effects of AVP are confirmed by specifically recording excitability of fluorescent OTR-neurons from OTR-Cre transgenic male rats after application of AVP or selective OTR agonist, TGOT. Considering the well-established role of $BNST_{DL}$ in avoidance and fear-related behaviors, we next used chemogenetics in OTR-Cre rats and demonstrate that silencing of OTR-$BNST_{DL}$ neurons significantly reduces exploration of open arms of the elevated plus-maze and increases cue-induced vigilance in the fear-potentiated startle. We demonstrate how OTR-$BNST_{DL}$ neurons exited by hypothalamic AVP play a major role in regulating $BNST_{DL}$ excitability, overcoming threat avoidance, and reducing fear responses to ambiguous threats. Therefore, changes in the activity of internal state-sensitive hypothalamic nuclei will directly impact OTR neuron excitability in the $BNST_{DL}$ via hypothalamic inputs to shape appropriate, physiologically relevant levels of defensive behaviors.

Sex-specific regulation of social motivation by extrahypothalamic vasopressin

Petrulis A.

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It is well known that arginine-vasopressin (AVP) regulates social behavior and communication, often sex-specifically, but the sources of AVP release relevant for these behaviors have not been precisely determined. We have recently demonstrated using lesion, RNA knockdown, and optogenetic approaches, that the AVP-expressing cells in the bed nucleus of the stria terminalis (BNST), the major source of sexually-differentiated AVP in the brain, primarily drive male investigation of other males in mice. The effect of BNST AVP cells on male-male social interest may be due, in part, to action on the lateral septum (LS), a major output of BNST AVP cells and an area that strongly expresses vasopressin 1a receptor (V1aR). Stimulation of BNST AVP cells terminals in the LS increased male, but not female, social investigation as well as increasing male anxiety-like behavior, effects that could be blocked by V1aR antagonist. Stimulation of BNST AVP terminals ex vivo phasically increased, then decreased LS cell activity (V1aR-dependently), mimicking the timeline of in vivo increase in social investigation, suggesting that AVP-V1aR mediated inhibition of LS permits high levels of social investigation in males. Using a newly developed V1aR-cre driver mouse line, we have preliminarily data indicating that V1aR+ LS cells receive their strongest inputs from ventral hippocampal CA1 and project primarily to the diagonal band of Broca, lateral habenula, and supermammillary nucleus, all areas that also receive steroid-sensitive, sex-different AVP fibers and express substantial amounts of V1aR. Moreover, both BNST AVP and LS V1aR cells change their activity in response to social investigation. Consequently, BNST AVP cells may modulate contextual information (from hippocampus) to alter activity of an interconnected AVP-sensitive circuit and ultimately facilitate sex-specific social approach and investigation. As disorders of social behavior, such as autism, often show sex differences in prevalence, this work suggests that sex differences in

the neurochemical underpinnings of social behavior may contribute to sex differences in disorders of social behavior and communication.

Supported by grant funding from NIMH (R01MH121603, R21MH111104, R03MH120549, F31MH125659)

Transcriptional effects of social stress on oxytocin neurons in female California mice

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Oxytocin is a versatile neuropeptide that modulates many different forms of social behavior. Recent hypotheses pose that oxytocin enhances the salience of rewarding and aversive social experiences, and the field has been working to identify mechanisms that allow oxytocin to have diverse effects on behavior. In a series of studies in the California mouse (Peromyscus californicus) we observed that oxytocin acting in the nucleus accumbens promotes social approach whereas oxytocin acting in the bed nucleus of the stria terminalis promotes social avoidance and social vigilance. We also determined that in females but not males, social stress induces an enduring increase in the reactivity of oxytocin neurons in novel social situations. We performed single nucleus sequencing in the paraventricular nucleus (PVN) of female California mice assigned to control or stress conditions. A preliminary analysis of oxytocin neurons revealed at least two populations of oxytocin neurons. One population contained hundreds of transcripts that were differentially expressed after correcting for the false discovery rate. The second population of oxytocin neurons had many fewer transcripts affected by stress. These results mirror earlier c-fos analyses in PVN oxytocin neurons which demonstrated that in females, stress increased oxytocin/c-fos colocalizations in the anterior but not posterior PVN. These data suggest that it will be possible to identify molecular pathways that contribute to the increased reactivity of oxytocin neurons following social stress exposure and open new avenues for studying the neurobiology of social behavior.

Regulation of juvenile social behaviors by oxytocin and vasopressin systems in the brain

Samantha M. Bowden, Jessica D.A. Lee, and Alexa H. Veenema

Neurobiology of Social Behavior Laboratory, Department of Psychology & Neuroscience Program, Michigan State University

The neuropeptides vasopressin and oxytocin regulate various social behaviors across a wide range of taxa. Most research has focused on adult social behaviors and less is known about these neuropeptides in the development of social behaviors in both sexes. In this talk I will focus on how oxytocin and vasopressin regulate social play, a behavior displayed by juveniles of some avian and many mammalian species. Our research demonstrates that oxytocin and vasopressin regulate social play through distinct neural circuitries and in unique sex-specific ways.

WCNH Awardees

GLENN I. HATTON TRAVEL AWARD

The Glenn I. Hatton Memorial Travel Awards ($1000 each) will be presented for travel to WCNH 2024 in Atlanta GA for (2) meritorious students and (2) postdocs studying the neurobiology of vasopressin and oxytocin.

Please join the 2024 WCNH Local Organizing Committee in congratulating the winners of the Glenn I. Hatton Travel Award.

Michael Perkinson, Postdoc
University of Otago

Joshua Wyrosdic, Student
McGill University Health Centre

Francesca Galbiati, Postdoc
Massachusetts General Hospital

Camilla Paolini, Student
University of Milano Bicocca

POSTER AWARDS

Best Poster and Best Runner-Up Poster will be awarded to (2) graduate students and (2) postdocs for posters presented during the conference.

Winners will be announced at the Awards Ceremony and Closing Banquet.

In Memoriam
Dr. Larry Young
1967-2024

In late March 2024, the scientific community got the gut-wrenching news of the unexpected passing of Dr. Larry J. Young, William P. Timmie Professor of Psychiatry and Behavioral Sciences at Emory School of Medicine, a mere few hours before he was to kick off the 2024 meeting of the Society for Social Neuroscience (S4SN) in Tsukuba, Japan. With his passing, the meeting now afforded an immediate opportunity to mourn, process, and reminisce, but also to inspire, synthesize, and galvanize the scientific community to achieve a grander scientific vision -- one that Larry would certainly have shared. Larry was a key member of a number of scientific societies such as the S4SN, the Society for Behavioral Neuroendocrinology and a frequent participant in the WCNH. Larry's research has contributed significantly to our understanding of the role of oxytocin and vasopressin on in the regulation of social behavior and he was a member of the organizing committee of this year's WCNH meeting in Atlanta.

Comparative research has been the hallmark of Larry's career. He graduated from University of Georgia in 1989 with a degree in Biochemistry, one of the first in his family from a rural town in the south of Georgia to attend college. Larry was accepted into the graduate program at the University of Texas and worked with Dr. David Crews. After receiving a PhD in Neuroendocrinology in 1994, he switched to studying genes, brain, and behavior in mammals. Although he bid poikilotherms farewell, the combination of cutting-edge research and fun assimilated in the Crews lab and his laser focus on the benefits of studying non-traditional species had become firmly engrained in Larry's scientific psyche.

Larry did his one and only postdoctoral fellowship at Emory University in the Department of Psychiatry and Behavioral Sciences, working with Dr. Thomas Insel. Tom brought with him a prairie vole model developed by the early pioneering work by Dr. Sue Carter. At Emory, Larry worked closely with Dr. Zuoxin Wang and Dr. Jim Winslow on conducting comparative studies with multiple vole species (*Microtus ochrogaster, M. montanus, M. pennsylvanicus*) as well as monogamous marmoset and non-monogamous macaques. Larry was awarded the Frank Beach Award in 1997 for his foundational work on driving molecular approaches to understanding complex social behaviors in naturally occurring species -- being only the second awardee since the founding of the SBN and one of the youngest ever.

In 1996, Larry received his first solo PI grant from NIH, a First Award (R29), and was subsequently appointed as Assistant Professor at Emory. He was only 29 years old, but had the maturity of vision, work ethic, research skills, collaborative spirit, and leadership to rapidly establish his own lab and run with it. In 1999, Larry took on his first three PhD students: Heather Patisaul, Miranda Lim, and Elizabeth Hammock. By the time these first three students defended, his lab was publishing in top tier journals, including *Nature, Science, Neuron, and Journal of Neuroscience*. His PhD students pioneered cutting edge molecular techniques in prairie and meadow voles. By 2005, his lab was exploding at the seams, with a total of six PhD students, two Masters students, and two post-doctoral fellows, Steven Phelps and Darlene Francis. Word was spreading that Larry's brand of science was hot, and Larry himself was proving to be a more than capable mentor whose trainees routinely published rigorous and broad reaching findings.

Larry was a superb mentor. Over his 25+ years as a PI, Larry led an outstanding group of 26 graduate students and 17 postdocs through their journeys of scientific discovery. Larry's trainees are located all over the world, and many have made and are making their own substantial contributions to the field and continue to collaborate scientifically with each other. His trainees all appreciated that Larry was not

only a smart, quick-witted scientist and brilliant experimentalist, but was also a gifted writer, speaker, storyteller, and kindly father figure. Larry's human side is what will be missed most. His down-to-earth demeanor, humility, and gentle awkwardness, as well as lightheartedness and sense of humor, made science approachable and fun. Larry was famous in the lab for his laughing at his own jokes – for those who knew him, his laugh often took the form of snorts and giggles, which were highly infectious. Larry had continuous NIH funding from NIMH for his entire career, and support expanded to include a diversified portfolio that belied the transdisciplinary nature of his works. His belief in team science led to his creation of THREE major research centers in his short career: the Center for Translational Social Neuroscience, the Conti Center, and the Center for Social Neural Networks. He was instrumental in the early founding of the Center for Behavioral Neuroscience, an NSF-funded Center that integrated faculty and trainees across several Atlanta area institutions, including HBCUs. Larry strongly believed in service to the field, contributing to numerous editorial activities including as associated editor on four journals and editorial board member for seven journals.

The impact of Larry's research is reflected in his h-index (111 at the moment) and the total number of citations to his work (nearly 50,000). Larry's work was also of great interest to the lay public and was often featured in outlets such as CNN, NPR, and the BBC to name just a few examples. Media coverage was important to Larry, and he encouraged it not because of his vanity but because he was committed to bringing science into public awareness. This led him to writing a popular book, 'The Chemistry Between Us,' to bring a new understanding of love, sex, and attraction to the lay public. In 2018, Larry was recruited by Rev. Patti Ricotta to visit African teachers, community councilors, clergy and their spouses to campaign against female genital mutilation (FGM). Larry said, "Seeing firsthand how love and bonding research is eliminating harmful cultural practices and improving lives and communities has been life-changing for me". Larry embraced the opportunity to travel far and wide to meet new scientific collaborators and immerse himself in new adventures: from eating massive bugs during a research trip to Madagascar to spending two weeks in India, teaching neuroscience to Tibetan monks.

Larry's life was truly influential to the field of behavioral neuroscience, both in terms of his science and his service. In recognition of his efforts, he was elected Member of the American College of Neuropsychopharmacology, the American College of Neuropsychopharmacology (ACNP), and the American Academy of Arts and Sciences, and Fellow of the American Association for the Advancement of Sciences (AAAS), He was also a terrific role model and great friend for people near him. He mattered greatly to his students, colleagues, friends, his children, and to his loving wife and life partner, Dr. Anne Z. Murphy. Larry would frequently start his talks by showing a picture of him and Anne being married, as a natural segue into a discussion of love and bonding. Larry's communities continue to live on - they are self-sustaining, they serve as a source of joy, worldwide, in our shared mission to conduct social neuroscience research in his generous and collaborative way.

*This is an abridged version of an in Memoriam that will be published in Hormones and Behavior.

'To honor Larry's exceptional and unwavering dedication to mentoring graduate students and junior faculty,' the SBN has established the Larry Young Mentorship Award. Donations to sustain this award can be made at the SBN web site.

Elliott Albers
Regents Professor of Neuroscience
Director, Center for Behavioral Neuroscience
Neuroscience Institute
Georgia State University

Geert de Vries
Regents Professor
Chair, Department of Biology
Georgia State University

Miranda Lim
Associate Professor of Neurology, School of Medicine
Oregon Health & Science University

Zuoxin Wang
Professor, Department of Psychology
Florida State University

In Memoriam
Dr. Harold "Hal" Gainer

I first met Hal in 1981, at the Brattleboro Rat meeting in Dartmouth, organised by Heinz Valtin. It was my first trip across the ocean, and I'd had an eventful 24-hour journey getting there: the coach from Boston dropped me off at a burger joint 5 miles away at 5 in the morning, and after many cups of coffee, a kind waitress persuaded one of the other customers to drop me off at the University. I arrived at my destination just as the registration desk was being set up. Hal was one of the first people I met that early morning – it turned out that we were lodged in neighbouring rooms, and as I'd brought a bottle of whisky and played pool badly we soon established common interests.

Hal was generous with his time and wisdom; he seemed to have no interest in reputations, which was just as well because I had none. This began a friendship that we revived the following year, when Hal gave a plenary lecture at Babraham, at a Conference on the Neurohypophysis (one of the precursors to WCNH), and it was continued at many, many meetings thereafter.

I knew even then that Hal was a pre-eminent biochemist and a pioneer of molecular biology, but I also knew him as an electrophysiologist and comparative physiologist. One of his earliest studies was on phasic firing in a neurone in Aplysia - and, in what I can only think of a mad experiment inspired by a remarkable leap of imagination, he showed that bath application of vasopressin would modify that patterning. This was long before any hint that vasopressin might have a homolog in *Aplysia*, long before vasopressin was understood to be a neuronal messenger, and long before any idea that peptides might be modulators of activity patterns. His account of those early days you can listen to for yourselves here (podcast#24 on https://www.inf-neuroendocrinology.org/podcasts/).

Hal was a constant source of gentle wisdom on all matters neurohypophysial; he was passionate about oxytocin and vasopressin throughout his life. When Covid interrupted normal life, we had a long and lively e-mail discussion in which we delved into the details of papers that we had published 30 years ago on the role of dynorphin in magnocellular neurones, to try to reconcile differences that few others would recognise as more than fine details. But Hal cared about rigor, integrity was critically important, and details mattered.

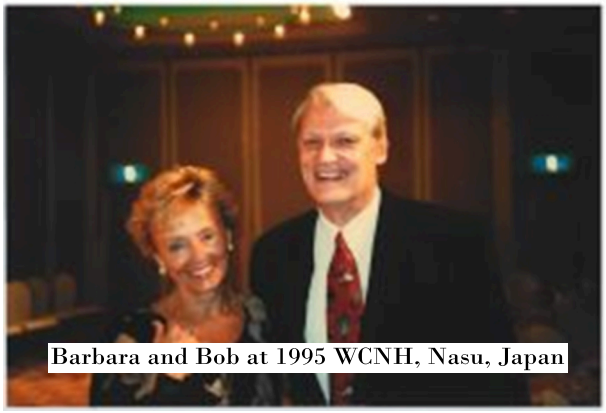
But I remember also his humanity and compassion; on 9/11 we were in Bordeaux at another WCNH meeting, and his quiet wisdom then was humbling.

We who knew him will miss him as no other.

Gareth Leng, Ph.D.
Professor of Experimental Physiology
University of Edinburgh

In Memoriam
Dr. Bob Schrier
1936-2021

When Bob Schrier passed away on January 23, 2021, the world of physiology, medicine and nephrology lost a giant in the field, and I lost a cherished friend. Bob's outstanding scientific, clinical, and academic leadership contributions have been well documented in memorial tributes published in academic journals such as *Kidney International*, *The Journal of the American Society of Nephrology* and *The Lancet*.


Barbara and Bob at 1995 WCNH, Nasu, Japan

In this brief tribute, Joe Verbalis will highlight Bob's groundbreaking clinical contributions. Since Bob is being remembered at this WCNH in Atlanta, I will focus on my personal friendship with him through our mutual interest in vasopressin research and our get-togethers at scientific meetings, many times with his wife, Barbara, over the years since our first encounter at the International Conference on the Neurohypophysis in Key Biscayne in 1976, followed six years later in 1982 by another highly memorable Neurohypophysis meeting at Cambridge University. In 1984, Bob initiated a series of four triennial vasopressin meetings in Aspen (1984), Smuggler's Notch (1987, where I shared accommodations with Bob and his collaborator, Dr. Daniel Bichet), Montpellier (1990), and Berlin (1993). Bob played a pivotal role in the initiation of the biennial WCNH meeting in Nasu, Japan (1995). He became a regular, highly enthusiastic participant in these biennial symposia. I was continually impressed by his amazing ability to summarize the key findings presented at the WCNH meetings in Nasu (1995), Montreal (1997), Edinburgh (1999), Bordeaux (2001), Kyoto (2003), Steamboat Springs (2005) and Regensburg (2007).

Historically, contributions to the human physiology of vasopressin and clinical disorders of water homeostasis have been made by endocrinologists and nephrologists, working on regulation of pituitary vasopressin secretion and end organ effects in the kidney, respectively. Bob's research encompassed both of these important aspects of vasopressin physiology and pathology. In that regard, he was a truly integrative physician scientist in the broadest sense of that term. The expansiveness of his research prowess is reflected in the >1,000 publications that covered acute kidney injury, autosomal dominant polycystic kidney disease, hypertension, diabetic kidney disease, and hormonal control of fluid and electrolytes in cirrhosis, heart failure, nephrotic syndrome and pregnancy. It is the latter for which we in WCNH most remember Bob's signal contributions to our field. He was able to integrate vasopressin into the bigger picture of regulation of body fluid homeostasis in a knowledgeable and understandable evidence-based framework. I have always, and probably always will, cite his landmark paper, "Pathogenesis of sodium and water retention in high-output and low-output cardiac failure, nephrotic syndrome, cirrhosis, and pregnancy". *N Engl J Med* 319:1065, 1988) as a classic example of integrative physiology applied to human pathophysiology. But perhaps of most long-lasting impact, Bob was a dedicated and superb mentor, not only to his own fellows, residents and students, but also to (then) young investigators like me across the globe. He was always eager to listen to new ideas from young investigators, and generously offered his sage advice and support for their research careers. I will be forever indebted to Bob for that support. As recounted by his family, one of Bob's favorite quotes was by George Bernard Shaw: "Some men see things as they are and ask why. Others dream things that never were and ask, why not." Bob epitomized that dream to the lasting benefit of all of us at WCNH.

Bob was one of the brightest and inspiring people we have had the privilege to know. He will be greatly missed but warmly remembered by all who knew, respected, and loved him. We know that he would have been deeply appreciative of having this WCNH symposium named in his honor. Our deepest condolences to his wife, Barbara, and to his immediate and (very large) extended family.

Maurice Manning, Ph.D., D.Sc.
Distinguished University Professor
Cell and Cancer Biology
The University of Toledo Health Science Campus

Joseph G. Verbalis, MD
Professor of Medicine
Chief, Endocrinology and Metabolism
Georgetown Director, Georgetown-Howard Universities Center for Clinical and Translational Science
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Speaker

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