



COGNITIVE NEUROSCIENCE OF  
DEVELOPMENT & AGING CENTER

# CONDA



## CoNDA Center Colloquium

March 11, 2025 | 9:00am - 1:05pm  
UNMC Durham Research Center 1 Auditorium

The **CoNDA Center** is a NIGMS/NIH-supported Center of Biomedical Research Excellence (COBRE) based at the University of Nebraska Medical Center in Omaha.

## Vision

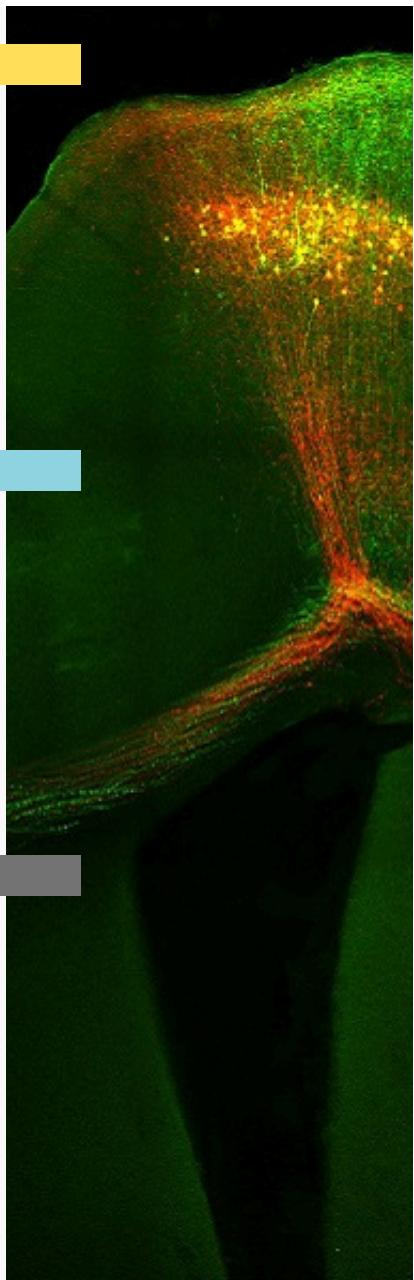
Our vision is to expand translational and clinical neuroscience research in the Omaha region and across the world, and to enhance the ability of current and future generations of local scientists to successfully compete for extramural funding, publish high-impact work and rise to the forefront of the field.

## Mission

The CoNDA Center's mission is to build and strengthen strong research infrastructure for junior investigators to thrive by supporting **Research Projects, Pilot Projects, Research Cores (TIBA and NA2), a Seminar Series, skills workshops, faculty recruitment**, and many more activities.

## CoNDA Campus

Based at the University of Nebraska Medical Center (**UNMC**) in Omaha, Nebraska, the CoNDA Center partners with the University of Nebraska at Omaha (**UNO**) and Creighton University (**CU**) to extend the "CoNDA-Campus" citywide in our quest to advance neuroscience research across the lifespan in our region.



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9:00-9:05am	<b>Welcome</b> CoNDA Center Director: <b>Anna Dunaevsky, PhD</b> Professor, Department of Neurological Sciences (UNMC)
9:05-9:25am	<b><i>The Role of Host Amino Acid Metabolism in Behavioral Changes During Latent Toxoplasmosis</i></b> Research Project Leader: <b>Leonardo da Silva Augusto, PhD</b> Assistant Professor, Department of Pathology, Microbiology, & Immunology (UNMC)
9:25-9:40am	<b><i>The Association between Brain Age and DNA Methylation Age at the Global and the Local Level</i></b> Pilot Project Leader: <b>Jieqiong Wang, PhD</b> Assistant Professor, Department of Neurological Sciences (UNMC)
9:40-10:10am	<b><i>TREM1 as a Novel Therapeutic Target for Global Ischemia Induced Neuroinflammation, Neuronal Death and Cognitive Deficits</i></b> Team Science Project Leaders: <b>Drs. Jee-Yeon Hwang, Holly Stessman, and Gopal Jadav</b> Department of Pharmacology & Neuroscience (Creighton University)
10:10-10:15am	<b>Break</b>
10:15-10:25am	<b><i>MEG Music Study</i></b> CoNDA Core Usage Voucher Awardee: <b>Kuan-Hua Chen, PhD</b> Assistant Professor, Department of Neurological Sciences (UNMC)
10:25-10:40am	<b><i>Impact of KMT5B Expression on Choroid Plexus Development and Macrocephaly</i></b> Pilot Project Leader: <b>Holly Stessman, PhD</b> Associate Professor, Department of Pharmacology & Neuroscience (Creighton University)
10:40-11:00am	<b><i>Hypothalamic Sleep-Wake Neuron Defects in Alzheimer's Disease</i></b> Research Project Leader: <b>Peng Zhong, PhD</b> Assistant Professor, Department Neurological Sciences (UNMC)
11:00am-12:00pm	<b>Poster Session</b> <i>and refreshments - in DRC1 lobby area</i>
12:00-1:00pm	<b>Keynote: Corticostriatal Circuits Underlying Neurodevelopmental Disorders</b> Keynote Seminar Speaker: <b>Ted Abel, PhD</b> Chair, Department of Neuroscience & Pharmacology Director, Iowa Neuroscience Institute
1:00-1:05pm	<b>Conclusion</b> <i>and poster award presentation</i>

1. Padmashri Ragunathan, UNMC. **Effects of Prenatal Alcohol Exposure on the Auditory System.** Co-Author: Stephanie Huntwork (UNMC).
2. Jania Williams, University of Nebraska at Omaha (UNO). **Assistive Shoes Affect Step Characteristics in Patients with Peripheral Artery Disease.** Co-Authors: Farahnaz Fallahtafti, Zahra Salamifar, Sara Myers (UNO), and Iraklis Pipinos (Omaha VA).
3. Luke Hamilton, UNMC. **Effect of *in utero* Oxycodone Exposure and Social Defeat during Adolescence on Neuroinflammation.** Co-Authors: Victoria Schall, Elizabeth Stone, Dalia Moore, Gurudutt Pendyala, and Sowmya Yelamanchili (UNMC).
4. Samantha Johnston, UNMC. **Implementation of a Brief Autism Assessment Model Incorporating EarliPoint (TM).**
5. Zahra Salamifar, UNO. **Walking with Assistive Shoes Enhanced the Vertical Ground Reaction Forces in Patients with Peripheral Artery Disease.** Co-Authors: Farahnaz Fallahtafti (UNO), Iraklis Pipinos (Omaha VA; UNMC), Jason Johanning (Omaha VA; UNMC), Hafizure Rahman (UNO; Omaha VA), and Sara Myers (UNO; Omaha VA).
6. Shaylah McCool, UNMC. **Microglia Morphology and Phagocytic Activation in Visual Brain Regions of the 5XFAD Mouse.** Co-Authors: Jennie Smith and Matthew Van Hook (UNMC).
7. Oleg Korzyukov, UNMC. **Effect of Healthy Aging on Auditory Predictive Processing.** Co-Authors: Valentina Gumenyuk and Sheridan Parker (UNMC).
8. David Ellis and Michele Aizenberg, UNMC. **Longitudinal Assessment of Brain Connectivity in High Grade Glioma Patients Through Tumor Recurrence.**
9. Jenny Thompson, UNMC. **Once a Pruner, Always a Pruner: Re-evaluating the Innate Role of C1q at Retinogeniculate Synapses in Neurodegenerative Contexts, Complements of Vision Loss.** Co-Authors: Matthew Van Hook (UNMC).
10. Ruobing Liu, UNMC. **Enhanced Identification of Brain Network Biomarkers in Alcohol Use Disorder Using Random Projection: A Machine Learning Approach.** Co-Authors: Lei Li, Ziyang Xu, Shibiao Wan, Jennifer Blackford, and Jieqiong Wang (UNMC).

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11. Karla Lynch and Melissa Hatch, UNMC. **Longitudinal Analysis of Driving Behavior and Cognitive Performance in Older Adults.** Co-Authors: Jun Ha Chang, Elizabeth Vlock, and Matthew Rizzo (UNMC).

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12. Gabriel Gauthier, UNMC. **Probing Metabolites in Antiretroviral and Nicotine Treated Mouse Brains using MRS and CEST-MRI.** Co-Authors: Aditya Bade, Balasrinivasa Sajja, Mariano Uberti, Micah Summerlin, Emma Foster, and Yutong Liu (UNMC).

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13. Ziyang Xu, UNMC. **The Triple Network Model of Cocaine Use Disorder: Default Mode Network, Central Exclusive Network, and Salience Network.** Co-Authors: Lie Li, Shibiao Wan, and Jieqiong Wang (UNMC).

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14. Ziyang Xu, UNMC. **Automated Segmentation of the Bed Nucleus of the Stria Terminalis (BNST): A Deep Learning-Based Method.** Co-Authors: Mohammed Azzam, Marshall Bivens, Marcus Dustin, Shibiao Wan, Jennifer Blackford, and Jieqiong Wang (UNMC).

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15. Meghan Ramirez, UNMC. **Investigating brain activity and relational memory in typically developing periadolescent children using task-based fMRI.** Co-Authors: Connor Phipps, Abi Heller-Wight, Anna Wilhelm, and David Warren (UNMC).

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16. Alex Anding, UNMC. **Investigating the role of astrocytes in deficient sleep in Fragile X syndrome.** Co-Authors: Padmashri Ragunathan, Lara Bergdolt, Peng Zhong, and Anna Dunaevsky (UNMC).

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17. Lara Bergdolt, UNMC. **Astrocytic contribution to auditory hypersensitivity in a mouse model of Fragile X syndrome.** Co-Authors: Ember Eldridge, Olga Taraschenko, Padmashri Ragunathan, and Anna Dunaevsky (UNMC).

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18. Jennifer Sexton, UNMC. **Relational Memory Across the Lifespan: Time, Space, and Association from 8-80 Years Old.** Co-Authors: Abi Heller-Wight, Connor Phipps, Meghan Ramirez, Anna Wilhelm, Emma Armbruster, and David Warren (UNMC).

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19. Anna Wilhelm, UNMC. **Hippocampal subfield volumes and relational memory in periadolescent children: findings from the PRANK study.** Co-Authors: Meghan Ramirez, Abi Heller-Wight, Jennifer Sexton, Emma Armbruster, Connor Phipps, and David Warren (UNMC).

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20. Emma Armbruster, UNMC. **Associations between sex and age with anterior and posterior hippocampal subfield volumes in periadolescent children.** Co-Authors: Meghan Ramirez, Connor Phipps, Abi Heller-Wight, Jennifer Sexton, Anna Wilhelm, and David Warren (UNMC).

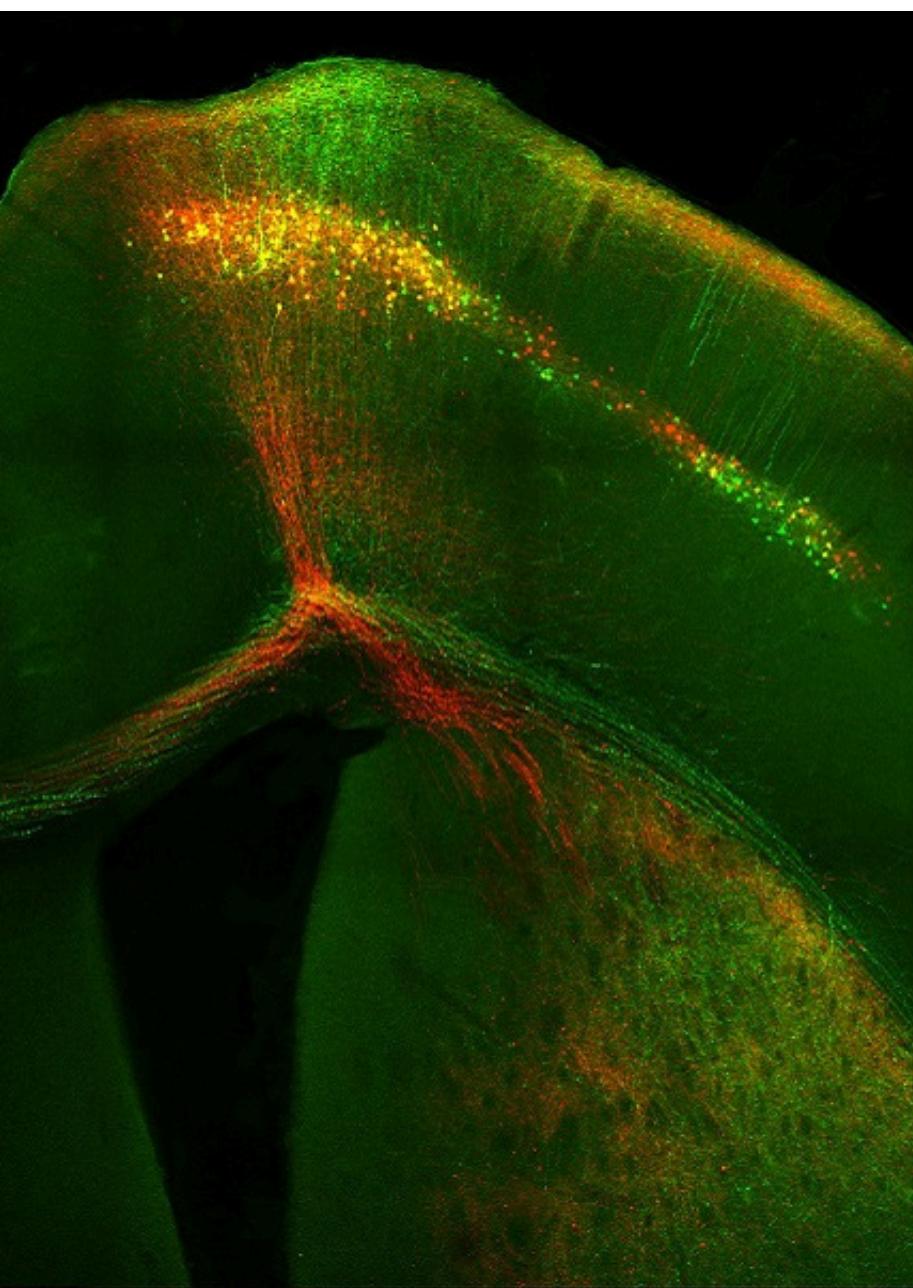
## Ted Abel, PhD

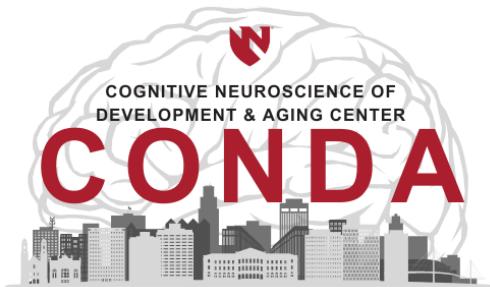


Edwin G. (Ted) Abel, PhD is the founding director of the Iowa Neuroscience Institute, an interdisciplinary center at the University of Iowa focused on the causes, treatments, and prevention of diseases that affect the brain and nervous system. He also chairs the Department of Neuroscience and Pharmacology at Iowa. Abel is recognized as a pioneer in defining the molecular mechanisms of long-term memory storage and in identifying how these processes go awry in neurodevelopmental and psychiatric disorders. He uses molecular and genetic approaches to define how neural circuits mediate behavior, including identifying the molecular impact of sleep deprivation on neuronal function and revealing epigenetic mechanisms that mediate memory.

## *Corticostriatal Circuits Underlying Neurodevelopmental Disorders*

**Abstract:** Neurodevelopmental disorders, including autism spectrum disorders, are highly male biased, but the underpinnings of this are unknown. Striatal dysfunction has been strongly implicated in the pathophysiology of neurodevelopmental disorders (Evans et al, 2023, Biol. Psychiatry), raising the question of whether there are sex differences in how the striatum is impacted by genetic risk factors linked to neurodevelopmental disorders. We have identified male-specific deficits in striatal function important to reward learning in a mouse model of 16p11.2 hemideletion, a genetic mutation that is strongly associated with the risk of neurodevelopmental disorders, particularly autism and attention-deficit hyperactivity disorder. We find that male, but not female, 16p11.2 deletion animals show impairments in reward-directed learning and maintaining motivation to work for rewards (Grissom et al., 2018, Mol. Psychiatry). In on-going work, we are investigating the specific role of neuronal circuits within the corticostriatal network in these reward learning deficits as well as in hyperactivity, which we observe in 16p11.2 deletion mice. The critical genes among the 27 genes in the 16p11.2 region that underlie these phenotypes remain unknown. Using a novel strategy to identify candidate genes associated with the sex-specific phenotypes of 16p11.2 del/+ mice we highlighted three genes within the deleted region: thousand and one amino acid protein kinase 2 (Taok2), seizure-related 6 homolog-like 2 (Sez6l2), and major vault protein (Mvp). Using CRISPR/Cas9, we generated mice carrying null mutations in Taok2, Sez6l2, and Mvp (3 gene hemideletion (3g del/+)). Hemi-deletion of these 3 genes recapitulates sex-specific behavioral alterations in striatum-dependent behavioral tasks observed in 16p11.2 del/+ mice, specifically male-specific hyperactivity and impaired motivation for reward seeking (Kim et al., 2024, Mol. Psychiatry). Moreover, RNAseq analysis revealed that 3g del/+ mice exhibit gene expression changes in the striatum similar to 16p11.2 del/+ mice exclusively in males. This work reveals the critical role of corticostriatal circuits in neurodevelopmental disorders and indicate profound sex differences in the impact of a genetic lesion linked with neurodevelopmental disorders, including mechanisms of male-specific vulnerability and female-specific resilience impacting intracellular signaling in the brain.





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research/conda/index.html](https://www.unmc.edu/neurologicalsciences/research/conda/index.html)

