Pitt Psychiatry
Annual Research Day

June 12, 2025 | 8:15am – 4:00pm
Soldiers & Sailors Memorial Hall | University of Pittsburgh Oakland Campus

Program Schedule

Start	End	Description
8:15 AM	9:35 AM	Poster Session I
9:35 AM	*9:55 AM*	*Transition to Poster Session II*
9:55 AM	11:15 PM	Poster Session II
11:15 AM	*11:40 PM*	*Transition to Lunch and Roundtables*
11:40 PM	12:40 PM	Lunch and Roundtables
12:40 PM	*1:10 PM*	*Transition to Auditorium for Afternoon Presentations*
1:10 PM	2:00 PM	Speed Dat(a)ing
2:00 PM	2:30 PM	Fast Pitch Psychiatry
2:30 PM	*2:50 PM*	*Break (light refreshments will be available)*
2:50 PM	3:40 PM	Psychiatry 2025: Where Are We Now? Where Are We Headed?
3:40 PM	4:00 PM	Awards Ceremony

Poster Session I: 8:15am-9:35am

Poster Session II: 9:50am-11:15am

ID #	Location	Session	Last Name	First Nme	Degree(s)	Title
1	Gettysburg Room	I	Adedokun	Jacqueline	BS	Maternal Cognition, Mental Health and Maladaptive Behaviors in Adults with Down Syndrom
2	Gettysburg Room	I	Akintola	Tomiwa	BS	Disparities in Recruitment and Engagement by Race and Parent Sex in Psychiatric Research on Early Risk for Childhood ADHD
3	Gettysburg Room	I	Allen	Erastus	BA, MBA	Enhancing 2P Calcium Imaging via Multi-Feature Spatiotemporal Analysis
4	Gettysburg Room	I	An	Su bin	BS	Impact of Stressful Family Life Events on Perceived Stress of Young People at Clinical High-Risk for Psychosis: Protective Role of Caregiver Relationship
5	Gettysburg Room	I	Annas	Ellen	BS	Sex Hormone Regulation of Prefrontal Cortex Parvalbumin Interneuron Physiology
6	Gettysburg Room	I	Antezana	Ligia	PhD	Characterizing Patterns and Correlates of Nonsuicidal Self-Injury in Autistic Adults
7	Gettysburg Room	I	Arion	Dominique	PhD	Shared transcriptional features of layer 3 pyramidal neurons projecting to the monkey dorsolateral prefrontal cortex
8	Gettysburg Room	I	Arora	Manan	MBBS	Elevated left ventrolateral prefrontal cortical(vlPFC) activity to reward expectancy(RE) is associated with higher mania/hypomania risk: a replication study in 3 independent young adult samples
9	Gettysburg Room	I	Arruda Da Costa E Silva	Sophia	BS	Differential Afferent and Efferent Connectivity Between Caudal and Rostral mPFC
10	Gettysburg Room	I	Asaoka	Yui	PhD	Effects of Ketogenic Diet on Reward Circuitry in Bipolar Disorder: A Preliminary Investigation
11	Gettysburg Room	I	Balogun	Wasiu	PhD	Plasma p-tau217 for early detection of brain amyloid pathology in community-dwelling older adults without cognitive impairment: evidence from three community-based studies
12	Gettysburg Room	I	Bamfo	Alexis	BS	Does It Matter How People Think About Suicide?
13	Gettysburg Room	I	Barko	Kelly	BS, MS	Sex Differences Detected in the Proteome of MDD Subjects
14	Gettysburg Room	I	Bauer Negrini	Guilherme	PhD	Longitudinal progression and harmonization of tau-PET tracers
15	Gettysburg Room	I	Bear	Shlomo		circRNA Derived from Extracellular Vesicles as Potential Biomarkers for Schizophrenia
16	Gettysburg Room	I	Beatty	Abigail	BSE	Contributions of neuronal oscillations and cortical SNR to developmental changes in inhibitory control from adolescence into adulthood
17	Gettysburg Room	I	Bellaver	Bruna	PhD	Head-to-head trajectories of MK6240, Flortaucipir, and plasma p-tau217 as a function of amyloid-β
18	Gettysburg Room	I	Bennett	Charles	PhD	Interactive Voice Response (IVR) Monitoring as an Adjunct to Outpatient Assessment of Suicidal Thoughts and Behaviors
19	Gettysburg Room	I	Berchulski	Mariah	BS	Simultaneous superficial and deep layer calcium imaging in midline cortex with preserved local cytoarchitecture
20	Gettysburg Room	I	Biver	Lizzie		Apathy and Neuroinflammation: A Dopamine-Independent Pathway
21	Gettysburg Room	I	Boito	Gina	BS	The Impact of Selective Serotonin Reuptake Inhibitors on Episodic Memory
22	Gettysburg Room	I	Brammell	Sarah	BS	Mental Health and Service Use Among Cisgender and Sexual and/or Gender Minority Autistic Young Adults
23	Gettysburg Room	I	Brantly	Nathan		Elucidating the roles of motor function and cognitive switching in locomotor switching after stroke
24	Gettysburg Room	I	Brockway	Dakota	PhD	Neuropeptide Modulation of Prefrontal Cortex Circuitry: Insights into VIP Signaling in Alcohol Use Disorder
25	Gettysburg Room	I	Brodnick	Zachary	BS	Dopamine availability and real-time baseline mood symptoms in depressed youth.
26	Gettysburg Room	I	BS	Aswathy	PhD	The Ketogenic Diet Alters Dopaminergic Activity in the Ventral Tegmental Area in a Mouse Model of Bipolar Disorder
27	Gettysburg Room	I	Budinich	Reece	BS	Xylazine Reduces Prefrontal Cortex Inhibition and Prevents Oxycodone Place Preference
28	Hallway A	I	Bustos-Robles	Lucía		Tolerability of a Single Dose of Buprenorphine, Naltrexone, or Placebo in Major Depressive Disorder: Insights from the RAISE Study
29	Hallway A	I	Buzanis	Sophia	BA	Developing a Brief Clinical Pathway for Trauma-Focused Management of Individuals with Trauma Histories Admitted to the Medical Hospital
30	Hallway A	I	Campbell	Beth	BA	Parsing the heterogeneity of multidimensional determinants of suicide risk in depressed older adults: focus on cognition, personality, and social risk factors

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Poster Session II: 9:50am-11:15am

ID #	Location	Session	Last Name	First Nme	Degree(s)	Title
31	Hallway A	I	Campion	Jacques-Yves	MD	Predicting Worry-Related Mental States using Regional Brain Activity with Long Short-Term Memory (LSTM) Recurrent Deep Neural Networks
32	Hallway A	I	Chae	Christopher	BS	Linking Neural Sensitivity to Social Rejection and Acceptance to Daily Depressive Symptoms
33	Hallway A	I	Chaichian	Omeed	BS	Transcranial Focused Ultrasound Neuromodulation in Psychiatry: Main Characteristics, Current Evidence, and Future Directions
34	Hallway A	I	Chamberlain	Britt		Medial orbitofrontal cortex representation of active avoidance and refinement over learning
35	Hallway A	I	Chapa	Danielle	PhD	Exercise Does Not Regulate Affect: An Ecological Momentary Assessment Study of Maladaptive Exercise in Women with Eating Disorders
36	Hallway A	I	Chen	Chang-Le (Charles)	MSc	Periventricular Diffusivity Reflects APOE4-modulated Amyloid Accumulation and Cognitive Impairment in Alzheimer's Continuum
37	Hallway A	I	Cheng	Cynthia	BS	Rapid Eye Movement (REM) Sleep Characteristics in Individuals with First-Episode Psychosis and Healthy Controls
38	Hallway A	I	Choity	Lamia	BS	Elevated p-Tau217 Disrupts Age-Related Resilience to Cognitive Decline in Older Adults
39	Hallway A	I	Christian	Caroline	PhD	Multivariate Trajectory Modeling of Eating Disorder Symptoms across the Perinatal Period
40	Hallway A	I	Chung	Daniel Wonjae	MD, PhD	Computational Modeling of Stimulus-locked and Persistent Gamma Oscillation Regimes Reveals Differential Vulnerability to Schizophrenia-Associated Synaptic Perturbations
41	Hallway A	I	Cole	Becca	BA	Oxycodone dependence alters Mu and Delta opioid receptor regulation of prefrontal cortex inhibitory transmission in a cell type-specific manner
42	Hallway A	I	Conaty	Kayla	BA	Perceived Hearing Loss is Associated with Processing Speed and Executive Functioning Deficits in Older Adults with Treatment-Resistant Late-Life Depression
43	Hallway A	I	Costa	Ana Paula	PhD	Peripheral Biomarkers of Lipid Dysregulation and Inflammation in Anxiety-Related Risk for Alzheimer's Disease and Related Dementias
44	Hallway A	I	Crawford	Amaya		**WITHDRAWN -** Trait Anhedonia Dampens Expectancy Effects While Anxiety Amplifies Reinforcement Induced Mood Responses in Depression
45	Hallway B	I	Crummy	Elizabeth	PhD	Investigating the Neural Substrates of Active Avoidance in the Bed Nucleus of the Stria Terminalis
46	Hallway B	I	D'Aiuto	Leonardo	PhD	Non-canonical Functions of Tau
47	Hallway B	I	Daniel	Joshua	BA	GET ActivE: Testing of a Behavioral Activation App for youth
48	Hallway B	I	Das	Aanika		The Relationship Between Traumatic Life Events and Mood Lability in Adolescents
49	Hallway B	I	Dauginikas	Emalee	MSc	An Explorative Analysis of Sibling's Impact on an Individual's Emotion Regulation
50	Hallway B	I	Deam	Megan	MA	Five-Year Trajectories of Psychotic-Like Experiences: The Influence of Negative Life Events on Screen Use
51	Hallway B	I	Des Ruisseau	Gabrielle	BS	**WITHDRAWN -** Circadian dysregulation and mood outcomes in young people at risk for bipolar disorder
52	Hallway B	I	DeSerio	Jillian	BS	More Than Mom Brain: A Qualitative Exploration of the Experiences of ADHD and Motherhood in Pittsburgh
53	Hallway B	I	Dewhurst	Hannah	BS	The Female Advantage in Verbal Memory Across Learning and Recall of the California Verbal Learning Test
54	Hallway B	I	Dickens	Jacinta	PhD	**WITHDRAWN** - Trajectories of insomnia and loneliness across a 12-week intergenerational dialog-driven intervention
55	Hallway B	I	DiDomenico	Dominique	BS	Trauma Exposure and Related Context Processing Disruptions
56	Hallway B	I	Ding	Xiaoshan (Victoria)		Pilot study to assess alterations in cortical and thalamic excitatory inputs to parvalbumin-expressing interneurons in prefrontal and primary visual cortices of schizophrenia
57	Hallway B	I	Dong	Yiwen	ScM	The association between amyloid and physical activity in a racially diverse cohort of older adults
58	Hallway B	I	Dowling	Kevin	BA	Patterns of Differential Gene Expression and Co-Expression in Layer 3 Pyramidal Neurons Across 3 Regions of the Human Cortical Visuospatial Working Memory Network in Schizophrenia
59	Hallway B	I	Dubovecky	Haley	BS	Personality Determinants of Loneliness Trajectories in Older Adults: Results from a 12-Week Intergenerational Dialog-Driven Intervention

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Poster Session II: 9:50am-11:15am

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60	Hallway B	I	Edery	Israel	BA	Circadian and homeostatic trends in mood and alertness across a 36-hour ultradian protocol in adolescents
61	Hallway B	I	Edmunds	Lylah	MS	How Do Black Moms Racially Socialize Girls vs. Boys?
62	Hallway B	I	Edvardsson	Hollis	MPH, CCRC	Differences in Cannabis Use Motivations Among Sexual and Gender Diverse Individuals Compared to Heterosexual and Cisgender Individuals and Their Associations with Self-Reported Cannabis Use and Problems
63	Hallway B	I	Eken	Hatice Nur	MD	Associations Between Obsessive-Compulsive Personality Disorder Traits and Symptom Dimensions in Obsessive-Compulsive Disorder: A Cross-Sectional Study
64	Hallway B	I	Eldeeb	Safaa	PhD	Variability in suicidal thoughts and behavior among autistic adolescents and adults: Subgroup identification, predictive features, and group differences
65	Hallway B	I	Ellis	Jaime	BS	Borderline personality features and suicidality in children: Examining associations in a high-risk sample
66	Hallway B	I	English	Gabrielle	BS	Social Rejection Enhances Frontal Pole Activity in Adolescents with Musculoskeletal Pain: A Potential Link between the Neural Responses to Social Threat and Physical Pain
67	Hallway B	I	Enwright, III	John	PhD	Transcriptional profiles of somatostatin and parvalbumin interneuron subtypes in the human dorsolateral prefrontal cortex: Implications for schizophrenia
68	Hallway B	I	Estrada	Yadira	BS, MSW	Two Fronts of Trauma: The Differential Effects of Intimate Partner Violence and Community Violence on PTSD in Rural Mexico
69	Hallway B	I	Fabian	Carly	BS	Mechanisms driving binge drinking: alcohol-induced alterations in PFC basket cell function and mGlu5 receptor signaling
70	Hallway B	I	Farinas	Marissa	MS	Plasma vs. serum: which is better for proteomic blood biomarker analysis? Evaluation of the novel NULISA platform
71	Hallway C	I	Feldman	Julia	PhD	The relation between paternal emotion regulation and inconsistent parenting is dependent on maternal emotion regulation
72	Hallway C	I	Felix	Cynthia	MD, MPH	Usefulness of MoCA in detecting preclinical AD
73	Hallway C	I	Fiske	Meghan	BS	Context Processing and the Implications for Mania Risk
74	Hallway C	I	Forbes	Camryn	BS	VGLUT2 knockdown in the VTA reduces acquisition of alcohol self-administration in a sex-specific manner
75	Hallway C	I	Fowler	Lauren	BS	Auditory and Motor Timing Dysfunction in First Episode Psychosis Indexed by Rhythmic Finger Tapping
76	Hallway C	I	Gallagher	Hannah	BS	Small Effects, Large Impact: An Illustration from National Mental Health Data
77	Hallway C	I	Gamwo	Isaac		**WITHDRAWN** - Is Age Really Just a Number? Effect of Age on Responsiveness of Young Adults with ADHD to a Smartphone Intervention
78	Hallway C	I	Gelber	Ashley	BS	Stress and Perceived Support in Parents of Children with and without Autism Spectrum Disorder
79	Hallway C	I	Ghafari	Kimia	BSc	Cell-Type-Specific Synaptic Proteomics in Postmortem Human Cortex via Proximity Labeling and Single-Cell Transcriptomic Integration
80	Hallway C	I	Glinsky	Michaela	BA	Associations between borderline personality disorder, self-other boundaries, and suicide risk in romantic relationships
81	Hallway C	I	Gogola	Alexandra	MS	Implementation of NIA-AA Multilevel Tau Staging for Predicting Tau Accumulation and Cognitive Decline in Non-Demented Individuals
82	Hallway C	I	Grace	Jennifer	MS	Father engagement in obstetrical care: Black fathers' perspective
83	Hallway C	I	Grad-Freilich	Melanie	BS	The role of childhood abuse and neglect on brain function during emotional interference: Implications for depression in adolescence
84	Hallway C	I	Grady	Alek	BS	Integrated functional proteomics nominate key phosphoprotein-regulatory nodes essential for synaptic function in Schizophrenia
85	Hallway C	I	Griffith	Julianne	PhD	Anticipatory and consummatory anhedonia in adolescent girls: Associations with daily-life positive affect."
86	Hallway C	I	Griffith	Rebecca	PhD	Longitudinal associations between shared and unique components of executive function and externalizing subdimensions: Findings from the ABCD Study
87	Hallway C	I	Grizzanti	John	PhD	Differential effects of Type II Diabetes Mellitus on plasma biomarkers in an Alzheimer's disease cohort: a large memory clinic study
88	Hall of Valor	I	Gu *(with Vivian Zhang)*	Jeremy	BS	NIH Toolbox Cognition Battery: Associations with plasma and imaging AD biomarkers in older adults without dementia

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Poster Session II: 9:50am-11:15am

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89	Hall of Valor	I	Hafenbreidel	Madalyn	PhD	Examining mechanisms of multiple memory encoding of cocaine- and fear-associated memories
90	Hall of Valor	I	Hall	Andrea	MD	Simplifying the risk discussion of antidepressant use in pregnancy, a prototype web-based learning module
91	Hall of Valor	I	Harris	Nicholas	MD, PhD	**WITHDRAWN** - Positive child experiences may mitigate associations between early life adversity and neural stress reactivity in the central visceral circuit
92	Hall of Valor	I	Harvie	V. Blair	BS	Breakfast skipping among children: associations with body mass index, food insecurity, and cognitive function
93	Hall of Valor	I	Ho	Kirsten	BS	Depressive Symptoms and Global Cognitive Functioning in Adults with Down Syndrome
94	Hall of Valor	I	Horan	Nicole	BS	The Barrier Has A Bedtime: Circadian Control of the Blood Brain Barrier in the Nucleus Accumbens
95	Hall of Valor	I	Horter	Chloe	BS	Exploring Dopamine Availability and Smartphone Derived GPS Patterns in Young People with Depression
96	Hall of Valor	I	Hudson	Emma	MA	Greater Stress Response Network (SRN) Connectivity is Associated with Higher Worry in Men and Women in Late Life
97	Hall of Valor	I	Ilina	Karolina	BS	A Social Support Online Intervention for Sexual and Gender Minority Youth to Increase Help-seeking for Anxiety and/or Depression: Pilot Randomized Controlled Trial
98	Hall of Valor	I	Izydorczak	Alexandra	PhD	Development of an Immunoprecipitation Mass Spectrometry method for Tau Peptides in Plasma for Alzheimer's Disease Diagnosis
99	Hall of Valor	I	Janecek	Michael	BA	Elevated dopamine signaling in the NAc of Shank3B-/- pups during maternal interaction.
100	Hall of Valor	I	Janssen	Sabine	BS	Descrying phasic and tonic REM sleep, from healthy control's EEG oscillatory activity, with no significant cognitive correlations
101	Hall of Valor	I	Jo	Alex	BS	Perceived Barriers and Facilitators to an Integrated Treatment for Insomnia and PTSD Symptoms in Women: a Qualitative Analysis
102	Hall of Valor	I	Johnston	Amanda	BS, BA	Public assistance as a risk factor for brain sequelae of chronic trauma
103	Hall of Valor	I	Joshi	Ila Abhijeet		μ-Opioid Modulation of Expectancy-Mood Dynamics During Acute Antidepressant Placebo Effects
104	Hall of Valor	I	Jouppi	Riley	MS	Exploring descriptives and correlates of distress associated with loss of control eating across the perinatal period
105	Hall of Valor	I	Joyce	Karla	MSW, LCSW	Benefits of Comprehensive Clinical Internship Programs
106	Hall of Valor	I	Julien	Megan	BHA	Changes in Anhedonia and Depression with a Single Ketamine Infusion in Youth with Depression
107	Hall of Valor	I	Kaminsky	Mariya	PhD	Ketogenic Diet as Potential Treatment for Bipolar Disorder
108	Hall of Valor	I	Kass	Judah		Parental Acceptance and Rejection: Examining Its Impact on Sexual and Gender Minority Youth Depression Severity
109	Hall of Valor	I	Kastner	Megan (Memphis)	BS	Mental Health Trends and Demographic Insights in STEAM Peer Support
110	Hall of Valor	I	Kavanagh	Jack	M.Phil	Investigating Auditory Segmentation Deficits in the Cingulate Motor Area of First Episode Psychosis
111	Hall of Valor	I	Keller	Lauren	BS	Melanopsin-Driven Light Responsivity and Reward Motivation in Young People at Risk for Mania
112	Hall of Valor	I	Kharade	Ameya		Predicting age using resting state connectomes with deep curriculum based learning
113	Hall of Valor	I	Kinkel-Ram	Shruti	MA	An Intersectional Examination of Weight and Gender Identity-Based Minority Stress on Depression Symptoms among Gender Minority Youth
114	Hall of Valor		Ko	Mei-Chuan (Holden)	PhD	Does a highly G protein-biased mu opioid receptor agonist have an improved therapeutic profile?
115	Gettysburg Room	II	Koganti	Sannidhi	BSA	Moderating Effects of Working Memory Capacity and Internalizing Symptoms on the Relationship Between Age and Emotional Interference Resistance
116	Gettysburg Room	II	Krishna	Maya	BS	Do Reasons for Living Buffer Suicide Risk Equally? A Race-Moderated Analysis
117	Gettysburg Room	II	Ku	Shih-Hsuan (Tiffany)	MS	Exploring the Role of OMGp Signaling in Dendritic Development
118	Gettysburg Room	II	Laifer	Lauren	MA	Associations between chronicity and severity of preconception stress exposure and maternal HPA-axis reactivity during pregnancy
119	Gettysburg Room	II	Langer	Beatrice	BS	Development of a Social Trust Paradigm to Measure Strategic Coaxing

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Poster Session II: 9:50am-11:15am

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120	Gettysburg Room	II	Lee	You-Rim	PhD	Potential of dried plasma spot: a comparative study of plasma biomarker quantification using NULISA
121	Gettysburg Room	II	Leon-Romero	Alejandro	BA	Leveraging the Functionality of RShiny and REDCap to Optimize Study Operations in Clinical Trial Research
122	Gettysburg Room	II	Lewis	Madison	BS	AI-based Region-of-Interest Selection for Schizophrenia
123	Gettysburg Room	II	Lukasewicz Ferreira	Pamela	PhD	Head-to-head association of plasma p-tau217 with MK6240, Flortaucipir, PI2620, and RO948 tau PET tracers
124	Gettysburg Room	II	Luo	Weiquan		Evaluation of image processing methods on biological relationships with tau burden for multisite cross-sectional and longitudinal studies of 18F-Flortaucipir PET
125	Gettysburg Room	II	Lussier	Firoza	MS	Longitudinal multicenter head-to-head harmonization of tau-PET tracers
126	Gettysburg Room	II	Mabry	Samuel	PhD	The Combined Roles of Vesicular Release and Dopamine Reverse Transport on the Psychostimulant Properties of Amphetamine
127	Gettysburg Room	II	Magee	Kelsey	PhD	Patterns of stability and change in pregnancy-to-postpartum depressive symptoms among first-time mothers
128	Gettysburg Room	II	Maier	Matthew	BS	Cortical Layer-Specific Alterations in Schizophrenia: Evidence for Inflammation and Elevated ZFP36
129	Gettysburg Room	II	Manna	Lillian	BS	Can We Distinguish Types of Suicidal Behavior? Examining the Role of Impulsivity, Physical Aggression, and Emotion Regulation
130	Gettysburg Room	II	Mannion	Katherine	BS	Feasibility of actigraphy and sleep diary collection in preschool-aged children with behavioral and sleep difficulties
131	Gettysburg Room	II	Marowski	Megan	BS	Stress and Conflict in Borderline Personality Disorder: The Protective Role of Agreeableness
132	Gettysburg Room	II	Mayorga	Lynnea	BA	Preliminary analysis on the role of physiological stress reactivity in mediating the relationship between puberty and anxiety symptoms
133	Gettysburg Room	II	McCarty	Erin		The relationship between Subjective Cognitive Decline and Objective Cognitive Performance in older adults with Treatment-Resistant Late-life Depression: Role of Depression Severity as a Mediator
134	Gettysburg Room	II	McCathern	Ali	MD	Restructuring psychotherapy didactic for medical student psychiatry clerkship
135	Gettysburg Room	II	McDonald	Nastasia	PhD	Oops!: Error-Related Negativity as a Neural Correlate of Chronic Health Stress in Adolescents Enriched for Depression Risk
136	Gettysburg Room	II	McDonald	Nicholas	BS	Closed-Loop Respiration-Timed Optogenetic Stimulation in Mice using Real-Time Forecasting
137	Gettysburg Room	II	McKeon	Shane	PhD	Intrinsic neural timescales decrease through adolescence into adulthood supporting cognitive development
138	Gettysburg Room	II	Mehalko	Jordan	MSCP	Adolescent Feedback on a Suite of Mobile Suicide Prevention Tools: Integrated Care to Help at Risk Teens (iCHART)
139	Gettysburg Room	II	Melchitzky	Darlene	MS	The Arrangement of Synapses in Layer 3 of Human Prefrontal Cortex
140	Gettysburg Room	II	Miller	Nora	BS	Discrimination of threat vs. non-threat stimuli is differentially regulated by rostral and caudal medial prefrontal cortex
141	Gettysburg Room	II	Mirchandaney	Riya	BA	Circadian preference, but not circadian phase, associates with state and trait levels of impulsivity in adolescents
142	Hallway A	II	Mizuno	Akiko	PhD	Using Natural Language Processing to Identify Reflections on Late-Life Loneliness After an Intergenerational Dialogue-Driven Intervention
143	Hallway A	II	Monto	Abdul Razak	PhD	Comparative evaluation of Blood Collection Tubes on Targeted Proteomic Profiles of Alzheimer’s Disease Plasma Biomarkers
144	Hallway A	II	Mossazghi	Nahom	BS, MS	The neural basis of cognitive deficits in adults with sickle cell disease: a task-based fMRI study
145	Hallway A	II	Mroué	Rayan	MD	Different Patterns of Propagation of Tau Tangle Pathology in Typical Alzheimer's Disease Determine Clinical Sub-Phenotypes
146	Hallway A	II	Myers	Teneisha	MS	Pharmacokinetic profiling of ∆9-THC metabolism and its association with cognitive impairment and modulation by stress
147	Hallway A	II	Nafash	Michel	BS	Analytical Validation of BD-tau Advantage Plus Kit with Clinical Corroboration in a Pilot Traumatic Brain Injury Cohort.
148	Hallway A	II	Niggemyer	Michael	BS	The Role of Puberty on Neural Activity to Reward Feedback in Early Adolescence
149	Hallway A	II	Nizam	Nawshad Binta		Cross-Species Mapping of Human and Mouse Medial Prefrontal Cortex Using Spatial Transcriptomics

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Poster Session II: 9:50am-11:15am

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150	Hallway A	II	Nooraeen	Sara	MD	Neural Responses to Social Rejection in Adolescents with Musculoskeletal Pain: Preliminary Findings of Altered Processing of Social Threat
151	Hallway A	II	Novacich	Isabel	BS	Cognitive Function and Depression in Adolescents Under Chronic Stress
152	Hallway A	II	O'Rourke	Ella	BS	Independent validation of regional Biochemical Markers of Neuropil Contraction in Early-Onset Schizophrenia Identified by 7T ^{31}P MRS
153	Hallway A	II	Ojha	Amar	BA	Developmental trajectories of prefrontal – nucleus accumbens subcircuits support cognitive and affective control across adolescence
154	Hallway A	II	Ouyang	Bowei	PhD	Brain Age Estimation Using Deep Learning on High-Resolution MRI
155	Hallway A	II	Pan	Yiyan		Women have Greater Tortuosity of Internal Carotid Artery Compared to Men
156	Hallway A	II	Pangburn	Mary		Periventricular White Matter Diffusivity as a Mediator Between Metabolic Syndrome Components and Cognitive Impairment
157	Hallway A	II	Papale	Andrew	PhD	Age and Sex Differences in Exploration and Related Representations in Ventral Prefrontal Cortex and Hippocampus from Adolescence to Adulthood
158	Hallway A	II	Parr	Ashley Clare	PhD	Substance use trajectories relate to variation in impulsivity, inhibitory control, and tissue iron indices of dopamine neurobiology during the transition from adolescence to adulthood
159	Hallway B	II	Pearcy	Leigh	PhD	Longitudinal changes in white matter hypointensities in recurrent late-life depression
160	Hallway B	II	Perez	Megan	BS	Gene Splicing Differences in Psychosis in the Striatum
161	Hallway B	II	Petersen	Kaitlyn	PhD	Adolescent circadian rhythm disruption leads to increased risk-taking and transcriptional changes in adulthood
162	Hallway B	II	Petrie	Daniel	PhD	Developmental trajectories of reward, goal-directed, and habitual brain circuits are differentially linked to alcohol use
163	Hallway B	II	Pierson	Jamie	PhD	Evaluation of Compulsive and Anxiety-Like Behaviors in a Heterozygous Global Slitrk5 Knockout Mouse Model
164	Hallway B	II	Ponce	Jacob	BS	Psychological Resilience as a Moderator of Cognitive Reserve: An Integrative Neuroimaging Study
165	Hallway B	II	Povala	Guilherme	PhD	Harmonization of Flortaucipir, MK6240, PI2620 and RO948 with the Uni scale
166	Hallway B	II	Raeder	Robert	MSc, MA	Pre-Supplementary Motor Area Activity During Reward Expectancy Linked to Mania/Hypomania Risk
167	Hallway B	II	Raminfard	Samira	PhD	Diffusion-Derived Subcortical Microstructural Changes Associated with Clozapine Response in Treatment-Resistant Schizophrenia
168	Hallway B	II	Rapp	Ellie	BA	The Association Between Lifetime Suicide Risk and Cognitive Function Among Older Adults with Treatment Resistant Depression
169	Hallway B	II	Regal	Abigale	BS	The Impact of Depressive Symptoms and Disorders on Breastfeeding Intent and Duration
170	Hallway B	II	Ren	Yuxin	BS	Scanner effects in longitudinal tau-PET imaging studies of Alzheimer's Disease
171	Hallway B	II	Renuka Sanotra	Monika	PhD	Combining p-tau217 with Other Blood Biomarkers to Enhance Prediction of Cognitive Decline: A Large Memory Clinic Cohort Study
172	Hallway B	II	Rhorer	Hayley	BA	Differential Auditory Segmentation Potentials in First-Episode Psychosis: Active vs Passive Attention
173	Hallway B	II	Roberts	Anna		Altered resting state hippocampal connectivity associated with amyloid and tau in older adults without dementia from a population-based cohort study
174	Hallway B	II	Rose	Morgan	BS	Collaborating to create a "Roadmap to ETUDES": Human-centered design informs study materials to engage families in suicide prevention research
175	Hallway B	II	Ruppert	Emma	MD	Harmonizing visual reads of tau PET tracers - HEAD cohort
176	Hallway B	II	Russell	Emily	BS	Contextual Influences on Emotion Socialization: An Examination of the Current Framework and Future Directions
177	Hallway B	II	Saha	Pampa	PhD	Association of plasma GFAP with tau PET in cognitively unimpaired Aβ-negative subjects
178	Hallway B	II	Sanchez Montenegro	Catalina	BA	Maternal Depression and Dyadic Neural Synchrony: The Moderating Role of Maternal Positive Affect

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Poster Session II: 9:50am-11:15am

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179	Hallway B	II	Saunders	August	BA	Exploring the Intersection of Emotion Dysregulation and Intervention Use in Autistic Children
180	Hallway B	II	Scarpatto Rodrigues	Matheus	PhD	Effects of AD modifiable risk factors to tau-pet tracer uptake and its association with cognition in early Braak stages
181	Hallway B	II	Schmitt	Tylar	BA	A Scoping Review of Cultural Adaptation Frameworks for Digital Mental Health and Substance Use Interventions
182	Hallway B	II	Scop Medeiros	Marina	MD	Comparison of MK-6240 and Flortaucipir tau PET for the biological staging of Alzheimer disease
183	Hallway B	II	Scott	Madeline	PhD	Age dependent changes in 24 hour gene expression rhythms across cells of the human dorsolateral prefrontal cortex
184	Hallway B	II	Scullin	Keeley	BS	Distinct Clinical and Neuroimaging Profiles by Amyloid Status in Mild Cognitive Impairment
185	Hallway C	II	Seah	Stanley	PhD	Day-to-Day Sleep Quality Moderates the Link between Social Stress and Suicidal Ideation among High-Risk Sexual and Gender Diverse Young Adults
186	Hallway C	II	Seebold	Dylan	BS	Melody and Rhythmicity Perception Deficits in First-Episode Psychosis
187	Hallway C	II	Sehrawat	Anuradha	PhD	Equivalence of plasma and serum for clinical measurement of p-tau217: comparative analyses of four blood-based assays
188	Hallway C	II	Shellhause	Karoline	BS	Neuromodulatory effects of bright light on threat and reward network metabolism in depressed adults
189	Hallway C	II	Shelton	Micah	MS	Dissecting Cortical Layer and Sex-Specific Transcriptional Differences within the Subgenual Anterior Cingulate Cortex in Major Depressive Disorder
190	Hallway C	II	Shih	Yi-Chun	MS	Early Postnatal Dysfunction of ACC PV Interneurons in Shank3B-/- Mice
191	Hallway C	II	Silva da Rocha	Andreia	PhD	Head-to-head comparison of MK6240 and Flortaucipir PET tracers for in vivo Braak staging
192	Hallway C	II	Silva do Amaral	Livia	MSc	Comparison of Topographical Patterns of Abnormalities of the Tau PET Tracers [18F]Flortaucipir, [18F]MK6240, [18F]PI2620, and [18F]RO948
193	Hallway C	II	Silva Oliveira, Jr.	Markley	PhD	Tau-phosphorylation and Oligodendrocyte Dysfunction in Alzheimer's Disease
194	Hallway C	II	Singer	Juli	MS	Clinical Utility of Plasma Biomarkers in Alzheimer's Disease (CliPAD)
195	Hallway C	II	Singh	Maya	MSc	**WITHDRAWN:** Predicting Postpartum Depression: A Data-Driven Approach to Early Risk Screening
196	Hallway C	II	Sinrich	Jacob		Brain Age in Autism: identifying factors associated with accelerated aging
197	Hallway C	II	Snider	Isabella	BS	High Fidelity Wraparound's Positive Effects on Daily Functioning, Living Satisfaction, and Caregiver Strain
198	Hallway C	II	Snyder	Ian	BS	Neuromodulation of Antidepressant Placebo Effects: A TBS Study
199	Hallway C	II	Soares	Carolina	PhD	Profiling Amyloid-Negative, Tau-Positive Individuals with Two Tau PET Tracers – The HEAD study
200	Hallway C	II	Son	Haeun	BS	Parvalbumin interneuron diversity in mouse visual and prefrontal cortices
201	Hallway C	II	Springer	Shale	BS	Altered protein expression and phosphorylation in higher-order thalamic nuclei in Obsessive-Compulsive Disorder
202	Hall of Valor	II	Stein	Dylan		Toddler Behavior and Preschool ADHD Outcomes among Children at High and Low Familial Risk of ADHD
203	Hall of Valor	II	Stewart	Holly	BS	Participant performance factors and improvement in depressive symptoms following real-time fMRI amygdala neurofeedback training
204	Hall of Valor	II	Stowe	Taylor Ashley	PhD	Diurnal Rhythms Underlying Cholinergic Interneurons May Mediate Reward-Related Behaviors
205	Hall of Valor	II	Su	Derica	BA	Brain Network Activity in Autistic and Non-Autistic Adults Thinking About Preferred Interests
206	Hall of Valor	II	Syta	Juliette	BS	Belief-updating computations underlying repetitive negative thinking in late life
207	Hall of Valor	II	Taglioni	Laura	BA	Trust, But Take: Individual Differences in Reward Sensitivity Influence Strategic Exploitation during a Social Exchange Game
208	Hall of Valor	II	Taraban	Lindsay	PhD	Maternal Parenting-Related Confidence is Associated with Neural Co-Regulation among Mother-Infant Dyads
209	Hall of Valor	II	Teixeira Leffa	Douglas	MD, PhD	ADHD Genetic Risk and Cognitive Decline in Older Adults: Findings from the Alzheimer's Disease Sequencing Project

Poster Session I: 8:15am-9:35am

Poster Session II: 9:50am-11:15am

ID #	Location	Session	Last Name	First Nme	Degree(s)	Title
210	Hall of Valor	II	Theis	Nicholas	MS	Brain Energy States are Diagnostically Distinct & Capture Neural Dynamics Better than Regional Activation and Connectivity
211	Hall of Valor	II	Thomas	Jacky	BS	Navigating Sexual Orientation Diversity: Investigating the Impact of Mental Health on Academic Success Amongst New College Students
212	Hall of Valor	II	Tomlinson	Claire	PhD	Meta-Analysis on the Effectiveness of mHealth Interventions for Mental Health: A 10-Year Update
213	Hall of Valor	II	Vaughan	Dylan	BS	Melanin-concentrating hormone reduces learned helplessness in male mice and modulates layer 2/3 medial prefrontal cortex neuron properties
214	Hall of Valor	II	Verma	Piya		Adolescent specific effects of cumulative lifetime stress on affective impulsivity
215	Hall of Valor	II	Verone	Kate		Predicting Biological Age from Brain MRI with Deep Learning
216	Hall of Valor	II	Wang	Linghai		A Computational Approach to Examining Performance Perception
217	Hall of Valor	II	Wang	Melanie	BS	Emotional Brain Responses during Light Exposure in Adults with Depression
218	Hall of Valor	II	Weaver	Shantele	BS, MSCP	**WITHDRAWN** Optimizing Suicide Prevention Strategies for Pediatric Primary Care Through End-User Feedback
219	Hall of Valor	II	Westbrook	Ceci	MD, PhD	Identifying Brain Signatures of Worry Among Adolescents And Adults: A Multivariate Pattern Analysis Approach
220	Hall of Valor	II	Wilson	Michelle	BS	The MomMA program: A novel behavioral intervention for ADHD in pregnancy
221	Hall of Valor	II	Win	Emma	MSW	Detecting and managing fraudulent participation in a clinical trial
222	Hall of Valor	II	Wong	Meghan	MS	Bereavement Overload and its Association with Psychological Distress among Physicians-in-Training at UPMC
223	Hall of Valor	II	Wright	Destiny	BS	Developmental Trajectories of Hippocampal Glutamate & GABA
224	Hall of Valor	II	Yau	Stephanie		Nanoscale probing of synaptic architecture in human prefrontal cortex with expansion microscopy
225	Hall of Valor	II	Yeoum	Joshua	BS	Comparing Circadian Preference and Self-Reported Sleep Quality in Retired Night Shift Workers and Retired Day Workers
226	Hall of Valor	II	Yi	Sophia		Auditory processing deficits of dual-rule complex MMN in first episode psychosis
227	Hall of Valor	II	Yoblinski	Andrew	BS	Behavioral Effects of a Novel Antidepressant in a Mouse Model of Depression
228	Hall of Valor	II	Zeng	Xuemei	PhD	Unveiling Tau Pathogenesis in Alzheimer's Disease: A Label-Free Mass Spectrometry Study of Autopsy-Confirmed Brain Tissues

Presenter Name/Degree(s): Jacqueline Adedokun, BS
Current Position: Research Associate

Primary Mentor in Psychiatry: Benjamin Handen, PhD

Title: **Maternal cognition, mental health and maladaptive behaviors in adults with Down Syndrome**

Author(s): ***Adedokun J[1], Piro-Gambetti B[2], and Handen B[1]***
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]University of Wisconsin-Madison,

Introduction: Mothers of adults with Down syndrome (DS) face an increased risk for Alzheimer's disease and greater mental health challenges compared to mothers of neurotypical children. Research suggests that cognitive functioning and mental health are interconnected, with mood disorders linked to neurocognitive deficits in attention, executive functioning, and memory. Considering a family-wide perspective, research also suggests that maternal mental health is associated with child maladaptive behaviors. This study aims to explore associations between maternal mental health and both the cognitive well-being of the mother as well as the maladaptive behaviors of the child with DS.

Methods: Mothers (n = 111) participated in the MOMs' study, a supplemental study affiliated with the Alzheimer Biomarker Consortium-Down Syndrome in which their adult child with DS participated. Mothers completed questionnaires assessing their own cognition (Ecog2) and mental health (depression: PHQ-9; anxiety: GAD-7; worry: PSWQ). Maladaptive behaviors (Reiss Screen for Maladaptive Behavior) for the adult with DS were assessed via study-partner report.

Results: Pearson correlations revealed that maternal cognition was associated with all three maternal mental health variables (depression: $r = .394$, $p < .001$; anxiety: $r = .353$, $p < .001$; worry: $r = .200$, $p = .035$). The adults with DS' maladaptive behaviors were correlated with maternal depression ($r = .286$, $p = .011$), but not with maternal anxiety ($r = .140$, $p = .220$) or worry ($r = .191$, $p = .094$).

Conclusion: Greater cognitive deficits are associated with greater depression, anxiety, and worry symptoms for mothers of adults with DS. Further, greater maternal depression is associated with greater maladaptive behaviors for the adult with DS. These findings highlight the complex and interconnected nature of mental health and cognition within the family and emphasize a critical need for resources that address both mental health and cognitive functioning for mothers of adults with DS and their family.

Presenter Name/Degree(s): Tomiwa Akintola, BS
Current Position: Psychiatry REACH Fellow

Primary Mentor in Psychiatry: Heather M. Joseph, DO

Title: **Disparities in recruitment and engagement by race and parent sex in psychiatric research on early risk for childhood ADHD**

Author(s): ***Akintola T, Joseph H, and Vaughn-Coaxum R***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Disparities in clinical research participation persist, particularly in psychiatric research where a long-standing history of exclusion, mistrust, and systemic racism continues to shape participation outcomes. These inequities are especially pronounced among Black and racially marginalized communities, who are often underrepresented in both initial engagement and study completion. The current project, supported by the Research Equity and Community Health (REACH) fellowship, builds on emerging efforts to evaluate not only who enrolls, but who stays. By identifying patterns of attrition across racial groups in clinical research, we aim to highlight potential barriers to equitable participation and generate data that can inform more inclusive research practices.

Methods: We analyzed deidentified data from 385 families approached for enrollment in the New(born)PARIS study, including 82 who enrolled and 303 who did not. Data were stratified by race (Black, White, Latino, Multiracial, Asian), recruitment method (website contact vs. in person screening) and parent sex. Descriptive analyses were conducted to evaluate enrollment patterns, recruitment source, and contact intensity.

Results: Screening success rates through in-person screening was higher among Black, Latino, and White families, whether or not they enrolled, except among Asian families, who showed 100% screening success via website contact. White families had the highest enrollment rates overall (50%), while Black families had the lowest (16%). Father engagement ranged from 17% to 65%, with Black fathers showing the lowest engagement in research.

Conclusion: Disparity in successful enrollment across racial groups were identified, and low father engagement was identified as a key barrier to enrollment. Better approaches are needed for engagement in research for underrepresented populations, particularly among Black families and fathers, and tailoring recruitment approaches may be essential for improving representation and reducing attrition in future clinical research.

Presenter Name/Degree(s): Erastus Z. Allen, BA, MBA
Current Position: Research Project Coordinator

Primary Mentor in Psychiatry: Joseph M. Stujenske, MD, PhD

Title: **Enhancing 2P calcium imaging via multi-feature spatiotemporal analysis**
Author(s): ***Allen EZ[1] and Stujenske JM[1,2]***
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Translational Neuroscience Program, University of Pittsburgh School of Medicine

Introduction: Two-photon calcium imaging enables large-scale recording of neuronal activity, but accurate identification of active neurons and extraction of fluorescence traces often depend on time-consuming manual curation, limiting scalability and reproducibility. While pipelines such as CaImAn and Suite2p offer effective solutions, they require careful tuning of hyperparameters, struggle with low signal-to-noise, lack motion correction algorithms robust to slow distortions, and do not robustly separate overlapping cells. Here, we propose a novel set of approaches to overcome these limitations, including a novel dimensionality reduction approach, in which we extract a set of spatiotemporal pixel features to predict pixels containing neurons and neurites prior to cell detection, thus limiting non-cellular detections.

Methods: We compute a diverse array of spatial, temporal, and spectral features across the imaging video, including local pixel correlations, transient kinetics, and frequency-domain signatures. These features form a modular representation that can feed into unsupervised clustering, supervised classification, or hybrid segmentation frameworks. Importantly, this architecture is designed to support feature sharing across experiments and labs—aligning with a ***Feature Store*** methodology emerging in machine learning—to promote collaboration, reproducibility, and cumulative improvement of models over time.

Results: Preliminary evaluations suggest that our approach reduces false detections of neurons across large fields of view, and resolves overlapping ROIs. We demonstrate robust performance across a range of imaging datasets with diverse parameters, and we present preliminary analyses benchmarking against established techniques and extending this methodology to successfully classify inhibitory interneurons.

Conclusion: Our approach is promising for improving throughput of large field of view calcium imaging, which will enable higher throughput applications of the technology for longitudinal tracking neural activity over many sessions.

Presenter Name/Degree(s): Su bin An, BS
Current Position: Undergraduate Student Researcher

Primary Mentor in Psychiatry: Leslie Horton, PhD

Title: **Impact of stressful family life events on perceived stress of young people at clinical high-risk for psychosis: Protective role of caregiver relationship**

Author(s): ***An Sb, Deam M, Griffith J, Gupta T, and Horton L***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Perceived stress is a critical contributor to increasing vulnerability to psychosis, especially in young people at clinical high-risk for psychosis (CHR-P), mainly identified by sub-threshold unusual perceptions, thought content, and disorganized communication. Perceived stress may be related to exposure to stressful life events, which have been linked to the development of psychosis. However, little is known about the specific impact of family-related stressful life events (e.g., death of close family member) among youth at CHR-P. Furthermore, previous studies suggest that young people's relationships with their caregivers may impact their stress levels and psychological outcomes, but few studies have explored whether caregiver relationship quality moderates the impact of stressful family life events on perceived stress in young people with and without CHR-P.

Methods: 32 CHR-P and 20 non-CHR-P controls, aged 13-20, completed self-report questionnaires, including Perceived Stress Scale, Adolescent Life Events Scale, and Social Relationship Index.

Results: The CHR-P group reported significantly more stressful family life events (***p*** < .001), greater perceived stress (***p*** < .001), and lower caregiver relationship quality (***p*** = .002) than controls. Across the full sample, more stressful family life events were associated with greater perceived stress (***p*** < .001), regardless of CHR-P status. In the whole sample, moderate to high relationship quality unexpectedly strengthened, rather than buffered, the positive link between stressful family life events and perceived stress (***p*** < .001).

Conclusion: Young people at CHR-P experienced more stressful family life events, higher perceived stress, and lower caregiver relationship quality than controls. Across the full sample, moderate to high caregiver relationship quality amplified, rather than reduced, the association between stressful family life events and perceived stress. More research in this area has potential to shed light on mechanisms contributing to psychosis risk, as well as negative psychological outcomes in young people, broadly.

Presenter Name/Degree(s): Ellen M. Annas, BS
Current Position: Graduate Student

Primary Mentor in Psychiatry: Max Joffe, PhD

Title: **Sex hormone regulation of prefrontal cortex parvalbumin interneuron physiology**

Author(s): ***Annas E[1,2,3] and Joffe M[1,2,3]***
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Center for Neuroscience, University of Pittsburgh; [3]Translational Neuroscience Program, University of Pittsburgh

Introduction: Sex differences in incidence and pathology are well-documented across many psychiatric disorders, including substance use disorder. Parvalbumin-expressing interneurons (PV-INs) exhibit many sex differences in regulating cognitive and motivated behavior. Previous studies from our group have identified metabotropic glutamate receptors 1 and 5 as a key molecule mediating sex differences in PV-IN function. PV-INs also express estrogen receptors, which functionally couple with $mGlu_{1/5}$ receptors on the cell membrane. Taken together, we hypothesized that circulating gonadal hormones like estradiol (E2) regulate sex differences in $mGlu_{1/5}$ receptor signaling, and therefore in PV-IN physiology. Here, we investigated the physiology of PV-INs in male and female mice with and without E2 present.

Methods: We performed ***ex vivo*** whole-cell electrophysiology in transgenic mice expressing tdTomato in PV-INs, recording from the prelimbic cortex of male and cycling female mice. Estrous stage of female mice used in electrophysiological experiments was determined by vaginal cytology. In a separate experiment, we optogenetically evoked inhibitory post synaptic currents (IPSCs) from PV-INs to pyramidal neurons. Brain slices from each mouse were incubated in either standard artificial cerebrospinal fluid (aCSF) or aCSF containing 300nM E2.

Results: PV-INs showed no significant differences in baseline membrane properties between incubation conditions within each sex. Non-estrus female PV-INs have a higher membrane resistance and lower rheobase when incubated in E2 than in standard aCSF, whereas estrus stage females do not exhibit significant differences between incubation conditions.

Conclusion: These experiments suggest that E2 modulates the excitability and membrane resistance of PFC PV-INs in an estrous cycle stage-dependent manner. Ongoing experiments aim to assess how E2 alters PV-IN-mediated inhibitory transmission onto pyramidal neurons, and utilizing pharmacological manipulations to validate the receptor pathways involved.

Presenter Name/Degree(s): Ligia Antezana, PhD
Current Position: Postdoctoral Scholar

Primary Mentor in Psychiatry: Carla A. Mazefsky, PhD

Title: **Characterizing patterns and correlates of nonsuicidal self-injury in autistic adults**

Author(s): ***Antezana L[1], Feldman JR[2], Manna LL[1], Kumar T[2], Eldeeb S[1], Conner CM[1], Scott LN[1], and Mazefsky CA[1]***
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Psychology, University of Pittsburgh

Introduction: While ~50% of autistic adults report nonsuicidal self-injury (NSSI), it remains understudied despite an association with suicide. In the limited work in autistic people, alexithymia and sensory sensitivities are correlates of NSSI. Interestingly, repetitive behaviors, cognitive rigidity, emotion dysregulation, impulsivity, and social anhedonia have not yet been examined in the context of NSSI in autism. Though these constructs are linked to self-injurious behaviors in autistic samples and/or with NSSI in nonautistic samples. This study aimed to examine group differences and correlates of NSSI in autistic vs. nonautistic adults.

Methods: Data were from the Pitt Autism Center of Excellence (Projected N=300 [Autistic n=200]). To date, 100 participants (Autistic n=75; M(SD) age= 32.96(10.77) years) were administered NSSI questions. Participants completed self-reports, including measures of repetitive behaviors, cognitive rigidity, sensory sensitivity, alexithymia, emotion dysregulation, impulsivity, and social anhedonia. Chi-square tests were used for group differences in history of NSSI. Within the autistic group, hierarchical logistic regressions were used to examine correlates of history of NSSI. Covariates for all regressions included age, sex, and IQ.

Results: Autistic adults were more likely to report history of lifetime NSSI compared to nonautistic adults (64% vs. 24%), $p<.001$. In autistic participants, more repetitive behaviors and worse cognitive reappraisal associated with history of lifetime NSSI, ps<.001. Significant models emerged for 6-month NSSI history, 6-month NSSI frequency, and number of NSSI methods with similar patterns of older age, and more repetitive behaviors associations, ps<0.05. No other models were significant.

Conclusion: Older age and more repetitive behaviors are strongly linked with NSSI in autistic adults. Future work should parse the ways in which repetitive behaviors and NSSI may be intertwined. Uncovering mechanisms related to repetitive behaviors will allow better understanding of the emergence and maintenance of NSSI and may aid in development of targeted NSSI prevention and intervention for autistic people.

Presenter Name/Degree(s): Dominique Arion, PhD
Current Position: Research Principal Sr

Primary Mentor in Psychiatry: David A Lewis, MD

Title: **Shared transcriptional features of layer 3 pyramidal neurons projecting to the monkey dorsolateral prefrontal cortex**

Author(s): ***Arion D[1], Enwright, III JF[1], Krienen FM[3], Gonzalez-Burgos G[1], and Lewis DA[1,2]***
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Neuroscience, University of Pittsburgh; [3]Princeton Neuroscience Institute, Princeton University

Introduction: Layer 3 pyramidal neurons (L3PNs) providing axonal projections to the primate dorsolateral prefrontal cortex (DLPFC) convey essential information for core cognitive processes such as working memory and attention. Thus, alterations in these L3PNs might contribute to the impairment of these processes in schizophrenia. However, it is not known if DLPFC-projecting L3PNs can be identified by a distinctive transcriptional profile.

Methods: We used laser microdissection to capture Nissl-stained L3PNs and retrogradely-labeled L3PNs that project to the DLPFC from the superior temporal (STC) and posterior cingulate (PCC) cortices of adult macaque monkeys and subjected the samples to RNA sequencing. These results were compared to similar data from L3PNs that project to the DLPFC from the posterior parietal cortex (PPC) or contralateral DLPFC, and to single nucleus RNAseq data from multiple cortical regions.

Results: Numerous differentially expressed genes were found between Nissl-stained L3PNs from STC and PCC, but many fewer were present between DLPFC-projecting L3PNs from these regions. Genes enriched in DLPFC-projecting L3PNs from STC and PCC showed concordant transcriptional profiles with DLPFC-projecting L3PNs from PPC and contralateral DLPFC. The transcriptional profile of DLPFC-projecting L3PNs was present in specific clusters of single nucleus RNAseq data from cortical regions known to project to the DLPFC and not in a cortical region lacking these projections.

Conclusion: These findings demonstrate that DLPFC-projecting L3PNs in monkeys can be identified by a shared transcriptional profile, regardless of cortical region, providing the means to determine if the homologous L3PNs in human are preferentially affected in schizophrenia.

Presenter Name/Degree(s): Manan Arora, MBBS
Current Position: Postdoctoral Associate

Primary Mentor in Psychiatry: Mary L. Phillips, MD

Title: **Elevated left ventrolateral prefrontal cortical(vlPFC) activity to reward expectancy(RE) is associated with higher mania/hypomania risk: A replication study in 3 independent young adult samples**

Author(s): Arora M, Chase H, Bertocci M, Skeba A, Eckstrand K, Bebko G, Aslam H, Raeder R, Graur S, Benjamin O, Wang Y, Stiffler R, and Phillips M
Affiliation(s): Department of Psychiatry, University of Pittsburgh School of Medicine

Introduction: The left ventrolateral prefrontal cortex (vlPFC) is a key region within the reward and salience network. Prior work from our group identified elevated reward expectancy (RE)-related left-vlPFC activity in individuals with Bipolar Disorder (BD) and in those at risk for mania/hypomania. In this study, we aimed to replicate and extend these findings using the MOODS-SR Lifetime manic-domain score—a robust, dimensional measure of mania/hypomania risk—in a new non-BD young-adult sample. Our goals were to determine whether: (1) mania/hypomania risk is positively associated with RE-related left-vlPFC activity; (2) this association is specific to mania/hypomania vs. depression risk; and (3) findings replicate across two independent samples.

Methods: In the discovery sample (n=113; 73 women; mean age=23.88±3.32), participants completed an RE task during 3T fMRI. Whole-brain multiple regression identified regions where RE-related activity correlated with mania/hypomania risk, controlling for depressive-domain scores, age, sex, education, and mean framewise displacement (FWD). We then tested for replication in two independent non-BD samples (n=53 and n=69; similar covariates).

Results: In the discovery sample, eight predominantly left-sided clusters—including the left-vlPFC (T=4.8, k=538, MNI=-44, 50, -4)—showed a positive association with mania/hypomania risk (uncorrected $p<0.001$, $k>20$). In the first replication sample, RE-related left-vlPFC activity was again positively associated with mania/hypomania risk ($\beta=0.160$, FDR-$Q<0.001$). This effect was not observed in the second sample ($\beta=-0.008$, FDR-$Q=0.905$), which had a higher proportion (72.4%) of individuals with lifetime depression. However, when excluding those with depression and combining the remaining participants (n=37), the positive association with mania/hypomania risk was significant and specific ($\beta=0.375$, FDR-$Q<0.001$).

Conclusion: Elevated RE-related left-vlPFC activity is associated with greater lifetime risk for mania/hypomania—but not depression. These findings replicate across samples and suggest that depression history may attenuate this neural marker of mania risk.

Presenter Name/Degree(s): Sophia Arruda Da Costa E Silva, BS
Current Position: Research Specialist

Primary Mentor in Psychiatry: Joseph M. Stujenske, MD, PhD

Title: **Differential afferent and efferent connectivity Between caudal and rostral mPFC**

Author(s): ***Arruda Da Costa E Silva S, Shimoda K, and Stujenske JM***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Prefrontal cortical dysfunction has been implicated in a wide range of psychiatric disorders, such as anxiety and post-traumatic stress disorder. The medial prefrontal cortex (mPFC) is known to exhibit functional and anatomical variations along its dorsoventral axis in mice, but the rostrocaudal organization of the mPFC remains poorly understood.

Methods: To determine differences in afferent and efferent connectivity between caudal mPFC (at the border with ventral anterior cingulate cortex) and more rostral mPFC, we injected viral vectors to label either mPFC projections or afferently projecting cells. The tissue was then cleared followed by whole brain imaging with a light sheet microscope.

Results: Our findings will highlight the projections from the different subcortical areas of the medial prefrontal cortex to the ventral-lateral and dorsal-lateral periaqueductal grey.

Conclusion: Thus, our data suggest significant variations in afferent connectivity along the rostrocaudal axis of the mPFC. Understanding the functions of these distinct afferent and efferent circuits can provide valuable insights into the overall function of the medial prefrontal cortex, generally and in the context of psychiatric disorders.

Presenter Name/Degree(s): Yui Asaoka, PhD
Current Position: Postdoctoral Associate

Primary Mentor in Psychiatry: Mary Phillips, MD, MD (CANTAB)

Title: **Effects of ketogenic diet on reward circuitry in bipolar disorder: A preliminary investigation**

Author(s): ***Asaoka, Y, Bertocci, MA, Raeder, R, Coroso, K, Nooraeen, SA, Bebko, G, Roberts, LT, Stiffler, RS, Didomenico, D, Atkinson, M, Graur, S, Caputo, S, Morris-Tillman, J, and Phillips, ML***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Bipolar disorder (BD) is a severe psychiatric disorder that metabolic disturbances and dysfunctional reward-related activity may characterize. The reward circuit, involving the prefrontal cortex, cingulate cortex, and striatal regions, is often over- or under-activated in individuals with BD, contributing to abnormal reward sensitivity and mood instability. Emerging evidence suggests that a ketogenic diet, by elevating ketone levels, could help restore normal reward function in BD. We hypothesize that magnitudes of abnormal levels of reward circuit function during reward related activity will be reduced in BD participants on a ketogenic diet.

Methods: In this randomized clinical trial, adults (12 female and 2 male, mean age = 31.1) with BD were randomized to complete 8 weeks of a normal diet and 8 weeks of a Ketogenic diet. At baseline and after completing each diet phase (Diet A and Diet B), the participants underwent neuroimaging with a reward task. Clinical rating scales for mania, depression, and anxiety were also administered at each time point. Analysts are blind to diet phase. 14 participants completed baseline neuroimaging, 6 participants completed post Diet A neuroimaging, and 4 participants completed post Diet B neuroimaging. Whole brain T-test analyses were completed in SPM (voxelwise threshold = 10, p = .001).

Results: Diet B > Diet A was associated with significantly increased reward expectancy–related activity predominantly within the central executive and salience networks, including cingulate gyrus (t=12.44, k=17), inferior frontal gyrus (t=9.62, k=46), cerebellar vermis (t=8.30, k=10), right posterior insula (t=6.85, k=14), and supramarginal gyrus (t=5.92, k=26). Diet A>Diet B showed no significant activity in reward expectancy.

Conclusion: These preliminary findings suggest that Diet B may enhance activity in predominantly right-sided prefrontal-parietal circuits involved in executive control and planning, as well as salience detection, potentially normalizing abnormal reward processing in BD. Larger studies and ongoing recruitment are warranted to confirm these results.

Presenter Name/Degree(s): Wasiu Balogun, PhD
Current Position: Postdoctoral Associate

Primary Mentor in Psychiatry: Thomas Karikari, PhD

Title: **Plasma p-tau217 for early detection of brain amyloid pathology in community-dwelling older adults without cognitive impairment: Evidence from three community-based studies**

Author(s): Balogun W[1], Triana-Baltzer G[2], Saeed A[3], Zeng X[1], Gogola A[4], Lopresti BJ[4], Villemagne VL[1], Ganguli M[1,4,5], Kolb H[2], Snitz B[6], Lopez OL[6], Cohen AD[1], Reis S[7], and Karikari TK[1]

Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Neuroscience Biomarkers, Janssen Research & Development; [3]Heart and Vascular Institute, Department of Medicine, University of Pittsburgh; [4]Department of Radiology, University of Pittsburgh School of Medicine; [5]Department of Epidemiology, University of Pittsburgh School of Public Health; [6]Department of Neurology, University of Pittsburgh School of Medicine; [7]Department of Cardiology, University of Pittsburgh School of Medicine

Introduction: Blood biomarkers represent the next generation of Alzheimer's disease (AD) diagnostics, enabling noninvasive, inexpensive, and scalable monitoring of amyloid-beta (Aβ) plaque (A) and tau neurofibrillary tangles (T) pathologies and neurodegeneration (N). Plasma p-tau217 has emerged as perhaps the most promising AD blood biomarker, prompting the development of several technologies to evaluate its prognostic and diagnostic utility. However, cross-cohort validation studies in community-based cohorts are lacking.

Methods: Here, we assessed the Janssen plasma p-tau217+ assay in three community-based cohorts: the Monongahela Youghiogheny Healthy Aging Team-Neuroimaging (MYHAT-NI) with 93 participants (Aβ-PET positivity=24.8%), the Human Connectome Project (HCP) comprising 201 participants (Aβ-PET positivity= 15.0%) and the Heart Strategies Concentrating on Risk Evaluation study (Heart SCORE) made up of 147 participants (Aβ-PET positivity=18.2%), all recruited from southwestern Pennsylvania, USA. We employed [^{11}C] Pittsburgh Compound B (PiB) positron emission tomography (PET) imaging for brain Aβ load. We utilized receiver operating characteristic (ROC) curves to evaluate p-tau217+ accuracies in detecting Aβ pathology, adjusting for age, sex, and APOE4 carrier status.

Results: The Janssen p-tau217+ assay exhibited high performance in identifying Aβ PET positivity in all three cohorts, with AUCs of 91% for MYHAT-NI, 93% for HCP, and 82% for Heart SCORE. Plasma p-tau217+ showed high specificity: MYHAT-NI (57%), HCP (81%), Heart SCORE (87%) but poor sensitivity to Aβ PET: MYHAT-NI (75%), HCP (70%), Heart SCORE (59%). P-tau217+ was strongly correlated with Aβ-PET SUVR and was stronger in the Aβ-PET-positive sub-groups MYHAT-NI (***r***=-0.1292; ***p***=0.2443), HCP (***r***=-0.1617; ***p***=0.0347), Heart SCORE (***r***=0.1586; ***p***=0.0896). Importantly, correlation with Aβ-PET was strongest in MYHAT-NI which had the highest proportion of Aβ-PET-positive participants.

Conclusion: The Janssen p-tau217+ assay identifies Aβ pathology in cognitively normal older adults in the community, underscoring its potential utility as a diagnostic tool for investigating AD at the population-level.

Presenter Name/Degree(s): Alexis Bamfo, B.S**.**
Current Position: Research Assistant

Primary Mentor in Psychiatry: Aliona Tsypes, PhD

Title: **Does it matter how people think about suicide?**

Author(s): ***Bamfo A[1], Hallquist MN[2], Wright AGC[3], Dombrovski AY[1], and Tsypes A[1]***
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine;
[2]Department of Psychology & Neuroscience, University of North Carolina at Chapel Hill;
[3]Department of Psychology, University of Michigan

Introduction: How do people experience their suicidal ideation? While many studies treat ideation as a singular construct, recent research has begun to distinguish between verbal and image-based ideation, with growing evidence suggesting these may uniquely impact suicide risk (e.g., Lawrence et al., 2022; Ng et al., 2016). This study extends previous work by introducing a distinct ideas-based mode of thinking and examining how each mode of thinking (words, images, and ideas) relates longitudinally to features of suicidal ideation in a high-risk sample.

Methods: Over 100 consecutive evenings, participants (n = 73, data collection ongoing; M age = 32.3, 84.9% female) rated the extent to which they experienced suicidal thoughts in three modes—words, images, and ideas—and reported on features of their suicidal thinking. The sample was enriched for individuals with histories of suicidal ideation and attempts.

Results: Multilevel (mixed-effects) modeling revealed that, at the between-person level, image- and idea-based thinking were related to greater suicidal urge. At both the between- and within-person level, suicidal thinking experienced in words was linked with lower suicidal urge.

Conclusion: These findings suggest that how suicidal ideation is experienced may influence its nature and intensity. As data collection continues, further analyses will offer deeper insights into these patterns. Implications for personalized suicide interventions based on modes of thinking will be discussed.

Presenter Name/Degree(s): Kelly Barko, BS, MS
Current Position: CNUP Graduate Student

Primary Mentor in Psychiatry: Marianne Seney, PhD and Matt MacDonald, PhD
(required field unless you are a member of the Department of Psychiatry faculty)

Title: Sex Differences Detected in the Proteome of MDD Subjects

Author(s): ***Barko K[1,2], Ruiz S[1,2], DeMarco A[1], Klei L[3], Devlin B[3], Lewin A[1], Grady A[1], Seney M[1,2], and MacDonald M[1,2]***
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Center for Neuroscience, University of Pittsburgh; [3]Department of Neurobiology, University of Pittsburgh

Introduction: MDD is a leading cause of disability that affects over 300 million people globally with core symptoms of emotion dysregulation, low mood, anhedonia, and poor affect. Despite the impact of MDD, limited treatments are available and mostly target the monoamine system with two-thirds of individuals prescribed reporting failed-remission of symptoms. Moreover, sex differences have been reported in MDD prevalence, symptom presentation, and corticolimbic brain activity. Collectively, these observations prompt the need to understand sex-specific molecular mechanisms underlying MDD pathology within mood-regulating regions of the brain.

Methods: Here, we used human postmortem tissue from the anterior cingulate cortex (ACC) of male and female MDD subjects and performed mass spectrometry (MS/MS) to explore sex differences within the proteome underlying MDD. The ACC from age/sex matched pairs of controls and MDD subjects (N=30/sex/group; 120 subjects) was homogenized, TMT-labeled, and quantified using Ultra-High-Performance Liquid Chromatography (UHPLC) and MS/MS. Proteomics data was analyzed using Proteome Discover and R (v4.3.2). Statistical significance was set a $q<0.05$.

Results: When examining the main effect of diagnosis (MDD vs. controls), we identified 160 dysregulated proteins. Interestingly, there were 309 proteins with a significant interaction of sex and disease, suggesting that sex modulates disease-associated proteomic changes. Notably, proteins dysregulated in MDD males and females were altered in opposite directions, indicating sex-specific molecular signatures of MDD. Proteins showing a significant sex-by-diagnosis interaction were enriched in pre- and post-synaptic ribosomal components within the synapse and pathways related to translation. Collectively, these findings highlight the importance of considering sex as a biological variable and suggest that MDD pathology is influenced by sex-specific mechanisms.

Conclusion: We observed striking sex differences in proteomic alterations associated with MDD. Ongoing studies are examining synaptic proteomic as well as phosphoproteomic alterations in the same cohort.

Presenter Name/Degree(s): Guilherme Bauer Negrini
Current Position: Postdoctoral Associate

Primary Mentor in Psychiatry: Tharick Ali Pascoal

Title: **Longitudinal progression and harmonization of tau-PET tracers**

Author(s): *Bauer-Negrini, G[1], Ferreira, P[1], Povala, G[1], Bellaver, B[1], Lussier, FZ[1], Amaral, L[1], Tudorascu, DL[1], Finn, Q[2], Rahmouni, N[3], Therriault, J[3], Servaes, S[3], Stevenson, J[3], Macedo, AC[3], Masdeu, JC[2], Soleimani-Meigooni, DN[4], Fortea, J[5], Lowe, VJ[6], Oh, H[7], Pascual, B[2], Gordon, BA[8], Rosa-Neto, P[9], Baker, SL[10], Pascoal, TA[1]*

Affiliation(s): *[1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Houston Methodist Research Institute; [3]McGill University; [4]Memory and Aging Center, Weill Institute for Neurosciences, University of California San Francisco;[5]Sant Pau Memory Unit, Hospital de la Santa Creu i Sant Pau, Institut de Recerca SantPau - Universitat Autònoma de Barcelona;[6]Mayo Clinic; [7]Brown University; [8]Washington University in St. Louis, School of Medicine; [9]McGill University Research Centre for Studies in Aging; [10]Lawrence Berkeley National Laboratory*

Introduction: Tau-PET tracers have been used to monitor the progression of Alzheimer's disease (AD). However, different tracers present distinct patterns of binding throughout the brain, challenging the harmonization of their findings. Leveraging the HEAD Study, we recently developed the Uniτ scale, which cross-sectionally harmonizes Flortaucipir and MK6240 onto a universal tau-PET measurement. Here, we provide a preliminary evaluation of the Uniτ scale's longitudinal performance in HEAD and two independent cohorts.

Methods: We assessed 422 individuals across the AD spectrum with longitudinal tau-PET from three cohorts: HEAD, ADNI, and TRIAD. Standardized uptake ratios (SUVRs) were harmonized to Uniτ using the Uniτ Ecosystem (unitau.app). Braak I-II and Meta-Temporal regions were used as regions of interest. Annual tau-PET uptake changes and effect sizes were calculated.

Results: In HEAD, annual change in tau-PET uptake in Braak I-II across CU and CI was only detectable in MK6240. However, both tracers detected significant annual changes in the Meta-Temporal ROI for CI. In both ADNI (Flortaucipir) and TRIAD (MK6240), changes in tau-PET uptake in Braak I-II regions were not significant. However, across all cohorts, the Meta-Temporal ROI consistently showed detectable annual tau-PET increases—most pronounced among CI—leading to larger effect sizes than in Braak I-II. The Uniτ harmonization did not fundamentally alter the pattern of the findings. However, in ADNI, Uniτ yielded a slightly higher effect size than SUVR in CU for both Braak I-II and Meta-Temporal regions.

Conclusion: In this large longitudinal sample, our findings confirm that Flortaucipir and MK6240 can detect tau PET changes over time likely associated with the progression of tau tangle pathology. While MK6240 appears to show greater progression in CU, both tracers progress similarly in CI. Our data also suggested that Uniτ harmonized tau PET measurements maintain the longitudinal characteristics of each tau PET tracer for use in clinical trials.

Presenter Name/Degree(s): Shlomo Bear
Current Position: Student Researcher

Primary Mentor in Psychiatry: Leonardo D'Aiuto, PhD

Title: **circRNA derived from extracellular vesicles as potential biomarkers for schizophrenia**

Author(s): Bear S[1], Wang L[1], Milosevic J[2], Stamm S[3], Wood J[1], Wesesky M[1], D'Aiuto L[1], Nimgaonkar V[1], and He H[2]
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Captis Diagnostics; [3]University of Kentucky College of Medicine

Introduction: Schizophrenia (SCZ) is a heterogenous neuropsychiatric disorder marked by diverse symptoms including psychosis, delusions, and cognitive impairment. Despite thoroughly studied changes in neurotransmission, clear genetic links, and physiological differences identified via MRI, fMRI, and biopsy studies, no reliable biomarkers or laboratory tests for SCZ exist. Diagnosis therefore relies on the judgement of clinicians who utilize diagnostic criteria and structured interviews, which are empirically based but not necessarily biologically grounded. Circular RNAs (circRNAs), a class of RNA molecules generated through a process called back-splicing, have shown SCZ-specific expression patterns in postmortem brain tissue and represent promising biomarkers. To identify novel biomarkers for SCZ, we quantified SCZ-linked circRNAs in extracellular vesicles (EVs)—small, membrane-bound particles secreted by cells and present in all biofluids.

Methods: Serum samples from 10 SZC subjects and 10 healthy controls were selected for pilot study. Diagnosis was determined by structured interview using the Arabic SCAN and expert clinical consensus. EVs were isolated from serum using magnetic lipid nanoprobe technology (Captis Diagnostics). EV-RNA was extracted using Zymo Research Quick-cfRNA kit, quantified by Qubit fluorometry, and reverse transcribed to cDNA using random hexamers and M-MuLV Reverse Transcriptase. qPCR targeting four circRNAs previously identified in postmortem SCZ brain tissue (circTOP1-10, circMYO9A-66, circZNF236-2, circHomer1a) was performed on a GFX Opus 96 Real-Time PCR System using circular isoform-specific divergent primers.

Results: Total EV-RNA yield from 650µL of serum: 23-47.5ng, mean 37.1 (patients), 22-46.8ng, mean 34.2 (controls). Our initial investigation indicates case-control differential expression of circZNF236-2.

Conclusion: The observed signal from circZNF236-2 aligns with known circRNA dysregulation in SZC. Our results support the feasibility of quantifying EV-circRNAs in archived serum samples, and highlight the need for further optimization of assay methodology. This work establishes a foundation for development of a diagnostic EV-circRNA panel for SCZ using minimally invasive biofluid biopsies; however, our preliminary results warrant further validation.

Presenter Name/Degree(s): Anne Beatty, BSE
Current Position: Graduate Student

Primary Mentor in Psychiatry: Beatriz Luna, PhD

Title: **Contributions of neuronal oscillations and cortical SNR to developmental changes in inhibitory control from adolescence into adulthood**

Author(s): Beatty A[1] , McKeon S[1] , Petrie D[2] , Foran W[2] , Calabro F[1,2], Luna B[2,3]
Affiliation(s): [1]Department of Bioengineering, University of Pittsburgh; [2]Department of Psychiatry, University of Pittsburgh School of Medicine; [3]Department of Psychology, University of Pittsburgh

Introduction: Adolescence is a critical period of development, marked by protracted improvements in cognitive control in parallel with brain maturation of executive systems. Brain systems supporting inhibitory control mature into adulthood, supporting more accurate performance on response inhibition tasks such as the anti-saccade (AS) oculomotor task. Differences in oscillatory power associated with successful response inhibitions compared to error trials are not well understood and will provide insight into the mechanisms that allows a correct AS response.

Methods: EEG data was collected on 107 participants (10-33yo) while performing the AS task. In the AS task, participants fixate on a central cross for 500ms (preparatory period) and when it is extinguished, they are instructed to make an eye movement to the mirror location of the visual stimulus. We computed time-frequency clusters of activation based on the event-related spectral perturbation (ERSP) through the preparatory period of correct AS trials and analyzed the developmental changes in power of these frequency bands.

Results: ERSP of the preparatory period of correct AS trials identified 3 significant clusters. There was a developmental decrease in theta band ($p<0.05$) during correct trials compared to baseline. Comparing correct and error trials, the average power within these clusters showed a significant main effect of trial type with increased theta power in error trials ($p=0.031$). There was a significant interaction between age and type of trial on high-beta power ($p=0.038$), adolescents had greater high-beta power during error trials.

Conclusion: These results show that underlying developmental improvements in inhibitory control is dampening of relevant oscillatory mechanisms during the preparation to stop a prepotent response. Adolescents fail to dampen preparatory midline high-beta response that may interfere with their ability to specifically prepare an inhibitory response. Adults have error trials Instructions: Please refer to the example appearing on page 2 of the Call for Abstracts when entering your abstract information. This form has been preformatted. Do not change the margins, font type or size, or delete any of the abstract fields. Word Limit: The body of your abstract (Introduction, Methods, Results, Conclusion) must not exceed 300 words. when they do not effectively suppress the midline theta response, which may reflect specificity of engaging performance monitoring processes.

Presenter Name/Degree(s): Bruna Bellaver, PhD
Current Position: Research Assistant Professor

Title: **Head-to-head trajectories of MK6240, Flortaucipir, and plasma p-tau217 as a function of amyloid-β**

Author(s): *Bellaver B[1], Povala G[1], Ferreira PL[1], Bauer-Negrini G[1], Lussier F[1], Amaral L[1], Soares C[1], Rocha A[1], Tudorascu DL[1], Karikari TK[1], Masdeu J[2], Soleimani-Meigooni D[3], Fortea J[4], Lowe V[5], Oh H[6], Pascua Bl[2], Gordon BA[7], Rosa-Neto P[8], Baker S[9], and Pascoal TA[1,10]*
Affiliation(s): *[1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Houston Methodist Research Institute, Department of Neurology; [3]University of California San Francisco, Memory and Aging Center; [4]Hospital de la Santa Creu I Sant Pau, Sant Pau Memory Unit; [5]Mayo Clinic, Department of Radiology; [6]Brown University, Department of Psychiatry and Human Behavior; [7]Washington University in St. Louis, Department of Radiology; [8]Translational Neuroimaging Laboratory, McGill University Research Centre for Studies in Aging, Douglas Research Institute; [9]Lawrence Berkeley National Laboratory, Berkeley; [10]Department of Neurology, University of Pittsburgh School of Medicine*

Introduction: Tau PET tracers present distinct binding characteristics that might influence their trajectories and relationship with other biomarkers along the AD continuum. In a head-to-head study, we investigated the relationship between the emergence of PET tracers MK6240 and Flortaucipir, and plasma p-tau217 abnormalities as a function of Aβ PET deposition. We further assessed the concordance between tau PET and plasma p-tau217 positivity and its relationship with cognitive scores.

Methods: We evaluated 352 individuals from the HEAD study (205 cognitively unimpaired and 147 cognitively impaired) with Aβ PET, MK6240 and Flortaucipir tau PET, and plasma p-tau217 and GFAP measures. Tau PET Braak regions, plasma p-tau217 trajectories were modeled as functions of Aβ burden (Centiloid scale) using the Lowess method. Biomarkers were z-scored anchored on young individuals (n=19, < 28 years old). Tau PET (Braak 1 region) and plasma p-tau217 were considered positive/abnormal when surpassing 2.5 z-score.

Results: Among the tested markers, MK6240 was the earliest to show abnormality as a function of Centiloid, occurring at 22 Centiloids, followed by plasma p-tau217 at 38 Centiloid and Flortaucipir at Centiloid 56. Tau PET and plasma p-tau217 positivity showed an overall high concordance (~80% for both tracers). In MK6240 discordant cases, most individuals were MK6240+/p-tau217- (13.8%), while 6.6% were MK6240-/p-tau217+. For Flortaucipir, 15% of discordant cases were Flortaucipir-/p-tau217+, while 4.8% were Flortaucipir+/p-tau217-. The discordant groups present higher Centiloid than the ptau217-/Tau- group and overall decreased scores in cognitive tests, except for p-tau217+/MK6240- group.

Conclusion: MK6240 becomes abnormal at lower levels of Aβ burden compared to plasma p-tau217 and Flortaucipir. The relatively high prevalence of discordant tau PET positive or plasma p-tau217 positive and suggests that some individuals may show tau PET positivity first, while others may exhibit plasma p-tau217 positivity first. The distinct cognitive profiles of this groups suggest potential clinical relevance, warranting further investigation.

Presenter Name/Degree(s): Charles Bennett, PhD
Current Position: Advanced Postdoctoral Fellow

Primary Mentor in Psychiatry: Gretchen L. Haas, PhD

Title: **Interactive voice response (IVR) monitoring as an adjunct to outpatient assessment of suicidal thoughts and behaviors**

Author(s): *Bennett CB[1,2], McShea M[1], Congedo B[1], Luther JF[1,3], Theodore E[1], Goodman M[5,7], Wolkin A[6,8], and Haas GL[1,2,4]*
Affiliation(s): *[1]VISN4 MIRECC, VA Pittsburgh Healthcare System; [2]Department of Psychiatry, University of Pittsburgh School of Medicine; [3]Epidemiology Data Center, University of Pittsburgh School of Public Health;[4]Department of Psychology, University of Pittsburgh; [5]James J. Peters VA Medical Center; [6]VA New York Harbor Healthcare System; [7]Department of Psychiatry, Mount Sinai School of Medicine; [8]Department of Psychiatry, New York University Grossman School of Medicine*

Introduction: Suicide is a leading cause of death among Veterans. To address this concern, the Veteran Health Administration (VHA) identified suicide prevention as a top priority. The current study aimed to determine the effectiveness of IVR as a tool for daily monitoring and detection of suicidal thoughts and behaviors (STBs) among Veterans following hospital discharge and between outpatient visits.

Methods: Participants (N = 18) were Veterans admitted to psychiatric inpatient units with recent suicidal thoughts and behaviors (STBs) at three VA Medical Centers. Upon discharge, participants received automated daily IVR calls for 12 weeks, as well as clinical interviews at 2-, 4-, 8-, and 12-weeks. For this report, we evaluated the incremental value of IVR in terms of endorsements and denials of STBs relative to: a) clinical interviews, using the Beck Scale for Suicidal Ideation (BSS) and the Columbia-Suicide Severity Rating Scale (C-SSRS), and b) outpatient appointment and phone contact records documented in the electronic health record (EHR).

Results: There was a total of 22 endorsements out of 39 responses on BSS and IVR, with the BSS eliciting more endorsements of STBs than IVR calls. On examination of the C-SSRS and IVR, there was a total of 24 endorsements out of 43 responses. IVR calls elicited more endorsements of STBs than did the C-SSRS. There was also a total of 125 clinical encounters (i.e., outpatient appointments or phone calls) documented in the EHR. IVR elicited more endorsements of STBs than were documented in the EHR.

Conclusion: Findings highlight the potential value of post-discharge tracking of STBs via remote assessment between outpatient appointments. This pilot study provides preliminary evidence that IVR could serve as an adjunctive tool for post-discharge monitoring of STBs in conjunction with outpatient clinical encounters. Further development of IVR is warranted before implementation in clinical practice.

Presenter Name/Degree(s): Mariah Berchulski, BS
Current Position: CNUP Graduate Student

Primary Mentor in Psychiatry: Joseph Stujenske, MD, PhD

Title: **Simultaneous superficial and deep layer calcium imaging in midline cortex with preserved local cytoarchitecture**

Author(s): *Berchulski, MR[1,3], Ozbay, B[4], Kilborn, K[4], and Stujenske, JM[1,2,3]*
Affiliation(s): *[1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Translational Neuroscience Program, University of Pittsburgh; [3]Center for Neuroscience, University of Pittsburgh; [4]Intelligent Imaging Innovations Inc.*

Introduction: *In vivo* two photon calcium imaging permits tracking neuronal population responses with high spatial precision. Most cortical areas are easily accessed via cranial window, while midline cortical areas like mouse medial prefrontal cortex (mPFC) are difficult to access without removing overlying cortex. Likewise, superficial and deep layers are difficult to simultaneously capture at high frame rate as long travel prevents use of methods that physically move the focal plane. Here, we present a means of overcoming these challenges using contralateral microprism, beam splitting with temporal multiplexing, and adaptive optics.

Methods: Collaborating with a commercial microscope company, a novel microscope was designed that combines short pulse width femtosecond laser (Vision-S, pulse width < 80 fs), temporal multiplexing, and adaptive optics using a deformable mirror. Here, we describe the design and quantify the optical properties of this microscope, with and without a microprism. We demonstrate the capabilities of this microscope for *in vivo* imaging using wildtype C57BL/6J mice injected with jGCaMP8s in mPFC and implanted with a 1.5mm microprism in contralateral mPFC. Calcium signals were imaged in awake mice on a stationary treadmill.

Results: We demonstrate the custom microscope provides resolution sufficient for single cell and neurite imaging. While the effective numerical aperture of the objective is decreased by introduction of the microprism, the sparse fluorophore expression, together with statistical methods for separating overlapping cell decontamination, permits imaging *in vivo.* When simultaneously imaging from superficial and deep cortex, we quantify ~20% of each plane's signal is due to crosstalk, and demonstrate a computational method for separation. We quantify the performance improvements yielded by using adaptive optics.

Conclusion: Our data demonstrate this custom microscope can be used for superficial and deep layer simultaneous imaging through a microprism in midline cortex, without altering its local cytoarchitecture. This microscope is complementary to other approaches for dual plane imaging.

Presenter Name/Degree(s): Lizzie Biver
Current Position: Undergraduate Student Researcher

Primary Mentor in Psychiatry: Marta, Peciña MD, PhD

Title: **Apathy and neuroinflammation: A dopamine-independent pathway**

Author(s): *Biver L[1], Strohecker E[1], Snyder I[1], Burghardt P[2], Zubieta JK[2], and Pecina M[1]*
Affiliation(s): *[1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Wayne State; [3]Mass General Brigham*

Introduction: Anhedonia –the inability to experience reward– is a core symptom of Major Depressive Disorder (MDD) and a strong predictor of poor treatment response. Dopamine (DA) systems, particularly in the striatum, are key regulators of reward processing and have been implicated in MDD. Prior work from our group demonstrated an inverse association between anhedonia and striatal DA binding. Emerging evidence suggests that proinflammatory cytokines may further contribute to anhedonia by disrupting these neuromodulatory systems, yet the interplay among inflammation, striatal DA, and anhedonia remains poorly understood. We hypothesized that greater anhedonia severity would be associated with: (1) elevated proinflammatory cytokine levels and (2) that these effects will be moderated by striatal DA receptor binding.

Methods: To investigate these mechanisms, 41 unmedicated individuals with MDD completed a PET scan using the $D_{2/3}$ receptor antagonist [^{11}C] raclopride. Anhedonia was measured using the Apathy Evaluation Scale (AES) and the Snaith-Hamilton Pleasure Scale (SHPS), and inflammatory markers (e.g., CRP, IL-6, IL-8, IL-18, TNF- α) were assessed from blood samples. Group-level PET analyses were conducted using voxelwise general linear models within a striatal mask (Peciña et al., 2015, 2017).

Results: We found a significant correlation between measures of $D_{2/3}$ binding potential, inflammation, and the severity of anhedonia symptoms. AES scores were positively correlated with the pro-inflammatory cytokine TNF- α ($r = 0.52$, $p = 0.02$) and negatively correlated with $D_{2/3}$ receptor availability in the left Globus pallidus ($r = -0.53$, $p = 0.02$). TNF- α did not moderate the effect of DA receptor availability on AES scores.

Conclusion: The observed associations among inflammation and reward deficits suggest a mechanistic pathway linking immune dysregulation with motivational and hedonic impairments in MDD, yet these effects were not moderated by baseline dopamine receptor availability.

Presenter Name/Degree(s): Gina Boito BS
Current Position: Research Specialist

Primary Mentor in Psychiatry: Neil Jones, PhD

Title: **The impact of selective serotonin reuptake inhibitors on episodic memory**
Author(s): ***Boito GM, Forbes EE, Franzen PL, Horton LE, and Jones NP***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Depression is a leading cause of disability and may cause impairments in memory and learning. Selective serotonin reuptake inhibitors (SSRIs) are one of the primary treatments for depression. However, current scientific literature is contradictory on how SSRIs affect brain regions and their impacts on episodic memory. In the current study, we examined the effects SSRIs have on hippocampal subregions associated with episodic memory: the dentate gyrus, subiculum, CA1, and CA3. We hypothesized that participants who are medicated with SSRIs will perform better on an episodic memory task than unmedicated participants, coinciding with greater subregion blood oxygen level dependent responses during memory encoding and larger subregion volume.

Methods: Participants (n = 175) were divided into three groups: controls with no lifetime DSM-5 psychiatric diagnosis (n = 38), unmedicated individuals diagnosed with a depressive disorder (n = 105), and those diagnosed with a depressive disorder on SSRIs (n = 32), Participants completed the Episodic Memory Task which entails encoding neutral faces and retrieving those faces from memory during fMRI assessment. Structural brain images were segmented and hippocampal subregion volumes extracted using Freesurfer.

Results: Although task accuracy did not differ between groups, participants on SSRIs reported more severe depression symptoms and significantly greater overall hippocampal volume compared to both unmedicated participants and controls. During encoding, medicated participants exhibited equivalent subiculum responses relative to controls, and both groups demonstrated greater subiculum responses relative to unmedicated participants. Similarly, in the encode condition, medicated participants showed equivalent dentate gyrus and CA1 responses relative to controls; whereas unmedicated participants demonstrated decreased responses relative to controls.

Conclusion: SSRIs may cause compensatory hippocampal enlargement and normalization of hippocampal function, but it remains unclear how these changes translate to episodic memory capabilities.

Presenter Name/Degree(s): Sarah Brammell, BS
Current Position: Research Specialist

Primary Mentor in Psychiatry: Carla Mazefsky, PhD

Title: **Mental health and service use among cisgender and sexual and/or gender minority autistic young adults**

Author(s): Brammell, S[1], Sivathasan, S[2], Crown, MJ[3], Rutenberg, E[4], Conner, CM[1], Beck, KB[1], Rofey, D[1], and Mazefsky, CA[1]
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Counseling, Development, and Educational Psychology, Boston College; [3]Department of Occupational Therapy, Boston University; [4]Dietrich School of Arts and Sciences, University of Pittsburgh

Introduction: Mental health challenges are prevalent among autistic adults, as recent estimates indicate that up to 50% of autistic adults experience at least one psychiatric disorder. Approximately around a third of autistic adults identify as a sexual and/or gender minority (SGM), with recent work suggesting that SGM autistic adults have worse mental health outcomes than cisgender heterosexual (CisHet) autistic adults. Yet, few studies have explored the intersectional impact of being both autistic and having an SGM identity in relation to mental health services utilization.

Methods: Secondary data analyses were conducted with a subset of autistic young adults recruited to a larger measurement development and online psychometric study. SGM (n=91) and CisHet (n=119) autistic participants (18-25 years) were identified from that dataset. Various relevant self-report measures were compared across the SGM and CisHet groups using chi-square, t-tests, and regression analyses.

Results: 91 participants comprised the SGM group (M_{age}=22.9 years) and 119 comprised the CisHet group (M_{age}=22.3 years).

SGM autistic adults endorsed significantly higher rates of mental health diagnoses (***M***=3.1) than CisHet autistic adults (***M***=2.3), p=.003. While both groups reported high rates of some diagnoses, the SGM group was likelier to report higher rates of depression (62% SGM; 44% CisHet; p<.001), anxiety (88% SGM; 59% CisHet; p<.001), and PTSD (39% SGM; 20% CisHet; p=.003). The SGM group had greater current anxiety (p=.005) and depression (p<.001) symptoms, even after controlling for age. Both groups were equally likely to be utilizing mental health services, however, service and medication type differed by group.

Conclusion: Nearly half (43%) of young autistic adults identified with an SGM identity. Both groups reported significant mental health challenges yet comparable mental health service utilization. It is critical that future research, in addition to supporting both autistic and LGBTQIA+ populations, identifies barriers to care resulting from the intersectional impact of SGM identity among autistic adults.

Presenter Name/Degree(s): Nathan W. Brantly
Current Position: Graduate Student Research

Primary Mentor in Psychiatry: Helmet T. Karim, PhD

Title: **Elucidating the roles of motor function and cognitive switching in locomotor switching after stroke**

Author(s): Brantly NW[1,2], Mariscal Olivares DM[1,2], Choi J[1,2], Weinstein A[3], Karim HT[1,3], and Torres-Oviedo G[1,2]
Affiliation(s): [1]Department of Bioengineering, University of Pittsburgh; [2]Center for the Neural Basis of Cognition, Pittsburgh, PA; [3]Department of Psychiatry, University of Pittsburgh School of Medicine

Introduction: Locomotor switching is the ability to shift walking patterns in response to shifts between familiar environments. Sombric et al. found a correlation between locomotor switching and the ability to switch between tasks cognitively (i.e., cognitive switching) in older adults suggesting cognitive switching brain resources may support locomotor switching[1]; however, the relationship between locomotor and cognitive switching post-stroke is unknown. We hypothesized that participants with better cognitive switching after a stroke would exhibit better locomotor switching due to shared neural resources underlying these behaviors.

Methods: To measure locomotor switching, we adapted 16 participants who have had a stroke (6 female, mean age 57.9 years +/- 13.0 years SD, one extreme outlier omitted) to split-belt treadmill walking for 900 strides (2:1 belt-speed ratio, +/- 33% of 85% of the 6-Minute Walk Test speed) before having those participants shift to walking overground. We characterized locomotor switching performance as the initial aftereffects, which is a measure of how perturbed the gait is, averaged over the first five strides of post-adaptation overground walking. We measured locomotor aftereffects in both the muscle activity (i.e., the magnitude of the EMG activity across 28 muscles) and kinematics (i.e., step length asymmetry). Cognitive switching was assessed using the Trail Making Test (parts A and B)[8]. We tested the association between locomotor switching and cognitive switching.

Results: We are actively collecting data toward our target N = 43 participants with stroke to detect a moderate effect size, and we have evaluated preliminary associations. We found a $\rho = -0.004$ Spearman rank order correlation between the muscle activity measure of locomotor switching and cognitive switching (df = 13, 95% CI = [-0.572,0.605]).

Conclusion: Our preliminary results suggest distinct neural substrates for locomotor and cognitive switching rather than shared brain resources, which has implications for gait rehabilitation strategies for stroke survivors.

Presenter Name/Degree(s): Dakota Brockway PhD
Current Position: Postdoctoral Scholar

Primary Mentor in Psychiatry: Max Joffe, PhD

Title: **Neuropeptide modulation of prefrontal cortex circuitry: Insights into VIP signaling in Alcohol Use Disorder**

Author(s): ***Brockway D, Boehm S, Ganny R, and Joffe M***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Neuropeptides within the prefrontal cortex (PFC) are gaining attention as critical modulators of cortical function and contributors to neuropsychiatric disorders. Among these, vasoactive intestinal peptide (VIP) has recently emerged as a promising therapeutic target for Alcohol Use Disorder (AUD), supported by clinical and preclinical evidence. However, the mechanisms through which VIP modulates neural circuits in the PFC remain largely uncharacterized.

Methods: To investigate how VIP influences PFC circuitry, we utilized ex vivo brain slice preparations from transgenic mice in combination with whole-cell patch-clamp electrophysiology and genetically encoded optical biosensors. Specifically, we employed GCaMP for calcium imaging to monitor neuronal excitability and cADDis to track changes in intracellular cyclic adenosine monophosphate (cAMP) levels in response to VIP application across different neuronal subtypes.

Results: Preliminary data indicate that VIP increases intracellular cAMP levels and enhances excitability in select PFC neuron populations. VIP-expressing interneurons were particularly responsive to VIP application, suggesting a potential autocrine signaling loop. In contrast, somatostatin (SST) and pyramidal neurons displayed more variable responses. These results highlight the specificity of VIP's modulatory actions and support a role for VIPR1-mediated signaling in local circuit regulation

Conclusion: These findings suggest that VIP signaling selectively modulates activity within prefrontal microcircuits and may contribute to the maladaptive changes observed in AUD. Future work will determine how VIP signaling dynamics are altered by alcohol exposure and assess the behavioral relevance of this pathway using in vivo models of alcohol consumption. This research advances our understanding of neuropeptide signaling and supports VIP as a potential target for pharmacological intervention in AUD.

Presenter Name/Degree(s): Zachary Brodnick, BS
Current Position: Research Project Associate

Primary Mentor in Psychiatry: Erika Forbes, PhD

Title: **Dopamine availability and real-time baseline mood symptoms in depressed youth**

Author(s): ***Brodnick ZM, Horter CM, Hart KP, Jones N, Calabro F, Seah TH[1], Luna B, Ryan ND, and Forbes EE***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Dopamine function (DA) is critical to motor and reward neural systems, which are postulated to go awry in depression and other serious psychopathology. Additionally, ecological momentary assessment (EMA) has been previously leveraged to capture real-time emotions and behaviors, which can provide a window into real-world experiences and functioning. This project aims to investigate the relationship between EMA-based mood ratings and DA synthesis and availability, which could aid in the field's understanding of the neurobiological underpinnings of depression.

Methods: The present study included subjects aged 15-25 years old (n = 43) who met criteria for a current DSM-5 depressive disorder via a SCID diagnostic interview and a MADRS score of ≥ 12. Additionally, all subjects completed a baseline MRI scan that included a neuromelanin (NM) scan for substantia nigra NM and an R2' scan for basal ganglia tissue iron, as well as self-report measures assessing anhedonia and depression severity. Following the MRI visit, subjects were enrolled in an EMA protocol where they answered short surveys about their mood 7x/day for 7 days. Regression models examined the associations between DA variables and mean happiness, sadness, energy, motivation, anxiety, and stress, while controlling for biological sex, age, and depressive symptoms.

Results: Left substantia nigra NM contrast was positively associated with mean sadness (t= [3.938], p= [0.019]. Additionally, caudate R2' was positively associated with mean energy (t= [2.812], p= [0.030]).

Conclusion: NM was associated with real-world sadness, and R2' was associated with real-world energy in youth with depression. Our NM findings could reflect history of stress and norepinephrine function, while caudate tissue iron findings could reflect motor and motivational processes. These findings indicate that variability in DA function can be assessed in this population and that DA proxy measures could shed light on the mechanisms of mood and behavior in youth.

Presenter Name/Degree(s): Aswathy BS, PhD
Current Position Postdoctoral Associate

Primary Mentor in Psychiatry: Colleen McClung, PhD

Title: **The ketogenic diet alters dopaminergic activity in the ventral tegmental area in a mouse model of bipolar disorder**

Author(s): ***Aswathy BS, Nelson L, Kaminsky M, Olmeda, S, Fairbanks N, and McClung C***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh, School of Medicine***

Introduction: Bipolar disorder (BD) is associated with elevated dopaminergic (DA) transmission, particularly within the reward network contributing to manic-like behaviors. Functional imaging studies reveal increased reward network activation during reward expectancy in individuals with BD and those at risk for mania/hypomania. The ClockΔ19 mutant mouse, a validated BD model, exhibits manic-like behaviors and increased DA transmission from the ventral tegmental area (VTA), paralleling these clinical features. Given the growing interest in metabolic interventions for neuropsychiatric disorders, this study explores whether a ketogenic diet (KD) influences VTA dopaminergic function in this model

Methods: Wild-type (WT) and ClockΔ19 mice were fed either a KD or standard chow for four weeks. Following the dietary intervention, brains were harvested. Whole-cell patch-clamp recordings were conducted in the DA neurons from the VTA to assess the electrophysiological properties of dopaminergic neurons, identified by spontaneous firing at zero current injection, a rebound spike after hyperpolarizing current, and a voltage sag during the hyperpolarizing step. The action potential (AP) firing properties were assessed in the current-clamp mode. Neurons were subjected to incremental current steps ranging from −80 to 200 pA, and the evoked action potential responses were recorded and analyzed.

Results: Preliminary results indicate altered dopaminergic transmission in ClockΔ19 mice on KD, including a higher rheobase and decreased excitability compared to ClockΔ19 mice on control chow, suggesting a genotype-specific response to the ketogenic diet.

Conclusion: These findings suggest that the ketogenic diet may modulate VTA dopaminergic neuron function in the BD model. Ongoing analyses aim to further characterize intrinsic membrane properties and synaptic inputs and to explore potential sex-specific effects. These early observations support the possibility that dietary interventions like the ketogenic diet can influence dopaminergic signaling in BD, offering a novel avenue for future therapeutic exploration.

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Presenter Name/Degree(s): Reece Budinich, B.S.
Current Position: Graduate Student

Primary Mentor in Psychiatry: Max Joffe, PhD

Title: **Xylazine reduces prefrontal cortex inhibition and prevents oxycodone place preference**

Author(s): ***Budinich RC and Joffe ME***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: The prevalence of xylazine adulteration in clandestine opioid supplies is increasing. This trend is troubling, as xylazine carries its own acute side effects and its long-term cognitive and motivational effects are not known. Furthermore, there are many open questions regarding xylazine's cellular and molecular mechanisms. Canonically known as an α2-adrenergic receptor agonist, recent evidence has implicated the kappa opioid receptor and sigma (σ) receptors in xylazine's mechanism of action. Thus, we investigated xylazine's modulation of rewarding effects of opioids and signaling in the medial prefrontal cortex (mPFC).

Methods: The capabilities of xylazine (100 μM) to modulate mPFC pyramidal cell spontaneous and evoked inhibitory post-synaptic currents (IPSCs) were measured using whole-cell patch-clamp electrophysiology. Evoked currents were driven by either electrical stimulation of mPFC layer 1 or optogenetic activation of somatostatin (SST) interneurons. Xylazine's interactions with opioid reward-mediated behaviors were assessed by comparing conditioned place preference (CPP) of oxycodone (1.0 mg/kg) with or without xylazine (0.5 mg/kg).

Results: While oxycodone administration produced CPP and caused hyperlocomotion, xylazine alone did not affect place preference or locomotion. However, when xylazine was administered with oxycodone, it prevented place preference and hyperlocomotion from forming. Xylazine did not change spontaneous IPSC frequency or amplitude, but did decrease the amplitude of evoked IPSCs. This decrease was attenuated by σ receptor, but not opioid or adrenergic receptor, antagonism. Additionally, xylazine was found to decrease the holding current of pyramidal cells, suggesting an effect on membrane properties in addition to inhibitory synaptic strength.

Conclusion: Xylazine administration decreases mPFC pyramidal cell evoked inhibitory currents via a σ receptor-mediated mechanism, and xylazine induced a depolarizing current in these cells. Behaviorally, xylazine can prevent oxycodone-mediated place preference and hyperlocomotion.

Presenter Name/Degree(s): Lucía Bustos-Robles
Current Position: Undergraduate Research Student

***Primary Mentor in Psychiatry*:** Marta Peciña, MD, PhD

Title: **Tolerability of a single dose of buprenorphine, naltrexone, or placebo in major depressive disorder: Insights from the RAISE study**

Author(s): *Bustos L, Strohecker E, Badhan G, Karim H, Price R, Ferrarelli F, Dombrovski A, and Peciña M*
Affiliation(s): *Department of Psychiatry, University of Pittsburgh School of Medicine*

Introduction: Buprenorphine, a partial μ-opioid receptor agonist, and naltrexone, a μ-opioid receptor antagonist, are being explored for their potential to modulate affective and motivational symptoms in Major Depressive Disorder (MDD). However, their tolerability in experimental settings remains under characterized. This study evaluated the tolerability of a single dose of buprenorphine (0.3 mg/mL), naltrexone (50 mg), or placebo in unmedicated individuals with MDD.

Methods: A total of 101 unmedicated adults with MDD were randomly assigned to buprenorphine (n = 36), naltrexone (n = 33), or placebo (n = 32) in a between-subject design. Following drug administration, participants completed an fMRI task probing expectancy-related mood changes. Each participant completed three weekly sessions (drug + fMRI). Adverse effects were recorded and categorized as central nervous system (CNS), gastrointestinal (GI), or general.

Results: Out of 255 reported adverse events, the majority occurred in the buprenorphine group 68.2%), followed by naltrexone (16.5%) and placebo (15.3%). CNS effects, including dizziness and sedation, were reported most frequently with buprenorphine (n = 48), and to a lesser extent with naltrexone (n = 24) and placebo (n = 12). Interestingly, euphoria was only reported within the buprenorphine group (n = 6). GI symptoms, including nausea and vomiting, were prominent in the buprenorphine (n = 41) and naltrexone (n = 18) groups, with only buprenorphine-treated participants reporting GI symptoms. Of note, vomiting was only reported in this group (n = 14). General symptoms were reported predominantly in the buprenorphine (n = 85) and placebo (n = 24) groups. No serious adverse events were reported.

Conclusion: Buprenorphine was associated with the highest frequency of adverse effects, particularly sedation and GI symptoms. Naltrexone showed a milder adverse effect profile, while placebo was well tolerated. These findings highlight the need for close monitoring in opioid-based experimental paradigms for MDD.

Presenter Name/Degree(s): Sophia Buzanis, B.A.
Current Position: Medical Student

Primary Mentor in Psychiatry: Karen Jakubowski, Priya R. Gopalan, Meredith L. Spada

Title: **Developing a brief clinical pathway for trauma-focused management of individuals with trauma histories admitted to the medical hospital**

Author(s): ***Buzanis S[1], Jakubowski K[2], Christian C[2], Griffith J[2], Pale M[2], Gopalan P[2,3], and Spada M[2]***
Affiliation(s): [1]Department of Medicine, University of Pittsburgh; [2]Department of Psychiatry, University of Pittsburgh School of Medicine; [3]Department of Obstetrics, Gynecology, and Reproductive Sciences, University of Pittsburgh

Introduction: Roughly one-third of medically hospitalized adults have a history of post-traumatic stress disorder (PTSD). Among medical inpatients, comorbid PTSD is associated with higher healthcare utilization and poorer prognosis. We aimed to develop, implement, and evaluate a brief clinical pathway for trauma-focused management of psychosocial symptoms for adult female hospital patients with trauma histories.

Methods: The 4-session clinical pathway was designed to be incorporated into the workflow of a psychiatry consultation-liaison service and includes: (1) psychoeducation on trauma and health; (2) basic cognitive behavioral therapy (CBT) skills; (3) identifying trauma-related thoughts and behaviors; (4) mindfulness and relaxation skills. Patients complete pre- and post-intervention surveys assessing depression (PHQ-9≥10), anxiety (GAD-7≥10), PTSD (PC-PTSD-5≥3), sleep health (SATED; range: 0-12, higher=better), and self-compassion (Self Compassion Scale-Short Form; range: 12-60, higher=better), and a 6-week follow-up intervention satisfaction survey. Clinicians provide post-session process metrics. We aim to recruit n=96 patients. Here, we present initial results on patient pre-intervention symptoms and provider process metrics from the initial recruitment (Jan-March 2025).

Results: Nine women **(**56% African American, 44% White; mean age = 39) enrolled in the intervention. Pre-intervention, most (***n***=8, 89%) women reported clinically elevated depression, over half (***n***=5, 56%) reported clinically elevated anxiety, and three women (33%) reported clinically elevated PTSD symptoms. Women reported moderate sleep health (***M***=5.1, ***SD***=1.5) and self-compassion (***M***=38.7, ***SD***=8.9). Patients completed an average of 2 sessions. On average, sessions lasted 30 minutes and clinicians completed approximately 86% of the intervention content per session.

Conclusion: Preliminary data indicate that medically hospitalized patients with trauma histories reported clinically elevated levels of depression, anxiety, and PTSD symptoms, supporting the need for the intervention. Results reflect patient engagement with the intervention and support the ability of providers to incorporate the intervention into clinical workflow. We will discuss challenges and lessons learned from implementation of this ongoing intervention**.**

Presenter Name/Degree(s): Beth Campbell, BA
Current Position: Data Coordinator & Analyst

Primary Mentor in Psychiatry: Katalin Szanto, PhD

Title: **Parsing the heterogeneity of multidimensional determinants of suicide risk in depressed older adults: focus on cognition, personality, and social risk factors**

Author(s): *Campbell E[1], Galfalvy H[2], Wong M[1], Szücs A[3], and Szanto K[1]*
Affiliation(s): *[1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Columbia University, Department of Psychiatry; [3]Faculty of Behavioural and Movement Sciences, Vrije Universiteit Amsterdam*

Introduction: Suicide attempters assessed in old age differ in personality, cognitive, and familial characteristics based on whether their recent suicidal behavior is a first-time suicidal crisis (late-onset suicidal behavior (LO)) or is the continuation of recurrent or chronic early-life suicide risk (early-onset suicidal behavior (EO)). Yet, the relative importance of factors that determine suicide risk remains unclear. This study aims to identify the importance of multiple domains in discriminating EO and LO attempters from non-attempters.

Methods: Sample consists of EO and LO attempters (n=166) and depressed non-attempters (DNA; n=232). Missing data were imputed using Multivariate Imputation by Chained Equations by domain, and then combined, generating 100 versions of the dataset with 88 predictor variables grouped into 7 domains (Sociodemographics, Physical Health, Cognition, Social Dynamics, Behavioral Control, Emotional Regulation, Early History). Two penalized binomial logistic regression models were fit using cross-validation for each dataset to distinguish EO and LO attempters from DNAs. Models were trained on 70% of the data and tested on the remaining 30%. Variable importance was defined by the proportion of models in which the variable was retained. Domain importance was defined by maximum importance of all variables in the domain.

Results: "Behavioral Control" had a maximum variable importance above 99% for both EO and LO attempters compared to DNA. For EO, "Emotion Regulation" and "Sociodemographics" were also above 80%, whereas for LO, "Cognition" and "Social Dynamics" were 100%. Variables distinguishing EO from DNA in >90% of models were lifetime substance use, decreased motivation, recurrent depressive episodes, more negative life outcomes from acting out, and female sex. Variables distinguishing LO from DNA in 90% of models were current substance use, less subjective social support, more cognitive impairment, and higher impulsivity.

Conclusion: These findings highlight variations among the importance of risk factor domains between suicide attempters with distinct trajectories.

Presenter Name/Degree(s): Jacques-Yves P. Campion, MD
Current Position: Postdoctorate Associate

Title: **Predicting worry-related mental states using regional brain activity with long short-term memory (LSTM) recurrent deep neural networks**

Author(s): *Campion JY[1,2,3], Desmidt T[2,3], Butters MA[1], Tudorascu DL[1,4], Karim HT[1,5], and Andreescu C[1]*
Affiliation(s): *[1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]UMR 1253, iBraiN, Université de Tours, Inserm; [3]Centre Hospitalier Régional Universitaire (CHRU) de Tours; [4]Department of Biostatistics, University of Pittsburgh; [5]Department of Bioengineering, University of Pittsburgh*

Introduction: Worry is common, spontaneous and evolutionary adaptive phenomenon. By contrast severe worry is a pathological condition prevalent in late-life mood and anxiety disorders. While previous research mapped neural correlates using traditional statistical approaches, this study employed an explainable long short-term memory (LSTM) neural network to predict worry-related mental states through brain activity timeseries.

Methods: The research involved two functional magnetic resonance imaging (fMRI) studies: FINA (n=116) and RAW (n=88), recruiting older adults across a worry spectrum. Participants underwent a clinical interview to identify personalized worry induction, neutral and reappraisal statements, which were presented in a block design fMRI task. Data were preprocessed and normalized in MNI space, extracting fMRI timeseries from 481 cortical, subcortical, and cerebellar regions. The LSTM model was bidirectional with two layers, using a 20-second sequence length, MAE loss, and 32-sample batch size, trained over 250 epochs. The FINA dataset was split into training and validation sets, with the RAW dataset serving as a test set.

Results: Model's accuracy in predicting mental states was, with validation and full length, AUC of 0.89 for worry, 0.77 for reappraisal, and 0.91 for neutral states. The independent test set showed lower performance with 0.78, 0.63, and 0.81. Key predictive networks for worry included dorsal attention, anterior hippocampus, cerebellum, left executive control, and basal ganglia networks.

Conclusion: The study's conclusion highlights the LSTM model's success in predicting worry-related mental states in older adults, potentially offering a neural marker for treatment response in severe worry. This approach provides a novel method for understanding complex worry-related brain activity patterns.

Presenter Name/Degree(s): Christopher Chae BS
Current Position: Medical Student

Primary Mentor in Psychiatry: Helmet Karim, PhD

Title: **Linking neural sensitivity to social rejection and acceptance to daily depressive symptoms**

Author(s): Chae C[1], Silk J[2], Cotter M[2], Karim HT[3,4], and Ladouceur CD[3]
Affiliation(s): [1]University of Pittsburgh School of Medicine; [2]University of Pittsburgh Psychology Department; [3]Department of Psychiatry, University of Pittsburgh School of Medicine; [4]University of Pittsburgh Department of Bioengineering

Introduction: Depression rates increase sharply during adolescence, with girls showing particularly elevated vulnerability. This heightened vulnerability is thought to result from both greater exposure to interpersonal stress and increased sensitivity to peer evaluation during this key developmental stage. Studies suggest links between depressive symptoms and heightened neural responses to social rejection in regions involved in emotion regulation and social cognition. We examined how neural sensitivity to social feedback is associated with depressive symptoms in a sample with varying risk for depression.

Methods: Participants were 108 adolescent girls (ages 12–17), majority at elevated risk for depression and suicidality from the greater Pittsburgh area as part of a larger longitudinal study. During fMRI, participants completed the Chatroom Interact (CHAT-I) task, a social evaluation paradigm in which they believed they were being accepted or rejected by peers based on shared interests. Whole-brain voxel-wise analyses examined neural responses to acceptance and rejection. Activation patterns were regressed onto depressive symptoms measured by the Mood and Feelings Questionnaire (MFQ).

Results: Compared to the control condition, both acceptance and rejection activated executive control and salience networks, including the dorsolateral and ventrolateral prefrontal cortex, caudate, and orbitofrontal cortex. Acceptance elicited greater activation than rejection in regions such as the cerebellum, inferior temporal gyrus, hippocampus, and orbitofrontal cortex. Rejection showed stronger activity only in the visual cortex. Preliminary analyses found that increased activation in the orbitofrontal cortex, precuneus, and inferior temporal gyrus during acceptance was associated with higher MFQ scores. Similarly, greater activation in the superior and inferior frontal gyrus during rejection was positively associated with depressive symptoms.

Conclusion: Our results show that adolescent girls with higher depressive symptoms exhibit greater neural sensitivity in specific regions during peer feedback. Increased activation during both acceptance and rejection suggests altered social processing, highlighting potential neural markers of depression-related social sensitivity.

Presenter Name/Degree(s): Omeed Chaichian, BS
Current Position: Research Specialist

Primary Mentor in Psychiatry: Fabio Ferrarelli, MD, PhD

Title: **Transcranial focused ultrasound neuromodulation in psychiatry: Main characteristics, current evidence, and future directions**

Author(s): Keihani A[1], Sanguineti C[1,2], Chaichian O[1], Huston CA[1], Moore C[1], Cheng C[1], Janssen SA[1], Donati FL [1], Mayeli A[1], Moussawi K[3], Phillips ML[1], and Ferrarelli F[1]
Affiliation(s): [1]Department of Psychiatry, University of. Pittsburgh School of Medicine; [2]Department of Health Sciences, University of Milan; [3]Department of Neurology, University of California

Introduction: Non-invasive brain stimulation (NIBS) techniques are designed to selectively target specific brain regions, thus enabling focused modulation of neural activity. Among NIBS technologies, low-intensity transcranial focused ultrasound (tFUS) can safely and non-invasively stimulate deep brain structures with millimetric precision, thus offering advantages in accessibility to non-cortical regions over other NIBS methods. However, there are only a handful of studies that have utilized tFUS in psychiatric populations.

Methods: This narrative review provides an up-to-date overview of key aspects of this NIBS technique, including the main components of a tFUS system, the neuronavigational tools used to precisely target deep brain regions, the simulations utilized to optimize the stimulation parameters and delivery of tFUS, and the experimental protocols employed to evaluate the efficacy of tFUS in psychiatric disorders.

Results: The tFUS studies on depression, which targeted both cortical (i.e., left DLPFC, right frontotemporal cortex) and subcortical (i.e., SCC, and ANT) regions, showed some improvements in depressive symptoms, sometimes lasting weeks or months after stimulation. Furthermore, only four studies utilized fMRI alongside clinical assessments, with mixed findings on brain connectivity changes after active tFUS , while in anxiety and SUD patients, the targeted brain regions, the amygdala and nucleus accumbens, respectively, induced a reduction in clinical symptoms after active stimulation, although no neural activity measures were examined.

Conclusion: Altogether, tFUS represents a safe, novel, and promising approach in neuromodulation, particularly in neuropsychiatric disorders, in which deep brain regions play a critical role. While preliminary studies have provided encouraging results, the field is still in its infancy, with much work needed to standardize protocols and demonstrate consistent efficacy.

Presenter Name/Degree(s): Britt Chamberlain
Current Position: MD/PhD Student

Primary Mentor in Psychiatry: Susanne Ahmari, MD, PhD

Title: **Medial orbitofrontal cortex representation of active avoidance and refinement over learning**

Author(s): ***Chamberlain B, Geramita M, Crummy E, Mohan K Namboodiri V, and Ahmari S***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Negative reinforcement-based theories of obsessive compulsive disorder (OCD) suggest that the manifestation and maintenance of compulsions is driven by temporary relief from obsession-related anxiety. Medial orbitofrontal cortex (mOFC) has emerged as a critical region underlying excessive avoidance in OCD, but how mOFC representations of avoidance emerge and are refined with learning remains unknown.

Methods: We performed calcium imaging in C57Bl6 mice (n = 17) that were trained to avoid foot shocks predicted by a cue light by lever-pressing within a cued period ('avoid response') or after shock delivery ('escape response'). Single-cell calcium fluorescence was extracted (CNMFe) and time-locked to task-relevant events. Individual neurons were tracked across sessions (CellReg). In a separate cohort of mice, we bilaterally lesioned mOFC with NMDA infusions to investigate mOFC necessity for avoidance learning.

Results: Avoidance learning is significantly impaired in mOFC-lesioned mice (25% acquisition rate vs. 83% for sham control, $p=0.0194$), supporting a causal role for mOFC in this task. Because mice transition from making escape to avoid responses over learning, we investigated whether discrete or overlapping populations of mOFC neurons encode these defensive behaviors. We found that only 3% of neurons are co-activated to avoid and escape responses, and response type can be successfully decoded from population activity (linear SVM F1-macro score compared to shuffled data: $p<0.0001$). As avoidance behavior is learned, mOFC avoidance representation strengthens and crystallizes, reflected by 1) an increased number of avoid-modulated neurons, 2) increased single-cell stability of avoidance encoding, and 3) increased population stability of avoidance encoding.

Conclusion: mOFC is necessary for active avoidance learning, and mOFC neurons have discrete representation of instrumental defensive responses (avoid vs. escape behaviors). Increased recruitment of avoid-modulated neurons and increased stability of avoidance encoding at single-cell and population level in late training sessions may reflect consolidation of a well-learned behavior following initial learning.

Presenter Name/Degree(s): Danielle A. N. Chapa, PhD
Current Position: Postdoctoral Scholar

Primary Mentor in Psychiatry: Andrea B. Goldschmidt, PhD

Title: **Exercise does not regulate affect: An ecological momentary assessment study of maladaptive exercise in women with eating disorders**

Author(s): ***Chapa DAN[1], Forbush KT[2], Chen Y[2], Costain CE[2], and Rasheed SI[2]***
Affiliation(s): ***[1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Psychology, University of Kansas***

Introduction: Maladaptive exercise is present in 50% of people with an eating disorder (ED). Maladaptive exercise includes exercise that is excessive or compulsive (i.e., completed at extreme intensities/durations or despite pain, illness, or social repercussions). When present, maladaptive exercise is associated with more severe ED pathology, slower rates-of-recovery, faster rates-of-relapse, and medical complications. Despite its detrimental impact on psychological and physical health, the function of maladaptive exercise is unknown, further limiting our ability to reduce maladaptive exercise behaviors effectively in treatment.

Methods: The objective of this study was to test the affect-regulation theory of exercise through a 7-day ecological momentary assessment study of women with EDs who engaged in frequent maladaptive exercise (***N***=84). Participants completed six, semi-random signal-contingent and event-contingent (pre- and post- exercise) surveys of positive and negative affect. Wrist-worn accelerometers were used to objectively monitor and identify periods of activity. Piecewise multilevel models were used to quantify the direction and magnitude of affective change in the hours leading up to and following engagement in exercise. In line with the affect-regulation theory, we hypothesized that negative affect would increase before and decrease after exercise.

Results: Unexpectedly, negative affect decreased in the hours prior to exercise and increased after exercise. Positive affect increased in the hours prior to exercise and decreased after exercise.

Conclusion: The current study offers preliminary longitudinal evidence that exercise may have a negative impact on mood for a subset of individuals with an ED. Results of this study highlight a need to better understand the function of maladaptive exercise and why individuals with EDs are not receiving the known psychological benefits of positive affect after exercise.

Presenter Name/Degree(s): Chang-Le Chen, MSc
Current Position: Doctoral Student

Primary Mentor in Psychiatry: Howard Aizenstein, MD, PhD, Dana Tudorascu, PhD and Minjie Wu, PhD

Title: **Periventricular diffusivity reflects APOE4-modulated amyloid accumulation and cognitive impairment in Alzheimer's continuum**

Author(s): Chen C-L[1], Son SJ[2], Schweitzer N[1], Jin H[1], Li J[1], Wang L[1], Yang S[1,3], Minhas D[4], Laymon C[1,4], Stetten G[1], Tudorascu DL[3,5], Aizenstein H[1,3], and Wu M[3]
Affiliation(s): [1]Department of Bioengineering, University of Pittsburgh; [2]Department of Psychiatry, Ajou University School of Medicine; [3]Department of Psychiatry, University of Pittsburgh School of Medicine; [4]Department of Radiology, School of Medicine, University of Pittsburgh; [5]Department of Biostatistics, University of Pittsburgh

Introduction: Impaired glymphatic clearance of interstitial solutes like amyloid-beta (Aβ) may contribute to Alzheimer's disease (AD) progression. The APOE4 allele, a major genetic risk factor for AD, may exacerbate this dysfunction. As an indirect marker, diffusion tensor imaging (DTI)-based metrics such as DTI-ALPS have shown promise in assessing glymphatic activity but are limited in spatial scope and robustness. This study generalizes the notion and introduces a novel periventricular diffusivity (PVeD) metric to capture fast diffusion signals along perivenous spaces and explores its association with Aβ deposition, APOE4 genotype, and cognitive impairment.

Methods: We analyzed multimodal neuroimaging and clinical data from two independent cohorts (BICWALZS, N=440; MCSA, N=414) spanning the Alzheimer's continuum. PVeD was derived from DTI data by quantifying the transverse diffusivity in periventricular white matter areas covering deep medullary veins. Associations with Aβ burden (via PET), cognitive measures, neurodegeneration (hippocampal volume), and APOE4 status were assessed using partial correlation, mediation, and interaction analyses, respectively. We further evaluated PVeD's ability to predict longitudinal cognitive decline. Findings were validated across cohorts differing in clinical characteristics, enhancing reproducibility.

Results: PVeD was significantly associated with cognitive performance, hippocampal atrophy, and Aβ burden. Notably, lower PVeD correlated with greater Aβ deposition, especially in APOE4 carriers, indicating a genetic modulation on the relationship between Aβ burden and diffusion process in the periventricular area. Mediation analysis revealed that Aβ burden partially mediated the relationship between PVeD and cognitive decline. Baseline PVeD also significantly predicted future cognitive deterioration in both cohorts. Compared to DTI-ALPS, PVeD demonstrated superior associations with core AD features and stronger sensitivity to APOE4 modulation effects on Aβ accumulation.

Conclusion: PVeD is a novel and robust diffusion marker reflecting glymphatic-related integrity, sensitive to APOE4 effects, and predictive of cognitive decline, offering promise for early detection and monitoring of Alzheimer's progression.

Presenter Name/Degree(s): Cynthia Cheng B.S.
Current Position: Research Specialist

Primary Mentor in Psychiatry: Fabio Ferrarelli, MD, PhD

Title: Rapid eye movement (REM) sleep characteristics in individuals with first-episode psychosis and healthy controls

Author(s): ***Cheng C[1], Mayeli A[1], Sanguineti C[1], Moore C[1], Wilson JD[2], and Ferrarelli F[1]***
Affiliation(s): ***[1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]University of San Francisco, Department of Mathematics and Statistics***

Introduction: Sleep disturbances are common in psychotic disorders and may be implicated in their pathophysiology While alterations in non-Rapid eye movement (REM) sleep have been well-studied in individuals with psychosis and schizophrenia, less is known about REM sleep, especially its phasic and tonic states. These REM subtypes show distinct brain activity but have not been explored in first-episode psychosis (FEP). This study uses high-density EEG to compare REM sleep patterns in individuals with FEP and healthy control (HC) subjects, aiming to better understand early changes in sleep-related brain function**.**

Methods: High-density sleep EEG (N=64 channels) was collected from twenty (FEP) and twenty HC participants. REM sleep was divided into 6-second epochs and labeled as phasic or tonic. Power spectral analysis was computed on both raw and Z-scored REM sleep EEG data. Linear mixed-effects models assessed group, REM state, and interaction effects, controlling for age and sex, with false discovery rate (FDR) correction across channels.

Results: No main Group effects were established; however, several State effects were observed. HC had more theta activity during phasic vs. tonic REM, while FEP participants showed more sigma during tonic vs. phasic REM. A group × state interaction was also observed in four central EEG channels for z-transformed theta power ($11.8<F<24.93$; adjusted $p<0.005$). Post-hoc analyses revealed higher phasic vs tonic theta power in these channels in FEP ($4.7<t<6.9$, adjusted $p<0.0001$), but not in HC ($-0.16<t<0.44$, adjusted $p>0.9$).. In FEP, stronger phasic theta was linked to better cognitive scores.

Conclusion: Individuals with FEP showed distinct REM sleep patterns, especially in theta activity, that were linked to cognitive performance. Altogether, these findings highlight the importance of studying REM microstructure in early psychosis.

Presenter Name/Degree(s): Lamia T. Choity, BS
Current Position: Research Specialist

Primary Mentor in Psychiatry: Thomas Karikari, PhD

Title: **Elevated p-Tau217 Disrupts Age-Related Resilience to Cognitive Decline in Older Adults**

Author(s): ***Choity L[1], Zeng X[1], Deek RA[2], Gu JM[1], Farinas MF[1], Nafash MN[1], Lafferty TK[1], Bedison MA[2,3], Mercurio RB[2,4], Matan C[1,2], Gogola A[2], Kofler JK[1,2], Tudorascu DL[2], Shaaban CE[,4], Lingler JH[2,4], Pascoal TA[1,2], Klunk WE[2,4], Villemagne VL[2], Berman SB[1,2], Sweet R[1], Snitz BE[1,2,4], Ikonomovic MD[1,2], Cohen AD[1,2], Kamboh MI[2,3,4], LopezOL[1,2,4], and Karikari TK[2]***
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]University of Pittsburgh; [3]School of Public Health, University of Pittsburgh; [4]University of Pittsburgh Alzheimer's Disease Research Center (ADRC)

Introduction: Plasma p-tau217 is a key biomarker for Alzheimer's Disease (AD), strongly associated with amyloid pathology and cognitive decline. While younger age typically offers protection against cognitive deterioration, how this protection interacts with elevated p-tau217 remains unclear. Understanding this relationship is critical for refining risk assessment and early intervention strategies.

Methods: Participants from the University of Pittsburgh Alzheimer's Disease Research Center underwent baseline plasma p-tau217 measurement and Clinical Dementia Rating (CDR) assessments. Follow-up CDR evaluations were conducted annually for up to 29 years (median: 3.0 years, IQR: 1.9-5.9). A subset (n=243) received neuroimaging for AD biomarkers. Kaplan-Meier and Cox proportional hazards models were used to examine the association between p-tau217, age (<70 vs. ≥70 years), and cognitive decline, defined by an increase in CDR global score.

Results: Among 2394 individuals (mean age: 71.8 ± 9.5 years; 58.3% female; 91.5% non-Hispanic White), both high p-tau217 and older age were linked to increased cognitive decline risk (HR = 3.16 [2.82–3.53] and HR = 1.67 [1.49–1.87], respectively). Age significantly modified the hazard posed by high p-tau217 (interaction $p < 0.0001$). In younger individuals, high p-tau217 was associated with a hazard ratio of 9.4 (5.9–15.0) and a median survival of 3.9 years, versus 15.8 years for low p-tau217. In older individuals, high p-tau217 corresponded to a hazard ratio of 4.9 (2.9–8.5) and a median survival of 4.0 years, versus 7.9 years for low p-tau217. Younger and older adults with high p-tau217 exhibited comparable cognitive decline rates ($p = 0.08$).

Conclusion: High p-tau217 levels nullify the protective effect of younger age on cognitive decline. These findings emphasize the need for early detection and monitoring of p-tau217 in younger older adults to facilitate timely interventions in AD management.

Presenter Name/Degree(s): Caroline Christian, PhD
Current Position: Postdoctoral Scholar

Primary Mentor in Psychiatry: Andrea Goldschmidt, PhD

Title: **Multivariate trajectory modeling of eating disorder symptoms across the perinatal period**

Author(s): Christian C[1], Bridges-Curry Z[2], Donofry SD[3], Call CC[1], Brown L[1], Hawkins MS[4], and Levine MD[1]
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Durham VA Medical Center; [3]RAND; [4]University of Pittsburgh, Department of Psychology

Introduction: The perinatal period is a critical risk period for changes in eating disorder (ED) symptoms. Some studies have found that ED symptoms worsen across pregnancy and postpartum, whereas others have found decreasing symptoms, or no change. Person-centered research is needed to better understand differential trajectories of perinatal ED symptoms.

Methods: The current study (***N*** = 315) involved completing online assessments of mental health, body image, and eating during pregnancy, eight weeks postpartum, and one year postpartum. Group-based multivariate trajectory models were used to identify differential trajectories of ED symptoms (i.e., weight concerns, shape concerns, eating concerns, restraint, binge eating, compensatory behaviors) from pregnancy through one year postpartum.

Results: Based on fit and classification indices, group size, and parsimony, the four-group solution was identified as the best model. The four groups in this model included: high baseline, decreasing ED symptoms group (34%), low baseline, increasing ED symptoms group (29%), early postpartum ED symptom exacerbation group (i.e., increased symptoms during eight-weeks postpartum relative to pregnancy and one-year postpartum; 22%), and early postpartum ED symptom alleviation group (i.e., decreased symptoms during eight weeks postpartum; 15%). Across groups, cognitive-affective ED symptoms (e.g., weight concerns) exhibited greater changes, compared to behavioral symptoms (e.g., binge eating).

Conclusion: This study provides insight into the heterogeneity of ED symptom change during the perinatal period. The most-common profile (characterized by high ED symptoms during pregnancy, which decreased postpartum) is consistent with past research suggesting ED symptoms often reduce or resolve during the postpartum period among individuals with an ED history. Future research should examine if identified trajectories are related to patient characteristics (e.g., ethnicity; socioeconomic status) or perinatal health outcomes. Additionally, it is important to test if assessment and intervention can lead to changes in trajectory outcomes, in order to inform and improve perinatal ED care.

Presenter Name/Degree(s): Daniel Wonjae Chung, MD PhD
Current Position: Assistant Professor of Psychiatry

Title: **Computational modeling of stimulus-locked and persistent gamma oscillation regimes reveals differential vulnerability to schizophrenia-associated synaptic perturbations**

Author(s): Chung DW[1] and Ermentrout GB[2]
Affiliation(s): [1]Translational Neuroscience Program, Department of Psychiatry, University of Pittsburgh; [2]Department of Mathematics, University of Pittsburgh

Introduction: Core cognitive deficits in schizophrenia (SZ) include impairments in visuospatial working memory (vsWM). Distinct aspects of vsWM rely on γ-oscillations with different dynamics: stimulus-locked γ-oscillations are essential for stimulus encoding, while persistent γ-oscillations maintain information in the absence of stimuli. Our prior computational study showed that multiple synaptic alterations found in the PFC of SZ synergistically (greater than the sum of individual impact) reduce the power of stimulus-locked γ-oscillations. In this study, we investigated whether this synergistic effect of synaptic alterations extends to both stimulus-locked and persistent γ-oscillations, and if these two types of γ-oscillations exhibit distinct vulnerabilities to such alterations.

Methods: We constructed a population model network of pyramidal neurons (PNs) and parvalbumin-expressing interneurons (PVIs) that can generate either stimulus-locked or persistent γ-oscillations. We then simulated the individual and combined impact of three synaptic alterations previously found in the PFC of SZ: lower excitatory synaptic strength to PVIs, lower inhibitory synaptic strength to PNs, and higher variability in excitatory synaptic strength across PVIs.

Results: By adjusting NMDA synaptic strength among PNs, the model generated either stimulus-locked or persistent γ-oscillations. The three synaptic alterations found in the PFC of SZ had a synergistic effect in reducing the power of both types of γ-oscillations, and this effect was mainly driven by lower excitatory synaptic strength to PVIs. Finally, persistent γ-oscillations exhibited a greater synergistic reduction in power than stimulus-locked γ-oscillations in response to SZ-associated synaptic alterations.

Conclusion: Our findings suggest that persistent γ-oscillations exhibit greater vulnerability to the synergistic effects of SZ-associated synaptic alterations than stimulus-locked γ-oscillations. Given that stimulus-locked γ-oscillations arise in early sensory areas while persistent γ-oscillations emerge primarily from PFC, this differential sensitivity implies that a greater magnitude of synaptic alterations may be required to disrupt γ-oscillations in early sensory areas than in the PFC in SZ.

Presenter Name/Degree(s): Becca Cole, B.A.
Current Position: Graduate Student

Primary Mentor in Psychiatry: Max Joffe, PhD

Title: **Oxycodone dependence alters Mu and Delta opioid receptor regulation of prefrontal cortex inhibitory transmission in a cell type-specific manner**

Author(s): ***Cole RH and Joffe ME***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine; Translational Neuroscience Program, University of Pittsburgh; Center for Neuroscience University of Pittsburgh***

Introduction: Opioid use disorder (OUD) is a chronic and relapsing brain disorder that is characterized by an inability to control drug use and an intense withdrawal syndrome upon cessation. While Mu opioid receptor (MOR)-based therapies alleviate somatic discomfort and attenuate craving in OUD, these treatments are less effective at addressing long-lasting psychological symptoms that maintain drug use, necessitating a more complete understanding of changes to opioid signaling that occur during dependence. The delta opioid receptor (DOR) system has been consistently linked to hedonic deficits in rodent models of affective and reward behavior, supporting a role for dysregulated DOR signaling in motivational and affective components of OUD. The prefrontal cortex (PFC) is heavily implicated in OUD, and a substantial preclinical literature has linked PFC function with affective and motivational behavior. Though existing studies have focused mainly on opioid actions at excitatory synapses, the PFC endogenous opioid system is strongly localized to GABAergic interneurons (INs) which tightly regulate PFC circuitry and affective behavior.

Methods: Whole cell patch-clamp electrophysiology, pharmacology, optogenetics

Results: We show that show that PFC MOR and DOR signaling suppresses spontaneous and evoked PFC inhibitory transmission through dissociable mechanisms. Furthermore, cell type-specific optogenetics revealed that PFC MOR and DOR signaling is synapse-specific and differentially expressed by PV, SST, and CCK-INs. An escalating dose regimen of oxycodone that produces physical dependence and motivational and affective behavior alters SST- and PV-IN MOR and DOR signaling in a cell type-specific manner, suggesting that cell- and receptor-specific adaptations to PFC GABAergic transmission may promote different facets of opioid-related behavior following dependence.

Conclusion: Ongoing work uses cell type- specific calcium imaging and chemogenetics to monitor PFC IN activity during the development of opioid dependence and withdrawal and probe their role in behavior.

Presenter Name/Degree(s): Kayla Conaty BA
Current Position: Research Coordinator

Primary Mentor in Psychiatry: Meryl A. Butters, PhD

Title: **Perceived hearing loss is associated with processing speed and executive functioning deficits in older adults with treatment-resistant late-life depression**

Author(s): *Conaty K[1], Bobrow A[1], Voineskos AN[2], Lavretsky H[3], Shimony J[4], and Butters MA[1]*

Affiliation(s): *[1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Center for Additions and Mental Health, University of Toronto; [3]University of California, Los Angeles, Department of Psychiatry and Biobehavioral Sciences; [4]Washington University School of Medicine Department of Psychiatry*

Introduction: Hearing loss has been identified as a modifiable risk factor for cognitive decline and dementia, but its impact within treatment-resistant late-life depression (TRLLD)—a subgroup at heightened risk for poor cognitive outcomes—remains unclear. This study examined whether perceived hearing loss was associated with cognitive performance across multiple domains in older adults with TRLLD.

Methods: Participants (n=396), aged 60 and older with TRLLD, completed a comprehensive neuropsychological battery assessing processing speed, attention, learning, memory, visuospatial ability, and executive functioning (RBANS and select D-KEFS tests). A subset of these participants (n=175) also completed a self-reported hearing assessment. Perceived hearing loss for both ears was assessed using a 1-6 rating scale per ear, summed, and then converted into a binary variable (hearing loss=Yes if total>3). Linear regression models were applied to evaluate the relationship between perceived hearing loss and cognitive performance, controlling for depression severity, education, race, and gender. All cognitive scores were age-normed

Results: Perceived hearing loss was significantly associated with poorer performance on measures of processing speed and attention (RBANS Coding subtest [β=-.437, p=.020] and D-KEFS Color Naming scaled score [β=-1.06, p=.047]) and executive functioning (D-KEFS Verbal Fluency scaled score [β=-1.22, p=.050]). However, these did not survive multiple comparison corrections. No significant associations were observed for memory or other executive function tasks (all p's >.1).

Conclusion: These findings suggest that even in a population with elevated baseline cognitive risk due to chronic depression, self-reported hearing difficulties are independently associated with slowed processing speed and executive dysfunction. While these associations didn't remain significant after adjusting for multiple comparisons, they align with prior research connecting hearing loss to cognitive decline. However, since hearing loss was self-reported, the impact on cognition may be underestimated, as older adults often underestimate hearing impairment and objective audiometric measures typically show stronger associations.

Presenter Name/Degree(s): Ana Paula Costa, PhD
Current Position: Postdoctoral Scholar

Primary Mentor in Psychiatry: Carmen Andreescu, MD and Meryl Butters, PhD

Title: Peripheral biomarkers of lipid dysregulation and inflammation in anxiety-related risk for Alzheimer's disease and related dementias

Author(s): ***Costa AP[1], Karim HT[1,2], Farinas MF[1], Zeng X[1], Butters MA[1], Gelhaus SL[3], Karikari TK[1], and Andreescu C[1]***
Affiliation(s): ***[1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Bioengineering, University of Pittsburgh; [3]Department of Pharmacology and Chemistry Biology, University of Pittsburgh***

Introduction: Anxiety and its phenotypes are risk factors for several major diseases of aging including cardiovascular and autoimmune diseases, as well as Alzheimer's Disease and related dementias (ADRD). However, little is known about the mechanisms underlying the association between anxiety and ADRD risk. Leveraging lipidomics and proteomics allows investigation of neurobiological markers contributing to ADRD risk among individuals with specific anxiety phenotypes (rumination, global anxiety, and worry; RAW).

Methods: We investigated the relationship between RAW and peripheral biomarkers of lipid dysregulation, inflammation and ADRD. Plasma from 42 participants was collected at baseline [19 high-RAW (PSWQ≥55 and/or RSQ>50 and/or HARS≥17) and 23 low-RAW]. Cross-validated Elastic Net regression and traditional multiple linear regression were applied to investigate associations between NULISAseq CNS Disease Panel 120, untargeted-lipidomics, and clinical outcomes, and their interaction with APOE4.

Results: Our analysis included data from 42 participants (54.8% females), with a mean age of 64.2 years. We identified a subset of inflammatory (contactin-2, TAFA5, IL-5, IL-9, CCL11, S100A2) and endothelial damage (SAA1, VCAM1) markers significantly associated with worry and rumination in APOE4 carriers, but not with global anxiety. Additionally, lipidomic profiling in plasma revealed multiple differential lipids that were substrates (phosphatidylcholine-PC, phosphatidylethanolamine-PE; $p<0.05$) and products (LysoPC-LPC, LysoPE-LPE; $p<0.05$) in the Lands Cycle for acyl-chain remodeling associated with RAW, adjusting for sex and age. Given the smaller sample, we focus on effect sizes and report ß>0.2.

Conclusion: Our results highlight a coordinated plasma lipid profile shift (decreased PC, PE; increased LPC, LPE) in high-RAW, suggesting disrupted membrane integrity and heightened neuroinflammation, consistent with changes observed in preclinical AD. Additionally, high-RAW APOE4 carriers showed alterations in markers of inflammation and endothelial damage, suggesting that chronic inflammation may contribute to early vascular dysregulation and serve as a biological pathway linking anxiety-related phenotypes to ADRD risk. Further research is needed to clarify these biological pathways and explore targeted prevention strategies.

Presenter Name/Degree(s): Amaya Crawford
Current Position: Undergraduate Lab Assistant

***Primary Mentor in Psychiatry*:** Marta Peciña, MD, PhD

Title: **Trait anhedonia dampens expectancy effects while anxiety amplifies reinforcement induced mood responses in depression**

Author(s): *Crawford A and Peciña M*
Affiliation(s): *Department of Psychiatry, University of Pittsburgh School of Medicine*

Introduction: Identifying clinical moderators of placebo responses—such as depression severity, anhedonia, and anxiety—is essential for improving treatment precision and clinical trial design in psychiatry. These factors may influence how individuals respond to expectancy-driven effects, with depression severity shaping cognitive biases, anhedonia affecting reward sensitivity, and anxiety altering attention to contextual cues. Understanding these moderators can enhance our ability to predict placebo responsiveness, refine therapeutic strategies, and improve the interpretability of trial outcomes.

Methods: Fifty unmedicated participants (24 female, 26 male) with symptoms of depression completed the Mood and Anxiety Symptom Questionnaire (MASQ) prior to participating in an "antidepressant placebo fMRI experiment." This task manipulated treatment expectancies of fast-acting antidepressant effects using visually cued placebo infusions and sham neurofeedback, while capturing trial-by-trial expectancy and mood ratings.

Results: Mixed effects models revealed that higher baseline anhedonia moderated the effects of both placebo and reinforcement conditions. Greater anhedonia predicted reduced placebo effects on expectancy ($b = -0.36$, $z = -5.96$, $p < 0.001$) and marginally reduced placebo effects on mood ($b = -0.11$, $z = -1.92$, $p = 0.05$), while enhancing reinforcement-related expectancy effects ($b = 0.38$, $z = 6.14$, $p < 0.001$). Baseline anxious symptoms did not significantly moderate placebo effects on expectancy ($b = 0.08$, $z = 1.12$, $p = 0.26$) or mood ($b = 0.01$, $z = 0.09$, $p = 0.93$), but significantly enhanced reinforcement-related effects on expectancy ($b = 0.17$, $z = 2.60$, $p = 0.01$) and mood ($b = 0.32$, $z = 5.49$, $p < 0.001$). Anxious arousal was associated with increased placebo effects on expectancy ($b = 0.30$, $z = 4.25$, $p < 0.001$) and reinforcement-related effects on mood ($b = 0.23$, $z = 4.06$, $p < 0.001$), but did not significantly moderate other condition effects. Greater baseline depressive symptoms were associated with increased reinforcement-related expectancy effects ($b = 0.24$, $z = 3.78$, $p < 0.001$), with no other significant interactions observed.

Conclusion: These findings suggest that symptom-specific factors—particularly anhedonia and anxiety-related dimensions—moderate expectancy-driven mood and expectancy responses in individuals with depressive symptoms. Anhedonia appears to blunt responsiveness to placebo cues while enhancing sensitivity to reinforcement, whereas anxious arousal and general anxiety amplify expectancy effects, especially under reinforcement. Depression severity showed limited influence, moderating only reinforcement-related expectancy. These moderators may be critical for tailoring expectancy-based interventions and optimizing clinical trial outcomes in depression.

Presenter Name/Degree(s): Elizabeth Crummy, PhD
Current Position: Postdoctoral Scholar

Primary Mentor in Psychiatry: Susanne Ahmari, MD, PhD

Title: **Investigating the neural substrates of active avoidance in the bed nucleus of the stria terminalis**

Author(s): ***Crummy E, Chamberlain B, Geramita M, and Ahmari S***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Compulsive behaviors performed to alleviate anxiety and avoid perceived threats are a characteristic symptom of Obsessive-Compulsive Disorder (OCD). The bed nucleus of the stria terminalis (BNST) is a promising treatment target for OCD due to its role in regulating anxiety, and deep brain stimulation of the BNST can reduce OCD symptom severity. How active avoidance behaviors are encoded and modulated in BNST neurons is unknown.

Methods: We performed calcium imaging in the BNST of C57BL6 mice (n=14) during acquisition of an active avoidance task (7 sessions). Mice learned to avoid or escape footshocks by pressing during cue periods (avoid) or during shock (escape). We evaluated cell modulation during lever presses and used a support vector classifier to determine BNST decoding of avoid vs. escape. Cells tracked across sessions were used to generate trajectories of population activity during avoidance. Trajectories for sessions 1, 6 and 7 were fit into shared principal component space (PCA) to examine changes over acquisition. Finally, a separate cohort of mice received optogenetic stimulation of the BNST during the cued avoid period to determine if BNST stimulation impaired avoidance.

Results: Escape-modulated and avoid-modulated BNST neurons are largely selective to response type, and response type can be decoded based on day 1 lever-press activity (f1 compared to shuffled data: $p<0.0001$). When plotted in PCA space, population trajectories during avoidance were more similar between days 6 and 7 than between days 1 and 7 ($p=0.01$). Overall, avoid-inhibited cell proportion increased and average activity decreased following acquisition. Finally, stimulating BNST neurons reduced avoid responses and increased response latency.

Conclusion: BNST neurons distinctly encode avoid and escape behaviors, and BNST activation impairs avoidance. BNST population activity changes as avoidance becomes well-learned. This furthers our understanding of how avoidant behaviors develop and may inform how these behaviors become maladaptive in OCD.

Presenter Name/Degree(s): Leonardo D'Aiuto, PhD
Current Position: Assistant Professor of Psychiatry

Title: **Non-canonical functions of Tau**
Author(s): ***D'Aiuto L[1], Filipponi C[1,2], and Stamm S[3]***
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Retrovirus Center, Department of Translational Research and New Technologies in Medicine and Surgery, University of Pisa; [3]University of Kentucky, Molecular and Cellular Biochemistry

Introduction: The tau protein, encoded by the MAPT gene, is a microtubule-associated protein primarily expressed in neurons, whose canonical functions include stabilizing axonal microtubules and regulating intracellular transport, critical for maintaining neuronal structure and function. Tau exists in six isoforms generated by alternative splicing. These isoforms and tau's phosphorylation states modulate its affinity for microtubules, influencing cytoskeletal dynamics. Beyond these well-characterized roles, **tau exhibits noncanonical functions that expand its biological significance**. Tau is present in the nucleus, where it binds nucleic acids, contributing to chromatin compaction and DNA protection under stress conditions. Furthermore, the MAPT gene generates circular RNAs (circRNAs) through backsplicing events, that are translated into aggregation-promoting proteins implicated in tau pathology. Exon 12 can be backspliced to either exon 7 or 10, generating two circRNAs that are translated into proteins. An abundant circular RNA from the tau gene is generated by backsplicing of exon 9 to 5, generating a protein composed of the first microtubule domain R1 and a circRNA-specific part. These circTau proteins have been shown to promote linear tau aggregation in vitro, suggesting a potential role in neurodegenerative diseases.

Methods: We employed brain organoids to investigate tau-mediated responses to herpes simplex virus 1 (HSV-1) infection, the virus most strongly associated with Alzheimer's diseases.

Results: HSV-1 infection caused nuclear accumulation of phosphorylated tau (p-tau) in neurons and neural precursor cells. Chromatin immunoprecipitation analysis indicated an interaction of p-tau with the viral chromatin HSV-1 infection. Furthermore, HSV-1 infection caused circTau 9->5 translation, suggesting that the virus induces cap-independent translation of circRNAs. The resulting protein binds to microtubules and RNA. RNA bound to circTau9->5 encoded protein could act as the anion needed to promote tau aggregation.

Conclusion: Our findings suggest that MAPT may regulate the host stress response during HSV-1 infection by modulating viral life cycle through non-canonical functions.

Presenter Name/Degree(s): Joshua A. Daniel, BA
Current Position: Research Assistant III

Primary Mentor in Psychiatry: Jamie Zelazny, PHD, MPH, RN

Title: **GET ActivE: Testing of a behavioral activation app for youth**

Author(s): *Daniel J[1], Gayle K[2], Boyd R[3], Jonassaint C[2], George-Milford B[1], Hamm M[2], Luiggi-Hernández J[2], Cameron F[2], Mehalko J[1], Zelazny J[3], and Brent D[1]*
Affiliation(s): *[1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]University of Pittsburgh Department of Medicine; [3]University of Pittsburgh School of Nursing; [4]Children's Hospital of Philadelphia*

Introduction: Depression is a prevalent and increasing mental health condition among adolescents. A major symptom is anhedonia, or loss of interest in previously enjoyable activities. Behavioral activation targets anhedonia by reducing isolation and increasing engagement in enjoyable activities. Digital BA is a scalable, low-cost treatment but has not been tested in diverse adolescent populations.

Methods: This study tests the efficacy of digital BA for reducing anhedonia among adolescents using a 3-phase approach. Phase 1 involved interviews to identify barriers and facilitators to implementing the GETActivE program, which uses an app called VIRA that passively collects data from sensing technology. In Phase 2, feedback from these interviews informed improvements to the GETActivE app. Phase 3 is a randomized pilot trial comparing GETActivE to a control condition. The intervention includes app-based health coaching that uses participants' mood and activity data to suggest mood-boosting activities and support behavioral activation.

Results: For Phase 1, interviews were conducted with 19 providers, 10 youth, and 10 parents. Both teens and parents found GETActivE beneficial overall but expressed concerns about privacy and data security while also giving preferences for health coach characteristics**.** The providers also suggested that depression should be the primary focus, rather than behavioral activation. During Phase 2, the usability phase, 15 participants were consented, and 10 completed the 4-week intervention. Overall, interview feedback was positive; however**,** some participants encountered technical difficulties with receiving notifications from the app.

Conclusion: Interviews from Phases 1 and 2 provided data to inform adaptations to the GETActivE app and the health coaching protocol to enhance intervention effectiveness. Aim 3 recruitment began in March 2025 with a randomized pilot trial involving 75 participants and a 2:1 allocation ratio, comparing the GETActivE intervention with activity monitoring alone. This trial will inform GETActivE's effectiveness in promoting healthy behaviors and improving mental health.

Presenter Name/Degree(s): Aanika Das
Current Position: Undergraduate

Primary Mentor in Psychiatry: Danella Hafeman MD, PhD

Title: **The relationship between traumatic life events and mood lability in adolescents**

Author(s): ***Das A, Merranko J, Vaughn-Coaxum R, Ruliffson M, and Hafeman D***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Previous research indicates that childhood traumatic events are associated with the onset of mood disorders and increased mood lability. Mindfulness-based interventions (MBI) can positively affect emotion regulation, behavioral problems, and mood disorders in adolescents. Mindfulness increases emotion regulation strategies and decreases stress responses. We expect to find mindfulness as a buffer between Traumatic Life Events (TLEs) and higher mood lability.

Methods: 73 adolescents (11-14 years old) at familial risk for a mood disorder (MDD or BD) were assessed for lifetime traumatic events using the K-SADS. Both parents and children reported on child mood lability via the Child Affective Lability Scale (CALS), Difficulties in Emotional Regulation Scale (DERS) - child reported, and Emotion Dysregulation Inventory (EDI) - parent reported. Mindfulness was assessed using the Child and Adolescent Mindfulness Measure (CAMM), which only the child completed. We used linear mixed models to assess the moderation of mindfulness in the relationship between TLEs and mood lability.

Results: The total number of TLEs was positively associated with CALS Parent (β=0.18, p=0.08) and EDI (β=0.26, p=0.02). The self-reported mindfulness (CAMM) moderated the effect of TLEs in CALS-P (p=.02). The association between TLEs and parent-reported mood lability was stronger in those with the lowest 25% of CAMM scores. There were no significant relationships between TLEs and child-reported mood lability. The total number of TLEs was categorized into clusters; abuse was the only predictor of higher mood lability in parent reports (CALS: β=0.79, p=0.002; EDI: β=1.04, p<0.0001).

Conclusion: Based on the positive relationship between TLEs and mood lability only existing in the group with the lowest mindfulness, and not in the rest of the population, mindfulness had a buffering effect on TLEs and mood lability. In line with previous literature, abuse seems to be a strong predictor of higher levels of mood lability in adolescents.

Presenter Name/Degree(s): Emalee Dauginikas, MSc
Current Position: Research Specialist

Primary Mentor in Psychiatry: Lia Ahonen, PhD

Title: **An explorative analysis of sibling's impact on an individual's emotion regulation**

Author(s): ***Dauginikas E, FitzGerald D, and Ahonen L***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Due to the complexity of a sibling relationship, it is recognized that an interdependence exists between sibling and parental subsystems that fosters a child's development (Kramer et al., 2024). Healthy sibling relationships play a positive role in promoting emotional resilience (McHale, Updegraff, & Whiteman, 2012). While most children in the United States live with at least one sibling, (79.8%; U.S. Census Bureau, 2020) studies of family structure often overlook an ecological perspective of family systems. Family relationships may not just be defined in technical terms, but rather personal and shared histories. This explorative analysis investigates to what extent the traditional definition of sibling (e.g. biological sibling) is valid and how it compares to previous conclusions about a sibling's impact on an individual's social-emotional development.

Methods: The Adolescent Brain Cognitive Development –Social Development study (ABCD-SD) is a prospective study on adolescent delinquency and victimization, and relationships to the developing brain. The ABCD-SD study (N=2,426) collected report of sibling status, child report of difficulties in emotion regulation (DERS), and child report of parenting styles (APQ) in five geographical locations in the U.S. Independent samples t-tests were conducted to compare child's emotion regulation and parenting style scores across sibling and non-sibling groups (ages 10 to 12, 49.8% female).

Results: Contrary to previous research, no differences in sibling status were found (p=0.7) in relationship to emotion regulation. In addition, no significant differences were found between sex, age, parenting styles, or emotion regulation difficulties across groups.

Conclusion: Results suggest that the definition of sibling relationship may have evolved over the last decade. Researchers must pose different questions to capture the quality of relationships, the lived experience of families, and begin to build upon the science of genetics within social relationships.

Presenter Name/Degree(s): Megan Deam, MA
Current Position: Lab Manager

Primary Mentor in Psychiatry: Leslie Horton, PhD

Title: **Five-year trajectories of psychotic-like experiences: The influence of negative life events on screen use**

Author(s): ***Deam M, Gupta T, Seah THS, Griffith J, and Horton L***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Over the past two decades, the increase of adolescent screen use as well as technological advances, particularly the rise of social media platforms, have significantly increased adolescent screen time, with research suggesting both beneficial and harmful effects of screen time on adolescent mental health. Youth with distressing psychotic-like experiences (PLEs) are at risk for unfavorable long-term outcomes including the emergence of psychosis and often report experiencing negative life events (e.g., trauma).

Methods: Here, we examined the influence of screen use and negative life events on five-year trajectories of PLE-related distress levels in a large community sample of children and adolescents from the Adolescent Brain Cognitive Development (ABCD) study (N=11,866).

Results: Latent Growth Mixture Models revealed three PLE distress trajectories across five-years: increasing PLEs, decreasing PLEs, and no PLEs. The increasing and decreasing PLE trajectories reported similar amounts of average screen use per day, both of which had higher levels of screen use compared to the no PLE trajectory, $ps<0.001$. The increasing PLE trajectory was characterized by more incidents of negative life events compared to the decreasing PLE trajectory, $F(1,321)=4.25$, $p=0.04$, $\eta p2=0.01$, and the associations between screen time on the weekends (including texting and social media use) at ages 11-12 and PLE distress levels at ages 13-14 were strongest for people who were exposed to more incidents of negative life events, $ps=0.006$-0.036.

Conclusion: These findings suggest that the timing, type, and context of screen use may play a more important role in the progression of PLE distress levels than screen time quantity alone. These results underscore the importance of continuing to understand intersections between digital behaviors and environmental contexts such as a life stressor in shaping adolescent mental health outcomes, and indicate a need to examine both positive and negative impacts of adolescent screen use.

Presenter Name/Degree(s): Gabrielle Des Ruisseau, BS
Current Position: Research Project Assistant

Primary Mentor in Psychiatry: Adriane Soehner, PhD

Title: **Circadian dysregulation and mood outcomes in young people at risk for bipolar disorder**

Author(s): ***Des Ruisseau G, Keller L, Kuzemchak M, Wallace ML, Sollie B, Caswell A, Chan S, Hasler B, and Soehner A***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Delayed and unstable circadian timing have been implicated as mechanisms driving mood impairment in bipolar disorder. However, there has been extremely limited investigation into whether relationships between circadian dysregulation and mood extend to atrisk samples. Using dim light melatonin onset (DLMO) as a biological measure of endogenous circadian phase, we evaluated whether circadian timing and stability were cross-sectionally associated with mood symptoms in young people at clinical high-risk for bipolar disorder.

Methods: In an ongoing study, [n=96] participants aged 16-24yr [M = 21.9; SD = 2.1] were recruited across a spectrum of non/low-to-high lifetime subthreshold mania symptoms (Mood Spectrum Measure-Lifetime Version; MOODS). The MOODS Mania and Depression subscales evaluated lifetime severity of these mood symptoms. Past-week clinician-rated depressive symptoms were measured using the Hamilton Depression Rating Scale (HDRS) and manic symptoms (Young Mania Rating Scale; YMRS) were also assessed. Participants completed two salivary DLMO assessments to evaluate circadian phase, weekday and weekend phases were measured and averaged to assess overall DLMO. All models were covaried for age and sex. Lifetime mood symptoms were covaried for current symptoms and vice versa.

Results: Later circadian timing was associated with greater past-week clinician-rated depressive symptom severity (β=0.89; p = 0.005).

Conclusion: These interim findings indicate that delayed circadian timing be a potential risk factor or severity marker for depressive symptoms.

Presenter Name/Degree(s): Jillian DeSerio, BS
Current Position: Research Specialist & Doctoral Trainee

Primary Mentor in Psychiatry: Heather Joseph, D.O.

Title: More than mom brain: A qualitative exploration of the experiences of ADHD and motherhood in Pittsburgh

Author(s): ***DeSerio JN[1], Fling K[2], Mannion KA[1], Wilson MA[1], Chronis-Tuscano A[2], Joseph HM[3], and Lorenzo NE[4]***
Affiliation(s): ***[1]University of Pittsburgh Medical Center; [2]Department of Psychology, University of Maryland; [3]Department of Psychiatry, University of Pittsburgh School of Medicine; [4]Department of Psychology, American University***

Introduction: There are notable gaps in care for women with ADHD in the perinatal period. Women are increasingly receiving delayed diagnoses of ADHD during young adulthood, and many discontinue pharmacotherapy in pregnancy. The absence of interventions tailored to pregnancy and ADHD, combined with the increased responsibilities and emotional challenges accompanying the perinatal period, leaves many mothers struggling with their symptoms while navigating motherhood.

Methods: The Moms Managing ADHD (MomMA) study was developed to address this underserved and under-researched population. Focus groups were conducted with three behavioral health therapists from OB clinics, two OB-GYNs, and ten mothers with ADHD. Information on provider screening, assessment, and intervention processes, as well as patient histories, experiences, symptom manifestation and management, was collected. The focus groups were guided by a patient-centered care framework, emphasizing diverse lived experiences and individual context in shaping a pilot intervention.

Results: Many mothers reported experiencing long-standing symptoms of ADHD before a formal diagnosis, often receiving one in adulthood through self-advocacy or following their child's ADHD diagnosis. Mothers faced challenges accessing resources, difficulty transitioning off medication, and symptom exacerbation due to the demands of motherhood. Providers identified barriers to care, such as limited training and knowledge about ADHD and billing and time constraints. Providers a lso shared they often refer pregnant patients to psychiatry for ADHD and/or medication-related concerns. Mothers frequently felt overwhelmed, frustrated by ineffective management strategies, and unsupported in their diagnosis, yet demonstrated incredible resilience. They emphasized a desire for earlier, individualized, flexible support, such as telehealth sessions from providers knowledgeable in ADHD and motherhood, along with structured resources for symptom management.

Conclusion: A behavioral intervention tailored for expectant women with ADHD is needed and desired by both women with ADHD and perinatal providers to improve symptom management, daily functioning, and emotional well-being for mothers with ADHD, and ultimately their children.

Presenter Name/Degree(s): Hannah Dewhurst, BS
Current Position: Research Specialist

Primary Mentor in Psychiatry: Ann Cohen, PhD

Title: **The female advantage in verbal memory across learning and recall of the California verbal learning test**

Author(s): ***Dewhurst HE[1], Natarajan N[2,3], Gogniat MA[1], Lopez OL[1], Cohen AD[2], and Snitz BE[1]***
Affiliation(s): [1]Department of Neurology, University of Pittsburgh School of Medicine; [2]Department of Psychiatry, University of Pittsburgh School of Medicine; [3]Department of Pathology, University of Pittsburgh School of Medicine

Introduction: Research has demonstrated sex differences in the ability to recall verbal information, with females outperforming males on memory tasks, but this phenomenon is usually investigated by measuring delayed recall only. Focusing only on delayed recall omits important context to understand how sex could differently impact learning and recall separately. This project examines sex differences in other aspects of a word list recall test to better understand what parts of verbal learning and recall are involved in the female advantage in verbal memory.

Methods: Verbal memory scores from a sample of older adults (N=337, 51.6%Female) were examined. Adjusting for age (M=78, SD=8.99), race (84% White) and education (M=15 years, SD=2.7) scores from the California Verbal Learning Test (CVLT) were compared between sexes in cognitively unimpaired (CU) individuals (n=228) and participants diagnosed with MCI (n=109). Individual learning trial scores were examined, along with short delay free and cued recall, long delay recall, hits and false positives, recognition discrimination (hits minus false positives), and percent retained (highest learning trial divided by delayed recall).

Results: In adjusted ANOVA models, long delay recall scores of the CVLT were higher in females than males (p=0.01) in the CU group. CU females outperformed males in learning trials two (p=0.009) and five (p=0.01), short delay cued recall (p=0.02), recognizing false positives (p=0.01), and recognition discrimination (p=0.005). In the MCI group, females scored higher on learning trial two (p=0.008), three (p=0.02), and four (p=0.01), and total words learned (p=0.04), but not long delay recall.

Conclusion: The female advantage in verbal memory recall was found in CU but not mildly impaired older adults. Females outperformed males on some learning trials in both CU and MCI groups, suggesting that while recall might be affected by female early vulnerability to cognitive impairment in aging, an advantage in learning might be maintained.

Presenter Name/Degree(s): Jacinta Dickens, PhD
Current Position: Postdoctoral Scholar

Primary Mentor in Psychiatry: Sarah Stahl, PhD

Title: **Trajectories of insomnia and loneliness across a 12-week intergenerational dialog-driven intervention**

Author(s): ***Dickens J, Mizuno A, and Stahl S***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: As geriatric healthcare increasingly embraces a person-centered approach it is necessary to focus on older adults' perceived sense of loneliness. Loneliness is prevalent in late life and associated with substantial mental and physical health risks. Loneliness can also be associated with increased stress which may be related to sleep. This study examines the changes in insomnia severity over a 12-week intergenerational dialog intervention and their association with loneliness in older adults.

Methods: We analyzed data from 26 older adults (age ≥ 60) who lived alone or reported elevated loneliness (UCLA Loneliness Scale > 28). Insomnia severity (Insomnia Severity Index) and loneliness were assessed over a 12-week intergenerational dialog intervention (focused on improving loneliness). A linear mixed-effects model included time and loneliness as fixed effects, with random intercepts for participants.

Results: Insomnia symptoms significantly improved over time (β = –0.20, $\boldsymbol{p}$ = .001). Higher loneliness scores were associated with greater insomnia severity across the intervention period (β = 0.15, $\boldsymbol{p}$ = .003), suggesting that participants who experienced more loneliness tended to report worse sleep. An additional model including a week × loneliness interaction term found no significant interaction ($\boldsymbol{p}$ = .86), indicating that the association between loneliness and insomnia remained stable across the intervention.

Conclusion: Loneliness and insomnia are closely related. Insomnia improved during our intervention suggesting that targeting/improving loneliness may have other health benefits for older adults including improved sleep. Addressing perceived loneliness through person-centered approaches may also enhance quality of life, though future research is needed to clarify the directionality of these effects.

Presenter Name/Degree(s): Dominique DiDomenico, B.S.
Current Position: Research Specialist

Primary Mentor in Psychiatry: Mary L. Phillips, M.D., M.D.

Title: **Trauma exposure and related context processing disruptions**
Author(s): ***DiDomenico D, Fiske ME, Afriyie-Agyemang Y, Bertocci MA, Chase HW, Stiffler RS, Graur S, and Phillips ML***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Trauma exposure can result in alterations to cognitive control processes and disturbances in attention. Psychologically stressful events require heightened attention to environmental stimuli and cues that symbolize the event. Trauma exposed individuals may fail to react to certain contextual information. The AX-Continuous Performance Task(AXCPT) is a validated computerized test used to measure context processing and cognitive control mechanisms. The errors participants incur indicate differential deficits with executive processing; "AY" errors reflect an inability to inhibit a conditioned response, while "BX" errors reflect an inability to maintain attenuated activity. There has been little research exploring the relationship between trauma exposure and context processing. The present analysis explores which types of errors occur more frequently for participants with a history of trauma exposure.

Methods: From a larger study(CR18110024-046) at the University of Pittsburgh, 144 young adults with bipolar disorder(n=27) and those at risk(n=117), aged 23.63(sd3.42), 91 female, completed the Trauma History Questionnaire(THQ) and the AX-Continuous Performance Task(AXCPT). Error transformed variables were calculated in SPSS to compare performance on the task with consideration to the errors that frequently occur: recency errors(d'context), priming errors(A-cue bias), and the Proactive Behavioral Index(PBI). Regression models were run for these variables, with predictors of age, gender, IQ, BPSD diagnosis, and THQ.

Results: Participants with greater trauma exposure(THQ total) demonstrated a tendency to make more recency errors(d'context; β=.071, ***p***<.05) on the AXCPT. Trauma was not influential to the number of priming errors made(A-cue bias; β=.009, ***p***=.331), or the type of control used(proactive/reactive) (PBI; β= -.014, ***p***=.346).

Conclusion: Trauma history influenced participants' ability to discriminate between the AX and BX trials in our sample. Those with more instances of trauma demonstrated difficulty maintaining the cue, and in turn made more recency errors, suggesting an inability to preserve previous stimuli and use contextual information to drive their behavior.

Presenter Name/Degree(s): ***Xiaoshan Ding***
Current Position: Undergraduate Student Researcher

Primary Mentor in Psychiatry: Daniel Wonjae Chung, MD, PhD

Title: **Pilot study to assess alterations in cortical and thalamic excitatory inputs to parvalbumin-expressing interneurons in prefrontal and primary visual cortices of schizophrenia**

Author(s): ***Ding X, Lewis DA, and Chung DW***
Affiliation(s): ***Translational Neuroscience Program, Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Deficits in visuospatial working memory (vsWM) in schizophrenia (SZ) are linked to lower γ-oscillation power in the prefrontal (PFC) and primary visual (V1) cortices. Generation of γ-oscillations depends on the proper excitatory drive to parvalbumin-expressing interneurons (PVIs). Prior work showed fewer cortical excitatory inputs, defined by VGlut1+/PSD95+ puncta, onto PVIs in the PFC of SZ. However, it remains unknown whether thalamic excitatory inputs, defined by VGlut2+/PSD95+ puncta, onto PVIs are also altered, and whether these changes are present in V1. To address these questions, we conducted a pilot study of cortical and thalamic excitatory inputs to PVIs in the PFC and V1 of four pairs of SZ and unaffected comparison (UC) subjects.

Methods: Fluorescent immunohistochemistry, confocal microscopy, and post-image processing were used to quantify VGlut1+/PSD95+ and VGlut2+/PSD95+ puncta density per surface area of PVIs, and their volume and protein intensity, in the PFC and V1.

Results: In SZ, the density of VGlut1+/PSD95+ puncta on PVIs was significantly lower in PFC (-48%) and to a greater degree in V1 (-69%). Also, VGlut1+ puncta volume and intensity were lower in both regions, with a greater reduction in V1 (volume: -45%, intensity: -43%) than in PFC (volume: -33%, intensity: -25%). Although the density of VGlut2+/PSD95+ puncta on PVIs was lower in both regions in SZ, these differences were not statistically significant. Finally, VGlut2+ puncta intensity was significantly lower in V1 (-55%), while VGlut2+ puncta volume in V1 and both the volume and intensity of VGlut2+ puncta in PFC did not differ between subject groups.

Conclusion: Findings from this pilot study suggest that the strength of both cortical and thalamic excitatory inputs to PVIs is lower in PFC and V1 of SZ, with greater deficits in V1 than in PFC. Validation in a larger cohort is needed to confirm these preliminary findings.

Presenter Name/Degree(s): Yiwen Dong, ScM
Current Position: PhD Student in Biostatistics

Primary Mentor in Psychiatry: Dana L Tudorascu, PhD

Title: **The association between amyloid and physical activity in a racially diverse cohort of older adults**

Author(s): ***Dong Y[1], Wu Q[1], Minhas D[2], Laymon CM[2,3], Villemagne V[4], Lopez O[5], Snitz B[5], Gogniat MA[5], Glynn NW[6], Tudorascu DL[1,4], and Cohen AD[4]***
Affiliation(s): [1]Department of Biostatistics and Health Data Science, School of Public Health, University of Pittsburgh; [2]Department of Radiology, University of Pittsburgh School of Medicine; [3]Department of Bioengineering, Swanson School of Engineering, University of Pittsburgh; [4]Department of Psychiatry, University of Pittsburgh School of Medicine; [5]Department of Neurology, University of Pittsburgh School of Medicine; [6]Department of Epidemiology, School of Public Health, University of Pittsburgh

Introduction: Physical activity (PA) is proposed to be an effect modifier of Alzheimer's disease (AD) risk, however, it remains unclear if this modification is a result of a direct impact on AD pathophysiology or through indirect mechanisms, like increased cognitive reserve or reduced cardiometabolic risk. In the present study of racially diverse older adults. we explored the association of PA and amyloid deposition measured by [11C] Pittsburgh Compound B-PET (PiB).

Methods: This study included 238 participants from Hearscore (HS, n=115) and the Human Connectome Project (HCP, n=123). Participants received PiB imaging on 2 scanners (Siemens HR+/Biograph mCT) to assess amyloid-β (Aβ) deposition. PA data were collected using wrist-worn Actiwatch (HCP) and ActiGraph GT9X (HS). PA metrics included daily step count, light, moderate, moderate-to-vigorous physical activity (MVPA), and inactivity minutes. Aβ deposition SUVRs were adjusted using Transfer Learning ComBat (TL-ComBat) to reduce scanner variability. Spearman's correlations with bootstrapped 95% CIs assessed associations between PA and Aβ. The associations between Aβ deposition in various brain regions and PA were further explored by using Multinomial logistic regression, with age, sex, ApoE4 status, race, and education adjusted.

Results: Higher PA levels were significantly associated with lower Aβ deposition across multiple brain regions. In multinomial logistic regression, each one-unit increase in moderate activity time (64.5 minutes) and MVPA (64.6 minutes) was associated with a 47.6% higher likelihood of being in the low Aβ group vs. high, and a 44.9% higher likelihood of being in the medium vs. high group. Comparison demonstrated that TL-ComBat more effectively reduced scanner-related variability.

Conclusion: This study provides evidence that higher PA levels, including daily steps and MVPA, are associated with lower Aβ deposition in a racially diverse cohort. And considering that the skewness observed in the brain imaging data, applying TL-Combat proved more effective in reducing scanner variability.

Presenter Name/Degree(s): Kevin F. Dowling, BA
Current Position: MD/PhD Candidate

Primary Mentor in Psychiatry: David A. Lewis, MD

Title: **Patterns of differential gene expression and co-expression in layer 3 pyramidal neurons across 3 regions of the human cortical visuospatial working memory network in schizophrenia**

Author(s): Dowling KF[1,2], Enwright JE[1], Arion D[1], and Lewis DA[1,3]
Affiliation(s): [1]Translational Neuroscience Program, Department of Psychiatry, University of Pittsburgh, School of Medicine; [2]Medical Scientist Training Program, University of Pittsburgh; [3]Department of Neuroscience, Dietrich School of Arts and Sciences, University of Pittsburgh

Introduction: Schizophrenia (SZ) is characterized by impaired visuospatial working memory (VSWM). VSWM depends on coordinated transfer of information across a hierarchical network of cortical regions, including primary visual (V1) and association cortices (posterior parietal cortex, PPC; dorsolateral prefrontal cortex, DLPFC). Alterations in layer 3 pyramidal neurons (L3PNs), a primary source of cortico-cortical projections, may be associated with VSWM deficits in SZ. Therefore, we quantified L3PN differentially expressed genes (DEGs) and local splice variation (LSV) from each region in SZ and unaffected comparators (UC).

Methods: For each region, pools of 100 L3PNs were collected from SZ (n=39) and matched UC by laser capture microdissection and underwent RNA-sequencing. RNA-seq data were analyzed using EdgeR/voomwithQualityWeights/limma for DEGs, LeafCutter for LSV, WGCNA for co-expression, and ChEA3 for transcription factor (TF) enrichment. Analyses were FDR-corrected for multiple comparisons (α=0.05).

Results: The number of DEGs (up/down) between SZ and UC was lowest in DLPFC (338/319), intermediate in PPC (557/705), and greatest in V1 (1909/1666) L3PNs. However, ***p***-value threshold-free analyses indicated the pattern of SZ-UC DEGs was highly similar across regions. The number of distinct groups of co-expressed genes was greatest in DLPFC (n=15), intermediate in PPC (n=5), and lowest in V1 (n=1) L3PNs. Co-expressed genes were regulated by distinct TFs and were not enriched for markers of excitatory neuron subtypes. This finding suggests that regional differences in co-expression complexity are attributable to transcriptional regulation. Finally, there were few significant LSV differences between L3PNs in the DLPFC (n=1), PPC (n=3), or V1 (n=16), suggesting differences in gene-level mRNA expression were a primary driver of L3PN transcriptome alterations in SZ.

Conclusion: L3PN transcriptome alterations in SZ were similar across regions, despite differences in the number and magnitude of DEGs. More complex transcriptional regulation in DLPFC L3PNs might account for fewer transcriptome alterations in SZ.

Presenter Name/Degree(s): Haley Dubovecky, BS
Current Position: Undergraduate Student

Primary Mentor in Psychiatry: Howard Aizenstein, MD, PhD

Title: **Personality determinants of loneliness trajectories in older adults: Results from a 12-week intergenerational dialog-driven intervention**

Author(s): ***Dubovecky H, Reider M, Kuppusamy M, Aizenstein H, Stahl S, and Mizuno A***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Loneliness (subjective) and social isolation (objective) are distinct yet interrelated health risks in late life. While Big Five Personality traits are cross-sectionally associated with loneliness, their role in shaping longitudinal loneliness trajectories during isolation remains unclear. We examined how personality and perceived-objective loneliness discrepancies predict change in loneliness during a 12-week intergenerational dialogue program for reducing loneliness in older adults.

Methods: We used linear mixed-effects models to examine change in perceived loneliness (UCLA Loneliness) over 12 weeks (n = 28). First, we tested whether loneliness trajectories differed by baseline residuals from a regression of loneliness on social isolation (LSNS-6), comparing participants with positive (lonelier-than-expected) versus. negative residuals (less lonely-than-expected). Second, we ran separate models to assess each Big Five (NEO-FFI) trait as a moderator.

Results: We observed a significant week × residual group interaction ***(b = –0.34, t = –2.03***), indicating that participants with positive residuals showed steeper declines in loneliness over time. Extraversion (***b = –0.025, t = –2.16***) and Agreeableness (***b = –0.057, t = –4.19***) predicted greater reductions. Neuroticism ***(b = 0.022, t = 1.76***) and Openness (***b = –0.027, t = –1.96***) predicted marginal change in loneliness.

Conclusion: These findings suggest that perceived social gaps—not objective isolation—may drive reduced loneliness. Personality traits like Extraversion and Agreeableness may facilitate more effective engagement with loneliness interventions. A small sample limits generalizability; future work should explore mechanisms sustaining loneliness despite social ties and traits, evaluating if a difference exists between genders.

Presenter Name/Degree(s): Israel Edery, BA
Current Position: Research Specialist

Primary Mentor in Psychiatry: Peter L. Franzen

Title: **Circadian and homeostatic trends in mood and alertness across a 36-hour ultradian protocol in adolescents**

Author(s): ***Edery I, Soehner AM, Buysse DJ, Kuzemchak M, Wallace MJ, Blake RG, Hasler BP, Levenson JC, Clark DB, McClung CA, and Franzen PL***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Circadian modulation of mood has been documented across several age groups and contexts but are less well documented in early/middle adolescents. We present novel findings on the trajectory of mood and alertness among adolescents during a controlled 36-hour ultradian protocol.

Methods: Adolescents (N=55, ages 13–15.9 yrs) participated in a 36-hour ultradian protocol consisting of 2-hour cycles (80-minutes of waking followed by a 40-minute sleep opportunity). Mood and alertness were assessed every cycle using 100-point visual analog scales (higher scores: more positive mood, higher alertness). Circadian and homeostatic trends were analyzed via Cosinor mixed effect modeling.

Results: Mood fluctuated significantly in a circadian pattern (B_cos=-2.21, [95% CI=-2.85, -1.56]; B_sin=2.43 [1.77, 3.10]; $p < 0.001$), with an amplitude of 3units (95% CI: 2.6–3.9). The mean mood level (mesor) was 74.44 (95% CI: 70.9–77.7), with peaks (acrophase) occurring at 5:49 PM). Mood declined slightly over the course of the 36-hour protocol at an average rate of 0.11 points per hour. Alertness also showed significant fluctuations (B_cos=-7.23, [95% CI=-8.41, -6.06]; B_sin=8.05 [6.83, 9.27]; $p < 0.001$), with an amplitude of 10.83 units (95% CI: 9.7–12.0) and a mean level (mesor) of 56 (CI: 51.5–60.0). Peaks occurred at 5:47 PM), and alertness declined steadily at 0.27 points per hour.

Conclusion: During an ultradian protocol, mood and alertness indicated a clear circadian pattern, decreasing across the night and increasing the following day, though with some evidence of accrual of a sleep debt across the 36-hour protocol. Our finding of a ~5:48 PM peak in mood and alertness aligns with previous naturalistic studies of daily rhythms that have reported positive mood peaks in mid-afternoon/early evening. Thus, daily rhythms in mood may have an endogenous origin rather than being driven solely by sociocultural factors, such as the end of the school/workday.

Presenter Name/Degree(s): Lylah Edmunds, MS
Current Position: Project Coordinator

Primary Mentor in Psychiatry: Kate Keenan, PhD and Stephanie Stepp, PhD

Title: **How do Black moms racially socialize girls vs. boys?**

Author(s): Edmunds L[1], Forrest J[1], Silva M[1], Stepp S[1], Walton S[2], Mbayiwa K[2], Moore M[2], Keenan K[2], and Anderson L[3]
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]University of Chicago; [3]Morgan State University

Introduction: Racial socialization plays an important role in the development of African American children; particularly, socialization that occurs within the family context. In fact, research shows that familial racial socialization is associated with psychological well-being, social and academic functioning, and physical health. While there is evidence to indicate that child factors such as age impact racial socialization little is known about whether practices differ as a function of child sex.

Methods: Thirty mother-child dyads (15 boys; 15 girls, with equal distribution across age 5-7) completed the ***Racial Socialization Observational Task – Early Childhood (ROST-EC)***. Raters coded parental behaviors such as scaffolding and guidance; as well as child behaviors such as negative affect. Mothers also completed the ***Parent's Experience of Racial Socialization***—a self-report instrument measuring how parents communicate messages about race to their children.

Results: Scaffolding codes differed by sex of child (***Kendall's Tau-b***=.37, ***p***=<.05), with less scaffolding observed in dyads with boys versus girls. Moms of girls also were significantly higher in parental guidance than mom of boys (***Kendall's Tau-b***=.33, ***p***<.05). Support of child ideas and dyadic security and mutuality also differed by sex. In contrast, there were no significant sex differences in self-reported practices of racial socialization on the PERS.

Conclusion: Mothers engage in conversations that aim to achieve deeper levels of understanding about racial socialization and these conversations are different in mother-daughter dyads compared to mother-son dyads. Factors such as language development and fluency, and parental concerns about consequences of direct experiences of racism may drive out observed sex differences in racial socialization.

Presenter Name/Degree(s): Hollis Edvardsson, MPH, CCRC
Current Position: Research Coordinator

Primary Mentor in Psychiatry: Sarah L. Pedersen, PhD

Title: **Differences in cannabis use motivations among sexual and gender diverse individuals compared to heterosexual and cisgender individuals and their associations with self-reported cannabis use and problems**

Author(s): *Edvardsson HM, Prakash K, Kennedy TM, Ross K, Björn B, Pedersen SL, Molina BSG, and Horton LE*
Affiliation(s): *Department of Psychiatry, University of Pittsburgh School of Medicine*

Introduction: Inequities in cannabis use and related health outcomes exist when comparing sexual and gender diverse (SGD) individuals to their cisgender/heterosexual (CisHet) peers. Given the central role of cannabis use motives in individuals' cannabis use and problems, we compared motives for cannabis use between SGD and CisHet young adults and their associations with cannabis use and problems.

Methods: Participants included 148 young adults who used cannabis at least weekly (M_{age}=21.75, *SD*=2.03; 42.6% self-identified as Black/African American, 57.4% as White/European American; 64.2% assigned female at birth; 50% identified as SGD, 27.0% of whom self-identified transgender and gender diverse). Participants completed a baseline survey as part of an ongoing study examining effects, thoughts, experiences, and inequities of cannabis use. We assessed cannabis use motives using the Comprehensive Marijuana Motives Questionnaire, cannabis use problems using the Marijuana Problem Index, and past 30-day and 12-month cannabis use.

Results: Independent samples *t*-test results showed that participants who identified as SGD endorsed using cannabis for "enjoyment" more frequently compared to their CisHet counterparts (p<0.001, t=3.20(134)). Conversely, SGD participants endorsed "conformity" and "alcohol co-use" (e.g., "because you were under the influence of alcohol") as motives with a lower frequency (p=0.016, t=-2.17(134) and p <0.001, t=-3.26(134) respectively). Linear regression results indicated no significant associations between either SGD identity or cannabis motives and cannabis use frequency (past 30-days or past 12-month) and problems, adjusting for age, assigned sex at birth, and race.

Conclusion: Differential motives in using cannabis between SGD and CisHet young adults did not appear to result in greater cannabis use or problems in this study. While we did not have the power to look at subgroups within SGD, future research should look for differences between these subgroups. Further research is needed to understand what mechanisms result in inequities in cannabis use and problems.

Presenter Name/Degree(s): Hatice Nur Eken, MD
Current Position: Resident Psychiatrist

Primary Mentor in Psychiatry: Rebecca Price, PhD

Title: **Associations between obsessive-compulsive personality disorder traits and symptom dimensions in obsessive-compulsive disorder: A cross-sectional study**

Author(s): ***Eken HN and Price R***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Obsessive-compulsive disorder (OCD) is a heterogeneous disease where patients present with a wide variety of different symptoms. OCD symptom dimensions have predominantly yielded a four-factor structure including cleaning/contamination, symmetry, doubts about harm and unacceptable/forbidden thoughts (Bloch et al., 2015; Moreno-Amador et al., 2023). OCPD is a personality disorder characterized by a lifelong pattern of excessive attention to detail, rigidity and perfectionism. While OCPD and OCD are distinct disorders, they co-occur and share certain traits, such as rigidity. It has also been shown that individuals who have comorbid OCD and OCPD have significantly more functional impairment (Garyfallos et al., 2010), but evidence on the link and divergence between these disorders is extremely limited. In this study, we aimed to investigate the relationship between OCPD and different symptom dimensions of OCD and related disorders.

Methods: We conducted a review of available literature on perinatal OCD. Google Scholar and PubMed were searched by authors. The following searches were performed: “OCD” and “consultation-liaison psychiatry”; “perinatal OCD”, “obsessive compulsive disorder.” We analyzed data from 229 individuals enrolled in a clinical trial on compulsive behaviors in our institution. We used the Pathological Obsessive Compulsive Personality Scale (POPS) to assess maladaptive OCPD traits (Sadri et al., 2019). We grouped individuals in two categories “low” or “high” based on calculated Z scores in different subscales of OCI-R (Obsessive Compulsive Inventory-Revised) scale (Abramowitz & Deacon, 2006), Threat-Related Reassurance Seeking Scale (Cougle et al. 2012), Massachusetts General Hospital Hair Pulling Scale (Keuthen et al., 1995), and Skin Picking Scale (Keuthen et al. 2001). We used linear regression to investigate relationships between groups, and t-tests to compare mean scores between the low and high categories.

Results: Overall, higher scores on the OCPD (POPS) scale were significantly and positively related to higher OCD (OCI-R) scores ($F(1,197)=43.996$, $p<.001$). Mean POPS scores were significantly elevated in those who have symptoms in washing ($p<0.001$, Cohen’s $d=-.53$), ordering ($p<0.001$, Cohen’s $d=-.54$), hoarding ($p=0.004$, Cohen’s $d=-.57$), neutralizing ($p= .001$, $d=-.51$) and checking ($p=0.009$, $d=-.38$) dimensions, but not in the obsessing ($p=.155$) dimension of the OCI scale. POPS score was also elevated in those who have an increased tendency to seek reassurance ($p<0.001$), but not in those who engage in hair pulling ($p=.636$) or skin picking ($p=0.061$).

Conclusion: Here in a sample of 229 individuals reporting compulsive behaviors, we demonstrate that there are significant positive associations between various obsessive compulsive and related disorders (OCRD) symptom domains and OCPD traits, suggesting that these disease concepts may not be as distinct as their current conceptualization. At the same time, we identified specific OCRD domains that did not co-vary with OCPD. Future studies should explore potential mechanistic mediators for this link and heterogeneity. This study is one of the first to highlight the link between OCPD traits and different symptom dimensions of OCD, highlighting that there is a significant positive association between OCPD scores and overall OCD symptom severity, as well as with different specific subtypes of compulsive behaviors. Understanding the phenomenological relationship between these disorders can inform the development of novel, targeted treatments for OCRD and OCPD.

Presenter Name/Degree(s): Safaa Eldeeb, PhD
Current Position: Postdoctoral Scholar

Primary Mentor in Psychiatry: Carla Mazefsky, PhD

Title: **Variability in suicidal thoughts and behavior among autistic adolescents and adults: Subgroup identification, predictive features, and group differences**

Author(s): ***Eldeeb S, Conner C, Antezana L, Beck K, and Mazefsky C***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Autistic people experience high rates of suicidal thoughts and behaviors (STB) and are three times more likely to die by suicide than non-autistic people. However, understanding who most at risk is limited. Identifying subgroups with varying STB levels could improve prevention efforts. This study aimed to use machine learning clustering to identify and characterize STB subgroups.

Methods: The sample included 831 autistic people who participated in an online study (age 37±12; range 14-86). We applied a random forests clustering to item-level data from the Autism Suicidality Inventory, Columbia-Suicide Severity Rating Scale, and Suicidal Thoughts and Behaviors Questionnaire. Feature importance analyses identified key items for subgroup membership, while demographics, health, and mental health variables were examined for subgroup differences.

Results: Our results identified five distinct groups of STB risk based on suicidal thought severity and past attempts. Groups range from lowest risk (G1, n=97: no current suicidal thoughts, 6.2% lifetime attempts) to highest risk (G5, n=128: 97% current thoughts, 62.5% past attempts). G2 (n=101) and G3 (n=166) differ in STB, while G3 and G4 (n=339) share past similarities but differ in current STB. G5 had the highest percentage of females (41% vs. 19% in G1), and exhibited the most psychiatric diagnoses, food and housing insecurity, physical pain, masking and low self-compassion. The STUQ item on suicidal thought frequency was the strongest factor influencing STB clustering, followed by ASI items on disclosure concerns, social challenges, and emotional masking.

Conclusion: This study highlights new areas to consider, such as food and housing insecurity, physical pain, and self-compassion, in addition to known correlates of STB in autism (e.g. masking and ED). Key factors influencing STB subgroups highlight areas for prevention and intervention, such as overcoming disclosure barriers, enhancing social support, and alternative ED strategies. Future work should examine short-term STB increases related to these risk factors.

Presenter Name/Degree(s): Jaime Ellis, BS
Current Position: Research Specialist

Primary Mentor in Psychiatry: Stephanie Stepp, PhD

Title: Borderline personality features and suicidality in children: Examining associations in a high-risk sample

Author(s): ***Ellis J[1], Glinsky M[1], Torres A[1], Vanwoerden S[1], Byrd A[1,2], and Stepp S[1,2]***
Affiliation(s): ***[1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Psychology, University of Pittsburgh***

Introduction: Borderline personality disorder (BPD) can first be diagnosed in adolescence, which is also a time of heightened risk for suicidal thoughts and behaviors (STBs). Although prior work has shown that certain BPD features can be detected in childhood, these studies have not yet examined whether STBs may also be an early risk factor. Given that STBs during childhood is a growing concern, with estimates that 11-19% of children experience suicidal ideation and 1.6-2.4% attempt suicide, research is needed to better understand these associations. The current study will examine the early emergence and co-occurrence of BPD symptoms and STBs during late childhood.

Methods: Data are currently being collected from a diverse sample of high-risk children aged 9-11 years whose mothers have a history of suicide behavior and current emotion dysregulation (***n***=67; $\boldsymbol{M}_{age}$=10.45 years, ***SD***=0.88; 48% female). The Borderline Personality Features Scale for Children was administered to mothers and children separately to assess the presence of early emerging BPD symptoms. The Columbia-Suicide Severity Rating Scale was administered to children to evaluate STBs in the past month and lifetime.

Results: Preliminary analyses found associations between child-reported BPD symptoms and suicide ideation in the past month (***r*****=** 0.25, ***p*****=** 0.038) and in their lifetime (***r*****=** 0.28, ***p*****=** 0.021). Parent-reported BPD symptoms were also associated with child suicide ideation, though only for lifetime (***r*****=** 0.35, ***p*****=** 0.004). BPD symptoms were not associated with suicidal behaviors.

Conclusion: The current findings indicate that children with early emerging BPD symptoms may also be at risk for suicide ideation. Longitudinal research is needed to evaluate whether suicide ideation is a precursor of early, emerging BPD features during late childhood.

Presenter Name/Degree(s): Gabrielle English, BS
Current Position: Research Project Coordinator

Primary Mentor in Psychiatry: Mary Phillips, MD, MD (CANTAB)

Title: Social rejection enhances frontal pole activity in adolescents with musculoskeletal pain: A potential link between the neural responses to social threat and physical pain

Author(s): *English G[1], Nooraeen S[1], Bertocci MA[1], Taylor M[1], Stiffler R[1], Wagle S[1], Caputo S[1], Roberts L[1], Anonick R[2], Vogt K[1], McClincy M[1], Oppenheimer C[2*], and Phillips ML[1] **

Affiliation(s): *[1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]RTI International*

*** Drs. Phillips and Oppenheimer are co-PIs for this project*

Introduction: Adolescence is a critical developmental period in mental health. Acts of suicide are the second leading cause of death within this age group. Suicide is often theorized as an attempt to escape unbearable psychological and physical pain. Social like physical threat, activates pain-related brain networks. However, the link between neural response to social threat, defined as threat to social status or relationships, and physical pain is not known. In adolescents with varying musculoskeletal-pain, preliminary analyses of adolescent brain activity examine the relationships between pain, brain activation, affective pain, and physical pain during a social rejection task.

Methods: Adolescents (n=19; M_{age}= 16.61; 68.42% female), recruited from the local Pittsburgh community who reported musculoskeletal pain ($M_{affective\ pain}$ = 4.16; $M_{pain\ intensity}$ = 4.58) completed an MRI scan while participating in the Peer Expressed Emotion task (PEER-EE) that simulates experiences of rejection from a close friend. Analysis was conducted using SPM12, with a regression model examining affective pain in response to Criticism (vs. Praise), adjusting for sex and age. SPM threshold p=0.005.

Results: Whole brain MRI analysis revealed a positive correlation between affective pain scores and increased activation in the left frontal pole (BA 10) (k=20, t=8.61, p<.005) during social rejection. Right and left BA10 (right:k=20, t=5.31, p<.005, left:k=20, t=3.98, p<.005) trended towards a positive relationship with intensity scores.

Conclusion: These findings suggest that social rejection may be linked to neural responses in adolescents within the frontal pole region involved in self-other relational reasoning when experiencing musculoskeletal pain. The activation of the frontal pole may indicate a key neural correlate in the intersection of social threat and pain. Thus, interventions targeting social threat, such as peer rejection, may help mitigate affective pain and reduce perceived threat of social rejection.

Presenter Name/Degree(s): John F. Enwright, III Ph.D.
Current Position: Research Assistant Professor

Title: **Transcriptional profiles of somatostatin and parvalbumin interneuron subtypes in the human dorsolateral prefrontal cortex: Implications for schizophrenia**

Author(s): *Enwright JF III[1], Tamburino AM[2], Tumkaya T[3], Lovatt D[4], Pan J[4], Gunaratna R[4], Fagegaltier D[3], Wang X[4], Marino MJ[4], Fish K[1], Arion D[1], Gonzalez-Burgos G[1], and Lewis DA[1]*
Affiliation(s): *[1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Spatial and Single Cell Multiomics Data, AI & Genome Sciences, Merck &Co, Inc.; [3]Early Discovery Genetics, Merck &Co, Inc.; [4]Neuroscience, Merck &Co, Inc.*

Introduction: Alterations in the somatostatin (SST) and parvalbumin (PVALB) classes of inhibitory GABAergic interneurons in the dorsolateral prefrontal cortex (DLPFC) are thought to contribute to the core cognitive impairments of schizophrenia. Both classes of interneurons are composed of multiple subtypes, but the distinguishing features and relative proportions of these subtypes have not been determined in the DLPFC from young and middle-aged adults.

Methods: We used single nuclear RNA sequencing to determine the transcriptional profiles of interneuron subtypes in DLPFC tissue samples from discovery and validation cohorts of neurotypical individuals; fluorescent in situ hybridization to validate selected findings; and comparisons of transcriptome and electrophysiology data from the Allen Brain Atlas to infer functional properties of selected subtypes.

Results: The relative proportions of 5 major interneuron classes and of 37 interneuron subtypes were highly consistent across 19 neurotypical individuals. We identified 11 SST subtypes, including 2 rare subtypes that likely produce dopamine or regulate blood flow, and a subtype derived from the caudal ganglionic eminence. Among the 9 PVALB subtypes identified, chandelier cells were transcriptionally distinct from PVALB basket cells (PVBCs) and PVBC subtypes, distinguished by high versus low PVALB expression, had distinctive molecular and electrophysiological characteristics.

Conclusion: The robust identification of discrete subtypes of SST and PVALB interneurons in the DLPFC from young and middle-aged adults provides the means to determine which specific subtypes are altered in proportional representation and/or gene expression in schizophrenia.

Presenter Name/Degree(s): Yadira Estrada, BS, MSW
Current Position: Research Assistant

Primary Mentor in Psychiatry: Karen Jakubowski, PhD

Title: **Two fronts of trauma: The differential effects of intimate partner violence and community violence on PTSD in rural Mexico**

Author(s): Estrada Y[1], Leonard AF[1], Jakubowski K[2], and Medrano AS[2]
Affiliation(s): [1]Department of Psychology, University of Pittsburgh; [2]Department of Psychiatry, University of Pittsburgh School of Medicine

Introduction: Intimate partner violence (IPV) and community violence exposure (CVE) are prevalent worldwide and linked to adverse psychological outcomes (Aguerrebere et al., 2021; Mora et al., 2021). Despite high rates of violence, scarce research has examined IPV and CVE simultaneously in Mexico, with no prior study in rural Mexico. The current study aimed to (1) identify rates of IPV and CVE among rural Mexican men and women, (2) examine how IPV and CVE independently and simultaneously contribute to PTSD symptoms, and (3) determine to what degree gender shapes how men and women experience PTSD symptoms after violence.

Methods: 200 adults (M_{age} = 40.7, SD = 9.29) from a rural community in Mexico, including 79.5% women and 20.5% men, self-reported IPV history (Partner Violence Scale), CVE (Survey of Exposure to Community Violence) and PTSD symptoms (PCL-C). Four hierarchical regressions were conducted to examine links between CVE and IPV, respectively, and PTSD symptoms, as well as the moderating role of gender in associations between violence and PTSD symptoms.

Results: Women, compared to men, reported significantly higher rates of psychological and sexual IPV, with similar rates of physical IPV. There was no gender difference in rates of CVE. Regression analyses indicated that psychological IPV was the strongest predictor of PTSD symptoms (b = .05, p < .001), followed by witnessing violence (b = .01, p < .05). Gender was found to moderate the association between psychological IPV and PTSD, with men reporting significantly greater PTSD symptoms at high levels of psychological IPV. No significant interaction emerged for CVE and PTSD symptoms. There was also no significant main effect found between poly-victimization (exposure to both IPV and CVE) and PTSD symptoms.

Conclusion: Findings highlight the mental health consequences of psychological IPV and suggest a need for gender-sensitive interventions addressing IPV in rural Mexico.

Presenter Name/Degree(s): Carly Fabian, BS
Current Position: Graduate Student Researcher; PhD Candidate

Primary Mentor in Psychiatry: Max Joffe, PhD

Title: **Mechanisms driving binge drinking: alcohol-induced alterations in PFC basket cell function and $mGlu_5$ receptor signaling**

Author(s): Fabian C[1,2,3], Jordan N[1,2], and Joffe M[1,2,3]
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Translational Neuroscience Program, University of Pittsburgh; [3]Center for Neuroscience at the University of Pittsburgh

Introduction: The prefrontal cortex (PFC) critically regulates cognitive functions such as decision-making, motivation, and impulsivity, which are disrupted in binge drinking. Proper PFC function depends on a delicate balance between excitation and inhibition, maintained by GABAergic interneurons. Among these, cholecystokinin (CCK)- and parvalbumin (PV)-expressing basket cells comprise two distinct classes of perisomatic-targeting interneurons that exert powerful control over cortical excitability. Our lab has shown sex-dependent adaptations to PV-INs following chronic drinking and identified a sex-specific role for the metabotropic glutamate receptor 5 ($mGlu_5$) in regulating voluntary consumption. However, due to broad CCK expression in both inhibitory and excitatory cells, along with limited selective genetic tools, much less is known about the impact of binge drinking on CCK-INs.

Methods: To address this, we used mice expressing Cre recombinase under a CCKxVGAT promoter, to selectively target inhibitory cells expressing CCK. Here we examined the impact of binge drinking, modeled by the Drinking in the Dark (DID) paradigm, on the function of PFC CCK- and PV-INs. After 4 weeks of binge drinking, we performed whole-cell electrophysiology from fluorescently labeled PV-INs and CCK-INs in the prelimbic PFC of sex-matched water control and DID mice.

Results: We found that binge drinking induces adaptations in membrane properties, synaptic strength, and $mGlu_5$ receptor signaling at PFC PV- and CCK-INs. In PV-INs, DID led to changes in the amplitude of spontaneous excitatory postsynaptic currents (sEPSCs) and $mGlu_5$-mediated inward currents in both male and female mice. By contrast, DID altered sEPSC frequency in CCK-INs and $mGlu_5$-mediated inward currents regardless of sex. Lastly, we employed ***in vivo*** fiber photometry during operant drinking to assess the role of PFC basket cells in alcohol reinforcement.

Conclusion: Together, these studies reveal how binge drinking differentially affects PFC CCK- and PV-INs function and underscore the potential involvement of PFC basket cells in drinking behaviors.

Presenter Name/Degree(s): Marissa Farinas, MS
Current Position: Senior Research Associate

Primary Mentor in Psychiatry: Thomas Karikari, PhD

Title: **Plasma vs. serum: Which is better for proteomic blood biomarker analysis? Evaluation of the novel NULISA platform**

Author(s): ***Farinas MF[1], Chen Y[2], Zeng X[1], Nafash MN[1], Gogola A[3], Kofler J[4], Tudorascu DL[1], Shaaban CE[5,6], Lingler JH[6,7], Pascoal TA[1], Klunk WE[1], Villemagne VL[1], Berman SB[8], Sweet RA[1,8], Kamboh MI[9], Ikonomovic MD[1,8], Snitz BE[8], Cohen AD[1], Lopez OI[8], and Karikari TK[1]***

Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Chemistry, University of Pittsburgh; [3]Department of Radiology, School of Medicine, University of Pittsburgh; [4]Department of Pathology, School of Medicine, University of Pittsburgh; [5]Department of Epidemiology, School of Public Health, University of Pittsburgh; [6]Alzheimer's Disease Research Center, University of Pittsburgh; [7]Health and Community Systems, University of Pittsburgh School of Nursing; [8]Department of Neurology, School of Medicine, University of Pittsburgh; [9]Department of Human Genetics, School of Public Health, University of Pittsburgh

Introduction: Blood biomarker studies most often use plasma samples, but the suitability of serum as an alternative sample type remains unclear. We compared the technical performance of the novel NUcleic acid-Linked Immuno-Sandwich Assay (NULISA), a blood-based targeted proteomic biomarker assay, in plasma and serum samples processed from identical blood draws in a memory clinical cohort.

Methods: Paired plasma and serum samples (N = 43) from the University of Pittsburgh ADRC were analyzed using the NULISAseq CNS disease panel 120 (v2) on an Alamar ARGOTM system. Protein levels were quantified by next generation sequencing, normalized, scaled, and log 2-transformed to NULISA Protein Quantification (NPQ) units. Spearman's rank correlation assessed concordance between plasma and serum NPQs, while the Wilcoxon rank-sum test evaluated differences in protein levels. Receiver operating characteristic (ROC) curves and the area under the curve (AUC) were used to determine diagnostic accuracy.

Results: The assay achieved high analyte detectability (95.7% ± 14.2%) with low variability (%CV: 4.9%). Strong correlations (Spearman rho: 0.75-0.96) were observed for Aβ42, p-Tau217, p-Tau231, p-Tau181, GFAP, and NEFL across both matrices. 29 targets were upregulated in plasma (log2FC vs. serum >0.5), while 16 were upregulated in serum (log2FC vs. plasma >0.5). Notably, Aβ42, p-tau181, p-tau217, and p-tau231 levels were significantly higher in plasma than in serum ($p<0.001$). Contrarily, GFAP and NEFL levels were similar in plasma and serum ($p>0.05$). 10 proteins in plasma and 8 proteins in serum displayed good diagnostic accuracy, with plasma p-Tau231, plasma MAPT, plasma p-Tau217, and plasma p-Tau181 showing AUCs>0.9 ($p<0.001$).

Conclusion: NULISAseq-based AD blood biomarkers in paired plasma and serum are highly correlated; however, the absolute levels significantly vary by matrix type. These findings highlight the importance of considering specimen type in clinical study designs to ensure the reliability and accuracy of AD biomarker diagnostics.

Presenter Name/Degree(s): Julia S. Feldman, PhD
Current Position: Postdoctoral Scholar

Primary Mentor in Psychiatry: Heather Joseph, DO

Title: **The relation between paternal emotion regulation and inconsistent parenting is dependent on maternal emotion regulation**

Author(s): ***Feldman JS[1], Lorenzo NE[2], Wilson M[1], Morgan J[1], Molina BSG[1], and Joseph HM[1]***
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Psychology, College of Arts & Sciences, American University

Introduction: Emotion regulation (ER) is important for navigating daily life, including parenting. Most research on ER and parenting has focused on mothers. Although it is likely that similar relations exist for fathers, this remains untested. The link between paternal ER and parenting is likely moderated by contextual factors, such as co-parent ER. This study tested longitudinal relations between paternal ER and parenting. Paternal ER was expected to be associated with lower levels of inconsistent parenting and higher levels of supportive parenting at 1-year follow-up. Maternal ER was hypothesized to moderate these relations, such that the negative impact of low paternal ER would be buffered in families with mothers who have high ER.

Methods: Participants come from the Pittsburgh Attention-Deficit/Hyperactivity Disorder (ADHD) Risk in Infancy Study (***N*** = 73 families). Parents rated their ER skills (child age 4) and fathers rated their supportive and inconsistent parenting practices (child age 5). Paternal ADHD symptoms were assessed via clinical interview (baseline in infancy) and controlled for in analyses.

Results: Path analysis revealed that paternal ER was not directly related to parenting one year later. Maternal ER moderated relations between paternal ER and inconsistent parenting (interaction ***B*** = 0.03, ***SE*** = 0.01, ***p*** < .05), but not supportive parenting (interaction ***B*** = -0.02, ***SE*** = 0.01, ***p*** > .24). Simple slopes were not significant across levels of maternal ER. However, the presence of a significant interaction term implies that the relation between paternal ER and inconsistent parenting varied by level of maternal ER.

Conclusion: Implications include the importance of considering the role of paternal ER in challenges in parenting and including mothers ***and*** fathers in studies of paternal caregiving.

Presenter Name/Degree(s): Cynthia Felix, MD, MPH
Current Position: Postdoctoral Associate

Primary Mentor in Psychiatry: Tharick Pascoal, MD, PhD

Title: **Usefulness of MoCA in detecting preclinical AD**

Author(s): *Felix C[1], Snitz BE[2], Kollasserry FJ[3], Rebok GW[4], Ferreira PCL[1], Tudorascu DL[1]; Povala G[1], Saha P[1], Amaral L[1], Rodrigues MS[1], Oliveira Jr. M[1], Lussier FZ[1], Masdeu J[5], Soleimani-Meigooni D[6], Fortea J[7], Lowe V[8], Oh H[9], Pascual B[5], Gordon BA[10], Rosa-Neto P[11], Baker S[12], Nasreddine Z[13], Pascoal TA[1,2]*

Affiliation(s): *[1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Neurology, University of Pittsburgh; [3]University of Pittsburgh; [4]Johns Hopkins School of Public Health; [5]Houston Methodist Research Institute, Department of Neurology; [6]Memory and Aging Center, University of California San Francisco; [7]Sant Pau Memory Unit, Department of Neurology, Hospital de la Santa Creu i Sant Pau; [8]Department of Radiology, Mayo Clinic; [9]Department of Psychiatry and Human Behavior, Brown University; [10]Department of Radiology, Washington University in St. Louis; [11]Translational Neuroimaging Laboratory, McGill University Research Centre for Studies in Aging, Douglas Research Institute [12]Lawrence Berkeley National Laboratory; [13]MoCA Cognition*

Introduction: Preclinical AD is difficult to detect using traditional clinical tools. Yet, it can exhibit a heterogeneous pattern of subtle clinical abnormalities. Identifying common clinical tools to detect preclinical AD can help tackle the disease early. We aim to evaluate the link between Montreal Cognitive Assessment (MoCA) scores and AD pathology in cognitively unimpaired (CU) individuals.

Methods: We studied 204 CU older adults who underwent both ^{18}F-FTP and ^{18}F-MK6240 scans, who were stratified based on their amyloid PET visual reading, into CU Aβ+ (n = 35) and CU Aβ- (n = 137), from the HEAD study. CU adults had Clinical Dementia Rating (CDR) of 0 and were clinically identified as non-MCI and non-demented.

Results: Voxel-wise linear regression models tested the association between MoCA and tau pathology. CU A+T+ individuals had significantly lower total MoCA scores than A-T- individuals Lesser MoCA total score was significantly associated with greater mediobasal temporal tau deposition, using ^{18}F-MK6240 and ^{18}F-FTP. These results were driven by the CU A+ group and absent in the CU A-.

Conclusion: Thus, the MoCA, a simple routine in-office clinical test, can detect subtle cognitive dysfunction associated with preclinical AD. This is an option for prescreening community-dwelling older adults in primary care settings, the initial interface for most older adults without cognitive complaints.

Presenter Name/Degree(s): Meghan Fiske, BS
Current Position: Research Specialist

Primary Mentor in Psychiatry: Mary Phillips, MD

Title: **Context processing and the implications for mania risk**

Author(s): ***Fiske M, DiDomenico D, Afriyie-Agyemang Y, Chase H, Bertocci M, Stiffler R, Graur S, and Phillips M***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Context processing, the ability to control behavior with maintenance of prior context, has been routinely researched in relation to schizophrenia. However, research examining cognitive control deficits in bipolar disorder is limited. We examined relationships between lifetime mania and depression risk and context processing using the AX-Continuous Performance Task (AXCPT), a well-validated computerized test used to measure context processing and cognitive control mechanisms. The errors participants incur indicate deficits with executive processing; "AY" errors reflect an inability to inhibit a prepotent response, while "BX" errors reflect an inability to maintain attenuated activity. We explored whether those with a greater lifetime depression and mania risk made certain error types more frequently.

Methods: As a part of a larger study (CR18110024-046) at the University of Pittsburgh, 148 young adults at risk for bipolar disorder (mean age=23.64, SD=3.43; 94 female) completed the Moods Spectrum Self-Report-Lifetime Version (MOOD_SR_L) manic and depressive domains and the AXCPT. Error transformed variables were calculated in SPSS to assess task performance, focusing on frequently occurring errors; recency errors (d'context), priming errors (A-cue), and the Proactive Behavioral Index (PBI). Regression models included predictors of age, IQ, gender and the MOODS lifetime mania and depression risk.

Results: Participants with a greater lifetime mania risk had an increased tendency to make priming errors (β=.012, ***p***<.001), and recency errors (β=.171, ***p***<.001) on the AXCPT. Furthermore, as lifetime mania risk increased, the use of proactive control decreased (β= -.009, ***p***<.016). Post-hoc analysis showed lifetime depression risk was significantly associated with priming errors (β=.011, ***p***<.001), recency errors (β=.015, ***p***<.001), and the use of proactive control (β=-.009, ***p***=.005).

Conclusion: Participants with an increased lifetime mania risk made more errors on the AXCPT across all error types. Furthermore, contrary to current literature on the AXCPT, lifetime risk of depression also predicted poor performance on the task.

Presenter Name/Degree(s): Camryn Forbes, BS
Current Position: Research Specialist

Primary Mentor in Psychiatry: Mary Torregrossa, PhD

Title: **VGLUT2 knockdown in the VTA reduces acquisition of alcohol self-administration in a sex-specific manner**

Author(s): ***Forbes C, Steinfeld M, Zeak J, and Torregrossa M***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Alcohol use disorders (AUDs) are increasingly prevalent and have few effective treatment options. Both dopamine and glutamate increases in response to alcohol are implicated in AUD, so here we focused on a subpopulation of neurons in the VTA that transmit both glutamate and dopamine. These neurons express vesicular glutamate transporter 2 (VGLUT2), which is responsible for transporting glutamate and plays a role in the acidification of synaptic vesicles. Therefore, we hypothesize that sex differences in VGLUT2 expression may be responsible for the increased rewarding effects of alcohol seen in females through the transmission and release of both glutamate and dopamine.

Methods: Starting at postnatal day 28, TH-Cre rats on a Long Evans background receive bilateral virus infusions using a Cre-dependent VGLUT2-shRNA virus or control virus into the VTA. After 4 weeks of virus expression, rats are food restricted and trained to self-administer alcohol on a fixed ratio 1 program. Following extinction, rats undergo a cue-induced reinstatement session to look at relapse-like behavior and a progressive ratio session to determine their alcohol-seeking behavior under increased pressure. A subset of rats underwent sucrose pellet self-administration following the progressive ratio testing to determine if any effects seen were specific to alcohol.

Results: Females in the active virus group consume significantly ($p<0.05$) less alcohol than control females during days 3-7 of alcohol self-administration with their consumption reaching similar levels as control females around day 10 of self-administration. While not significant, there is a trending difference in consumption between male control and active groups across training. There are currently no effects seen in either sex during cue-induced reinstatement or progressive ratio testing. Animals who underwent sucrose pellet self-administration acquired the behavior similarly in both groups.

Conclusion: Knockdown of VGLUT2 in the VTA reduces acquisition of alcohol self-administration in females and males, albeit differently, indicating that reducing VGLUT2 may impact the rewarding effects of ethanol in both sexes through different mechanisms.

Presenter Name/Degree(s): Lauren Fowler, BS
Current Position: Research Specialist

Primary Mentor in Psychiatry: Brian A. Coffman, PhD

Title: **Auditory and motor timing dysfunction in first episode psychosis indexed by rhythmic finger tapping**

Author(s): Fowler L, Seebold D, Rhorer H, Kavanagh J, Salisbury DF, and Coffman BA
Affiliation(s): Clinical Neurophysiology Research Laboratory, Western Psychiatric Institute and Clinic, Department of Psychiatry, University of Pittsburgh School of Medicine

Introduction: Schizophrenia (SZ) is associated with impairments in neural timing, which is linked to dysfunction in the cerebello-thalamo-cortical (CTC) and cortio-striatal-thalamo-cortical (CTSC) brain circuits, impacting perceptual and motor-based timing processes. Individuals diagnosed with schizophrenia show an increase in the speed of their "internal clock" along with abnormal patterns in temporal learning and an increase in motor-timing variability. Here, we examined if first-episode psychosis (FEP) individuals show similar impairments in neural timing and motor-timing variability to identify if the system-level dysfunction is apparent early in the course of the disorder.

Methods: We measured motor timing performance during EEG/MEG in 11 FEPs and 15 healthy comparison individuals via a 2Hz rhythmic finger tapping (RFT) task, where participants first listened to, then tapped along with an auditory tone (1000 Hz, 50ms duration). After ten repetitions, the tone was discontinued, and participants continued tapping at the same rate for 30 repetitions. Performance was measured by the interval timing of button-presses relative to the 500 ms target rate. Self-paced tapping variability was defined by the standard deviation of the mean inter-tap intervals (ITI) across ten trials (300 repetitions). Additionally, the Wing-Kristofferson model was applied to divide response timing variance into clock and motor implementation variance subcomponents.

Results: Tap rate variability was greater for FEP than healthy controls; Dividing variability into clock and motor variance components revealed that group differences were driven by significantly greater clock variance in FEP ($\boldsymbol{p} = 0.05$), with no difference in motor implementation variance ($\boldsymbol{p} > 0.5$).

Conclusion: Preliminary results indicate that FEPs display greater self-paced finger-tapping rate variability due to dysfunction in internal clock timing. Greater tapping variability suggests reduced temporal precision of CTC/CTSC in FEPs. These initial findings replicate prior research in schizophrenia and provide further evidence of neural timing impairments in FEPs at the level of internal clock timing and suggest dysfunction.

Presenter Name/Degree(s): Hannah D. Gallagher, BS
Current Position: Research Associate

Primary Mentor in Psychiatry: Oliver Lindhiem, PhD

Title: **Small effects, large impact: An illustration from national mental health data**

Author(s): ***Gallagher HD, Tomlinson CS, Vaughn-Coaxum RA, Kolko DJ, Yu L, and Lindhiem O***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Even small changes in mean scores can have large population-level impacts, as recently argued by Carey et al., 2023. In this study, we provide an empirical test of this using national data on paternal anxiety and depression symptoms from before and after the COVID-19 pandemic. We hypothesized that seemingly small effect sizes in mean score changes can translate to substantial increases in clinical symptom prevalence, demonstrating the need for contextualized interpretations.

Methods: Data were drawn from fathers from two nationally representative surveys conducted in 2018 (***N***=570) and 2024 (***N***=425). In both surveys, fathers completed the Generalized Anxiety Disorder-7 Assessment (GAD-7) and the Patient Health Questionnaire-8 (PHQ-8). T-tests and effect sizes were calculated to assess changes in mean anxiety and depression scores. The proportions of fathers reporting scores above the clinical cutoff on both the anxiety and depression measures were reported for each of the samples.

Results: Mean anxiety scores increased from 2.97 in 2018 to 3.98 in 2024 (***p***<.001), with a small effect size (***d***=0.22). Mean depression scores also increased from 3.52 to 4.55 (***p***<.01) with a small effect size (***d***=0.20). Despite these small effect sizes, the percentage of fathers above each measure's clinical threshold increased by 45.10% (from 10.2% to 14.8%) for anxiety and 55.36% (from 11.2% to 17.4%) for depression from 2018 to 2024.

Conclusion: These findings support the importance of considering the most appropriate measure of population-level effects. Even a slight change in mean scores at the population level can result in a much larger proportion of individuals exceeding clinical thresholds. Future research can continue to explore the best metrics of magnitude of findings within psychological research.

Presenter Name/Degree(s): Isaac S Gamwo, BS, BPhil
Current Position: Undergraduate Researcher

Primary Mentor in Psychiatry: Traci Kennedy, PhD

Title: **Is age really just a number? Effect of age on responsiveness of young adults with ADHD to a smartphone intervention**

Author(s): ***Gamwo I, Williard A, and Kennedy TM***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Attention-Deficit/Hyperactivity Disorder (ADHD) is a prevalent neurodevelopmental condition that causes significant impairments in the transition to young adulthood, necessitating tailored treatments for emerging adults (ages 18-21). While ADHD symptom expression and treatment responsiveness can evolve with age, no studies have examined whether younger emerging adults benefit more from interventions than their slightly older peers. This study examined the preliminary effectiveness of the START Smart mobile health (mHealth) intervention for ADHD in emerging adults and whether participant age predicted symptom improvement.

Methods: Seventy-two emerging adults (ages 18–21) with ADHD (75% female sex assigned at birth) completed the 3-week START Smart mHealth intervention, which delivered daily smartphone-based symptom tracking and feedback. Participants were randomized to receive low-dose (once daily) or high-dose (five times daily) feedback. ADHD symptom severity was assessed at baseline and follow-up via participant self-report and collateral informant ratings using the ADHD Rating Scale. Hierarchical regression analyses examined whether age predicted symptom reduction, while adjusting for dose group, medication status, and sociodemographic covariates.

Results: Significant reductions in ADHD symptoms from baseline to follow-up were observed across reporters (Participant: $t(68)=4.789$, $p<0.001$ Collateral: $t(67)=3.950$, $p<0.001$). The high-dose condition was associated with greater symptom reduction according to collateral informants ($b=4.01$, $p=0.049$), but not self-report. Age was not a significant predictor of symptom improvement, and no age-by=dose interaction was found. Younger participants completed more prompts ($r=-0.29$, $p=0.014$), indicating higher engagement, but this did not translate into great symptom improvement.

Conclusion: The START Smar intervention seemed to effectively reduce ADHD symptom severity in emerging adults without ADHD, regardless of age. While younger participants showed higher engagement, treatment responsiveness did not vary by age suggesting that age may not critically determine mHealth intervention outcomes during this developmental window. Future research should examine intervention effects across a broader age range.

Presenter Name/Degree(s): Ashley Gelber, BS
Current Position: Research Specialist

Primary Mentor in Psychiatry: Jessie Northrup, PhD

Title: **Stress and perceived support in parents of children with and without autism spectrum disorder**

Author(s): ***Gelber A, Saunders A, Christenson K, Manno M, Startari S, and Northrup J***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Numerous studies have shown that mothers of children with autism experience higher levels of stress compared to mothers of typically developing children. While previous research has explored the impact of various forms of support on parental stress, few studies have directly compared the efficacy of support in reducing stress and improving mental health among parents of children both with and without autism. The present study examines stress and perception of support in parents of preschool-aged children with and without autism.

Methods: Data was collected from parents of preschool-aged children (mean age 4.04 years) with (n = 36) and without (n = 48) autism. We summarized and compared perceived support and parent stress and mental health for each group. Next, we conducted regression analyses to examine how perceived levels of support and child autism diagnosis predict parent stress and mental health. Finally, we explored whether child's autism diagnosis moderated the association between perceived support and parent stress/mental health.

Results: There was no difference in the level of perceived support between parents of children with and without ASD. However, we found that parents with autistic children had higher levels of stress and anxiety overall. In regression analyses, both levels of perceived support and child's diagnosis predicted levels of parent stress and mental health. Child's diagnosis did not moderate the association between support and parent stress/mental health.

Conclusion: These findings show that, although they experience similar amounts of perceived support, parents of autistic children have significantly higher levels of stress and anxiety compared to parents of non-autistic children. As next step, we will look for potential covariates and confounding factors within these findings.

Presenter Name/Degree(s): Kimia Ghafari, BSc
Current Position: Graduate Student

Primary Mentor in Psychiatry: Matthew MacDonald, PhD

Title: Cell-type-specific synaptic proteomics in postmortem human cortex via proximity labeling and single-cell transcriptomic integration

Author(s): ***Ghafari K, Ruiz S, Barko K, Chou S, Newman J, Sweet R, and MacDonald M***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Psychiatric disorders are increasingly identified as circuit-level diseases, often involving disrupted excitatory/inhibitory balance and synaptic dysfunction. While single-cell RNA sequencing has outlined the transcriptional identities of cortical cell subtypes and cell-type specific alterations associated with psychiatric disease, mRNA profiles alone cannot fully define the molecular machinery operating at the synapse. Traditional proteomic approaches, which utilize tissue homogenization or biochemical fractionation, obscure spatial and cell-type-specific information, limiting their relevance for understanding brain architecture and disease mechanisms. To move beyond these constraints, we developed a spatially resolved proximity labeling platform optimized for postmortem human brain. By integrating this proteomic data with single-cell transcriptomics profiles, we provide a cell-type-resolved landscape of distinct synaptic proteomes in the dorsolateral prefrontal cortex (DLPFC).

Methods: Fixed DLPFC sections from postmortem neurotypical donors were immunolabeled for excitatory (vGlut1) or inhibitory (vGAT) synaptic marker antibodies, followed by secondary horseradish peroxidase (HRP)-conjugated antibodies. HRP catalyzed the localized biotinylation of proximal proteins. Biotinylated proteins were enriched by streptavidin pull-down and analyzed by LC-MS/MS. Protein identification and differential enrichment were conducted using FragPipe and R. To contextualize these proteome signatures, we compared them to cell-type-specific gene expression profiles from single-cell RNA sequencing data of the human cortex.

Results: Proximity labeling identified 1,511 proteins enriched in vGlut1- and vGAT-labeled samples relative to controls. Differential analysis revealed 379 proteins enriched in excitatory terminals and 311 in inhibitory terminals ($q < 0.05$). Functional annotation highlighted categories related to synaptic correspondence between transcript and protein-level enrichment, while also revealing additional proteins with cell-type-specific enrichment in the proteomic dataset.

Conclusion: This proximity labeling-based platform enables high-resolution, cell-type-specific proteomics in postmortem human brain. Coupled with single-cell transcriptomic integration, it offers a powerful framework for dissecting the molecular organization of human cortical circuits and provides new opportunities to investigate how these proteomic networks are disrupted in psychiatric disorders.

Presenter Name/Degree(s): Michaela Glinsky, BA
Current Position: Research Project Assistant

Primary Mentor in Psychiatry: Stephanie Stepp, PhD

Title: **Associations between borderline personality disorder, self-other boundaries, and suicide risk in romantic relationships**

Author(s): Glinsky M[1], Ellis J[1], Torres A[1], Lloyd J[1], Vanwoerden S[1], Byrd A[1,2], and Stepp S[1,2]
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Psychology, University of Pittsburgh

Introduction: Individuals with borderline personality disorder (BPD) experience heightened sensitivity to interpersonal rejection and tumultuous relationships—both of which have been repeatedly linked to increased suicide risk. Given that romantic relationships are central to adult social life and crucial for connection and belonging, the perceived closeness one feels to their romantic partner may predict risk for suicidal thoughts and behaviors (STBs), especially for those with BPD. The pictorial Inclusion of Other in Self (IOS) measure is widely used to assess relationship closeness, with studies finding lower perceived closeness among those with BPD. We aimed to explore how perceived closeness to a romantic partner during a dyadic interaction protocol may predict risk for STBs, particularly for those with BPD.

Methods: We collected data from a diverse sample of cohabitating romantic couples, with the proband reporting a recent history of STBs (N=91; mean age=26.46, 80% female). Probands repeatedly rated their perceived closeness via the IOS measure throughout a dyadic interaction protocol, with STBs and perceived relationship functioning evaluated in a 14-day daily life protocol.

Results: Preliminary analyses revealed that probands with higher BPD symptoms and lower mean levels of perceived closeness reported higher levels of suicide risk (r = 0.21- 0.27, $p < 0.05$, r = -0.25 - -0.23, $p < 0.03$, respectively) and lower levels of relationship functioning (r = 0.22 - 0.27, $p < 0.05$, r = - 0.25 - 0.30, $p < 0.03$, respectively) in daily life.

Conclusion: Although no evidence was found for a direct link between BPD and perceived closeness, both were associated with daily life outcomes that may heighten risk for STBs. Further analyses are required to better understand the relationship between these measures.

Presenter Name/Degree(s): Alexandra Gogola, MS
Current Position: Graduate Student

Primary Mentor in Psychiatry: Victor Villemagne, MD

Title: **Implementation of NIA-AA multilevel tau staging for predicting tau accumulation and cognitive decline in non-demented individuals**

Author(s): *Gogola A[1], Cohen AD[2,3], Snitz B[3,4], Minhas D[1], Tudorascu DL[2,3], Ikonomovic MD[3,4,8], Shaaban CE[3,5], Doré V[6,7], Matan C[1], DelBene A[1], Bourgeat P[7], Leuzy A[9], Aizenstein H[2,3], Lopez OL[3,4], Lopresti BJ[1], and Villemagne VL[2,3,6] for the Alzheimer's Disease Neuroimaging Initiative+*

Affiliation(s): *[1]Departments of Radiology, University of Pittsburgh; [2]Department of Psychiatry, University of Pittsburgh School of Medicine; [3]Alzheimer's Disease Research Center, University of Pittsburgh; [4]Department of Neurology, University of Pittsburgh; [5]Department of Epidemiology, University of Pittsburgh; [6]Department of Molecular Imaging & Therapy, Austin Health; [7]Commonwealth Scientific and Industrial Research Organisation Health & Biosecurity; [8]Geriatric Research Education and Clinical Center, Veterans Affairs Pittsburgh Healthcare System; [9]Critical Path for Alzheimer's Disease (CPAD) Consortium, Critical Path Institute*

Introduction: We evaluated the predictive performance of 18F-flortaucipir (FTP) tau imaging both within the NIA-AA multilevel tau staging framework and as a continuous measure with respect to cognitive decline and tau accumulation in non-demented individuals.

Methods: FTP scans from 213 non-demented participants were processed and sampled in Statistical Parametric Mapping software (SPM), version 8, using CenTauR masks. Tau accumulation and cognitive decline associations were assessed longitudinally via survival analysis. Individuals were categorized into 4 groups reflecting the NIA-AA imaging stages: Initial, with only β-amyloid (Aβ) pathology was present in PET; Early, with Aβ pathology and tau pathology in the mesial temporal region; Intermediate, with moderate tau pathology in the meta temporal region; and Advanced, with high levels of tau in the meta temporal region. Linear regressions were used to compare the longitudinal effects of baseline tau (SUVR) and tau accumulation (SUVR/year) on cognitive decline.

Results: When applying multiple levels of tau positivity, increasing stages of tau predicted both earlier tau accumulation and earlier cognitive decline. Linear regressions revealed that change in global measures of cognition (MMSE, CDR-SB) were significantly associated with baseline tau, while decline in Delayed Recall was significantly associated with both baseline tau and tau accumulation, where tau accumulation had a greater influence in the model, and Immediate Recall decline was significantly only associated with tau accumulation.

Conclusion: Implementing the multiple tau stages from the new NIA-AA biological staging framework clearly predicts distinct patterns of tau accumulation and cognitive decline. While baseline tau is predictive of global cognitive decline, tau accumulation is a better predictor of memory decline. Future work is needed to determine how the thresholds utilized here compare to visual reads and to determine the suitability of these thresholds in differentiating trajectories of individuals with cognitive impairment.

Presenter Name/Degree(s): Jennifer Grace, MS
Current Position: Research Principal Senior

Primary Mentor in Psychiatry: Michele Levine, PhD

Title: **Father engagement in obstetrical care: Black fathers' perspective**
Author(s): ***Grace J, Powe P, Joseph H, Conlon R, and Morgan J***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Obstetrical care prioritizes the health of the fetus and the mother. Research suggests that including fathers in obstetrical care improves maternal health outcomes, but fathers are often overlooked and research on their perspectives, particularly Black fathers, is lacking. Thus, we sought to better understand the involvement and experiences of Black fathers in obstetrical care to determine possible interventions and inform efforts to improve father engagement in the perinatal period.

Methods: 101 Black new or expectant fathers living in the Pittsburgh area completed surveys via Qualtrics from December 2023 until May 2024 about their experiences in obstetrical care. This survey was developed in collaboration with Black community partners with experience working with Black families, as well as a Pittsburgh-area obstetrical provider.

Results: Black fathers reported high levels of interest in and attendance at obstetrical appointments. 90% reported attending the initial OB appointment, mostly to provide support and/or advocacy for their partners (78%). While 69% reported feeling valued at obstetrical appointments, 33% reported that providers rarely asked for their opinions and 10% reported feeling ignored. Many wanted more information about supporting their partners, primarily during labor (70%), providing postpartum care (55%), recognizing symptoms of postpartum mental health concerns (54%), and mental health resources for themselves and their partners (45% and 47%, respectively). 20% of fathers reported feeling unprepared to manage their and their partner's postpartum physical or mental health.

Conclusion: Black fathers report involvement in obstetrical care and feel that their role is important, but many do not feel welcomed or included. Many fathers want to learn more about the perinatal period to better support their partner and unborn child. Some fathers feel dismissed and underutilized by providers in this setting. Medical training for providers does not typically address father engagement and inclusion, which is a possible area for intervention.

Presenter Name/Degree(s): Melanie Grad-Freilich, BS
Current Position: Graduate Student

Primary Mentor in Psychiatry: Cecile Ladouceur, PhD

Title: **The role of childhood abuse and neglect on brain function during emotional interference: Implications for depression in adolescence**

Author(s): *Grad-Freilich MJ[1], Silk J[1], Gonçalves S[2], Gonzalez N[2], Diler RS[2], and Ladouceur CD[2]*
Affiliation(s): *[1]Department of Psychology, University of Pittsburgh; [2]Department of Psychiatry, University of Pittsburgh School of Medicine*

Introduction: Childhood emotional abuse (EA) and emotional neglect (EN) are risk factors for adolescent depression. One mechanism by which these experiences could lead to depression may be their impact on emotional interference brain network functioning. We proposed a model whereby behavioral and brain correlates of emotional interference mediate the relationship between childhood EA and EN and the severity and longitudinal course of depressive symptoms.

Methods: Participants included 144 adolescents (ages 12-18, *Mage* = 15.26; 102F) with (n =113) and without (n = 31) clinical depression from a completed longitudinal study. Data included baseline Childhood Trauma Questionnaire (CTQ) measures of EA and EN, baseline emotional interference as indicated by slower reaction times and reduced negative amygdala – prefrontal functional connectivity during angry (vs. neutral) emotional face distracter conditions of an emotional working memory fMRI task, and baseline and 12-month follow-up depressive symptom severity from the child Mood and Feelings Questionnaire (MFQ) and a semi-structured interview (KSADS-PL).

Results: Baseline CTQ-EA was positively associated with baseline MFQ and KSADS depression scores, and with a longitudinal increase in MFQ and KSADS depression scores. Baseline CTQEN was positively associated with baseline MFQ and KSADS depression scores, but not with a longitudinal change in depression scores. These main effects were not partially mediated by baseline behavioral and neural markers of emotional interference. However, slower reaction times during angry (vs. neutral) conditions did uniquely predict a longitudinal decrease in MFQ scores.

Conclusion: Results support previous findings indicating a positive relationship between childhood emotional adversity and adolescent depressive symptoms. There may be a unique influence of childhood emotional abuse, but not neglect, on the course of depressive symptoms during adolescence. Neither of our behavioral nor brain emotional interference correlates appear to be the mechanism by which childhood EA and EN influence the severity and course of depressive symptoms.

Presenter Name/Degree(s): Alek Grady, BS
Current Position: Technician

Primary Mentor in Psychiatry: Matthew L. MacDonald, PhD

Title: **Integrated functional proteomics nominate key phosphoprotein-regulatory nodes essential for synaptic function in Schizophrenia**

Author(s): Grady A[1], DeMarco AG[1], Gilardi J[1], Lewin A[1], Klei L[1], Yocum AK[3], Sweet RA[1], Lewis DA[1], Devlin B[1], and MacDonald ML[1,2]
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Health Sciences Mass Spectrometry Core, University of Pittsburgh; [3]A2IDEA

Introduction: Schizophrenia (Sz) is a polygenic psychiatric disease characterized by impairments in sensory processing, social interactions, and cognition, as well as hallucinations and delusions. Decreased dendritic spine density, observed by multiple groups across multiple cortical regions, is believed to contribute to Sz's sensory and cognitive impairments. Still, the molecular mechanism driving spine loss remains unknown. Protein phosphorylation is a key driver of molecular function, altering protein localization as well as activity, and is highly dysregulated in Sz. Protein phosphorylation is stratified within the cell, with phosphotyrosine (pY) signaling acting as the upstream low-abundance gatekeeper of downstream serine/threonine phosphorylation (pS/pT). Synaptic pY signaling is activated by multiple receptor classes, including ***FGFR, NGL-3, and EPHB,*** triggering the localization of key proteins ***(VAV1, SRC, and TNK1***) from the cytosol to the synapse. Proper synaptic localization of pY proteins triggers secondary phosphorylation events, inducing the location of Sz risk gene ***MAPK3*** to the nucleus and pS/pT of transcription factors (***CREB***) and terminating the expression of synaptic proteins (***SYNGAP & PSD-95***). This study aims to understand the mechanistic role of phosphotyrosine signaling proteins in dendritic spine loss in Sz by integrating phosphoproteomics with subcellular localization data obtained from a single cohort.

Methods: We have developed a multi-faceted integrated proteomics workflow utilizing immunoprecipitation to capture tyrosine phosphorylation from human post-mortem brains. In adjacent tissue sections we have employed a previously established chemical subcellular fractionation protocol.

Results: Method development assays demonstrate >95% of pY peptides possessing >50% present call with minimal quantitative variability, accurately measuring 4,742 phosphosites on 2,030 proteins, enriched for post-synaptic density (pFDR: 2.32E-11) and calcium signaling (pFDR: 4.62E-05), key processes for synaptic function. In adjacent tissue sections from these same subjects, we adapted a subcellular fractionation protocol to enrich specific subcellular domains from brain tissue. Our analysis quantified 4,600 proteins across five fractions whose subcellular localization was assigned using a well-established algorithm

Conclusion: Together, these data will provide the foundation for elucidating the mechanistic model by which phosphotyrosine signaling cascades drive alteration in dendritic spine stability in Sz.

Presenter Name/Degree(s): Julianne Griffith, PhD
Current Position: Postdoctoral Scholar

Primary Mentor in Psychiatry: Leslie Horton, PhD; Jennifer Silk, PhD (Psychology)

Title: **Anticipatory and consummatory anhedonia in adolescent girls: Associations with daily-life positive affect**

Author(s): Griffith JM[1], Sequeira SL[2], James KM[3], Seah THS[1], Ladouceur CD[1], and Silk JS[3]
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Psychology, University of Virginia; [3]Department of Psychology, University of Pittsburgh

Introduction: Anhedonia, or the pervasive loss of interest and/or pleasure, is a common symptom of depression that predicts high levels of psychosocial impairment. Anhedonia comprises two distinct dimensions: anticipatory anhedonia ("wanting") and consummatory anhedonia ("liking") which may differentially relate to mood and depressive outcomes. One way in which anticipatory and consummatory anhedonia may lead to varying negative outcomes is through their effects on daily-life positive affect (PA) functioning and regulation. Little is known, however, regarding differential affective signatures of anticipatory and consummatory anhedonia as they appear in daily life. Research is particularly needed evaluating effects of anhedonia on daily-life PA in adolescence, a period during which PA normatively declines and depression rates rise. Thus, the present work evaluated associations between anticipatory and consummatory anhedonia and PA experience, reactivity, and regulation in a sample of adolescent girls enriched for risk for depression.

Methods: Participants included 117 adolescent girls (aged 11-13) enriched for depression risk (66.7% high-risk). Anticipatory and consummatory anhedonia were measured using the Temporal Experiences of Pleasure Scale (TEPS). Daily life PA experience, reactivity, and regulation were assessed over 16 days using ecological momentary assessment (EMA; n=54 observations).

Results: Bayesian multilevel structural equation models indicated that anticipatory pleasure (the inverse of anticipatory anhedonia) was positively related to momentary PA experience in daily life (β=.22, b=.07, 95% CI=[.02, .12]). Adolescents low in anticipatory pleasure (i.e., high in anticipatory anhedonia) reported lower PA in daily life. No other associations between anticipatory or consummatory anhedonia and daily PA functioning or regulation were found.

Conclusion: Among adolescent girls enriched for depression risk, anticipatory anhedonia relatively uniquely relates to reductions in momentary PA experience. Consistent with research in adults, other facets of daily-life PA are not related to trait anhedonia, suggesting that associations between anhedonia and everyday PA may be more complex than previously believed.

Presenter Name/Degree(s): Rebecca L. Griffith, PhD
Current Position: Postdoctoral Scholar

Primary Mentor in Psychiatry: Amy Byrd, PhD

Title: **Longitudinal associations between shared and unique components of executive function and externalizing subdimensions: Findings from the ABCD Study**

Author(s): Griffith RL[1], Vize C[2], Murtha K[3], Kahhale I[2], Henry L[4], Hawes S[5], Waller R[3], and Byrd A[1]
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Psychology, University of Pittsburgh; [3]Department of Psychology, Pennsylvania University; [4]Department of Neurological Surgery, University of Pittsburgh; [5]Department of Psychology Florida International University

Introduction: Deficits in executive function—including inhibition, working memory, and cognitive flexibility—are robustly linked to externalizing behaviors in youth. However, limited research has examined how shared versus unique components of executive function relate to distinct externalizing dimensions over time.

Methods: This study investigated differential associations between executive function components and developmental trajectories of externalizing behaviors in a large, diverse sample of youth (N = 11,875; Mage = 9.51; 48% girls). Executive function was assessed at baseline using the NIH Toolbox Cognition Battery, and externalizing behaviors were measured through parent-reported Child Behavior Checklist scores collected at four timepoints, six months apart. A confirmatory factor analysis identified a shared latent executive function factor, and latent growth curve models were estimated for a broad externalizing factor and three subdimensions: conduct problems, irritability, and neurodevelopmental difficulties. Models included time-varying covariates (i.e., other externalizing subdimensions) and time-invariant covariates (age, sex, site, income, and IQ proxy).

Results: Results showed that both shared and unique components of executive function were associated with higher initial levels of broad externalizing behavior, with the shared executive function factor showing the strongest effect ($\beta = -0.15$) and a small association with increases over time ($\beta = 0.057$). When examining subdimensions, these effects were primarily driven by neurodevelopmental problems. The latent executive function factor and individual executive function indicators were consistently associated with higher levels of neurodevelopmental difficulties (βs = -0.053 to -0.184), while associations with conduct problems and irritability were negligible/non-significant.

Conclusion: These findings suggests that the influence of executive function on externalizing behavior may be more strongly related to the development of neurodevelopmental problems rather than conduct problems or irritability. This underscores the need to account for heterogeneity within externalizing behaviors when studying executive function as a risk factor, with potential implications for understanding etiology and tailoring interventions.

Presenter Name/Degree(s): John Grizzanti, PhD
Current Position: Postdoctoral Associate

Primary Mentor in Psychiatry: Thomas K. Karikari, PhD

Title: Differential effects of Type II Diabetes Mellitus on plasma biomarkers in an Alzheimer's disease cohort: A large memory clinic study

Author(s): *Grizzanti J[1], Zeng X[1], Deek RA[4], Nafash MN[1], Gu J[1], Choity LT[1], Lafferty TK[1], Farinas MF[1], Bedison A, Mercurio RB[3], Matan C[5], Gogola A[5], Kofler J, Tudorascu DL[1], Shaaban B[3], Lingler J, Pascoal TA[1,2], Klunk WE[1], Villemagne VL[1], Ikonomovic MD[2], Berman SB[2], Sweet RA[1,2], Snitz BE[2], Cohen AD[1], Kamboh MI[3], Lopez OI[2], and Karikari TK[1]*
Affiliation(s): *[1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Neurology, University of Pittsburgh School of Medicine; [3]Department of Epidemiology, University of Pittsburgh School of Public Health; [4]Department of Biostatistics, University of Pittsburgh School of Public Health; [5]Department of Radiology, University of Pittsburgh School of Medicine*

Introduction: Alzheimer's disease (AD) is a complex, multifactorial disease, influenced by pre-existing and comorbid diseases. Type II Diabetes (T2D) remains one of strongest associated comorbidities that increases one's risk of developing AD. The use of lowly-invasive fluid biomarker assays facilitates the cheap, speedy, and accurate identification of individuals with considerable risk for developing AD or likely already have the disease. While AD fluid biomarkers have been well characterized during aging and cognitive impairment, little is known about how T2D affects AD biomarkers and even less so regarding tau-based biomarkers in blood.

Methods: A subset of participants with relevant demographic, diagnostic, and plasma biomarker data from the University of Pittsburgh's ADRC was utilized (N=1,140). Participants were stratified by cognitive diagnosis [CogDx] (Cognitively unimpaired [CU], MCI, or AD dementia) and clinically diagnosed diabetes status (non-diabetic or T2D). Specific plasma biomarkers were analyzed using Single molecule array (Simoa): Brain-derived (BD) tau, p-tau181, p-tau217, NfL, and GFAP. After a log2-transformation, AD plasma biomarkers were analyzed via 2-way ANCOVA with a Bonferroni correction.

Results: 60.6% of participants were women and 13.2% had T2D. Most had AD dementia (45.9%) or MCI (26.8%) while 27.3% were CU. There were no differences in age between men and women nor non-diabetics and T2Ds. In 2-way ANCOVA analysis, p-tau181, BD-tau, NfL, and GFAP showed stepwise increases according to CogDx (CU<MCI<AD-dementia, $p<0.001$) while GFAP ($p<0.001$) and NfL ($p=0.036$) were additionally separately affected by diabetes (T2D<CU). Lastly, p-tau217 showed an interaction between CogDx and T2D (CU=MCI<AD).

Conclusion: All AD plasma biomarkers showed a clear, stepwise increase based on CogDx (CU<MCI<AD). Novel data demonstrated a decrease in plasma GFAP and NfL levels in participants with T2D. Lastly, p-tau217 levels were increased in participants with AD+T2D vs. CU or MCI participants. These findings demonstrate potential effects of T2D status on plasma biomarker levels/performances.

Presenter Name/Degree(s): Jeremy Gu, BS
Current Position: Research Specialist

Primary Mentor in Psychiatry: Thomas K. Karikari, PhD

Title: **NIH Toolbox Cognition Battery: Associations with plasma and imaging AD biomarkers in older adults without dementia**

Author(s): ***Gu J[1] *, Zhang V[2] *, Roush R[4], Zeng X[1], Karikari T[1], Tudorascu D[3], Cohen A[1], and B Snitz[4]***
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Biostatistics, University of Pittsburgh School of Public Health; [3]Department of Psychiatry and Biostatistics, University of Pittsburgh; [4]Department of Neurology, University of Pittsburgh School of Medicine

****Each author contributed equally to this abstract.***

Introduction: The NIH Toolbox Cognition Battery (NIHTB-CB) evaluates fluid and crystallized cognitive abilities and has gained traction in AD research. Its relationship with AD biomarkers has been little investigated. This study examined these relationships using novel plasma and more established imaging biomarkers in older adults without dementia to assess NIHTB-CB's sensitivity to early-stage pathology.

Methods: This study included 258 participants (mean age 63.7 ± 8.9; 66.7% female; 49.2% non-Hispanic White) from the ***Connectomics of Brain Aging*** study. Participants underwent diagnostic classification, [11C] PiB-PET imaging, and MRI for cortical thickness (CT). Plasma BD-tau, p-tau217, p-tau181, p-tau231, Aβ42, Aβ40, GFAP, and NfL were measured using SiMoA. Linear regression analyses were conducted to explore associations between each NIHTB-CB measure and AD biomarkers controlling for age, sex, race, and years of education. Effect modification and sensitivity to outliers were also assessed.

Results: With outliers included, CT showed the most associations with NIHTB measures (Flanker, DCCS, Pattern Comp, Pic Seq, Oral Read, Fluid Composite, Crystal Composite, Total Composite, p value [0.004, 0.01]). Alzpath p-Tau 217 also showed significant relationships with DCCS [β = 0.015, p = 0.028]), Pattern Comp [β = 0.015, p = 0.002]), Pic Seq [β = -0.0018, p = 0.032]), Fluid Composite [β = 0.02, p = 0.0016]). GFAP was associated with Fluid Composite (β = -0.007, p = 0.049) and DCCS was associated with AB40 (β =-0.44, p = 0.026) and NfL (β = -0.008, p = 0.047).

Conclusion: NIHTB-CB reveals cognition-biomarker relationships, including plasma biomarkers. CT is a key marker of neurodegeneration's proximal impact on cognition. Positive associations between p-Tau 217 and NIHTB-CB may reflect selection bias towards cognitive reserve and compensatory mechanisms in early preclinical stages. Findings support NIHTB-CB's potential for detecting early cognitive changes in AD. Future research will assess predictive utility for cognitive decline and disease progression.

Presenter Name/Degree(s): Madalyn Hafenbreidel, PhD
Current Position: Postdoctoral Researcher

Primary Mentor in Psychiatry: Mary Torregrossa, PhD

Title: **Examining mechanisms of multiple memory encoding of cocaine- and fear-associated memories**

Author(s): ***Hafenbreidel M, Cole RH, and Torregrossa MM***
Affiliation(s): ***Department of Psychiatry University of Pittsburgh School of Medicine***

Introduction: Maladaptive memories, such as those associated with substance use disorder (SUD) or post-traumatic stress disorder (PTSD), are long-lasting and resistant to treatment. These memories link environmental stimuli (cues) with associated outcomes, such as drug effects or a threatening event(s). When the cues are encountered, the associative memories are recalled, which can lead to resumption of substance use or presentation of anxiety- or fear-like behaviors. These disorders are often comorbid. However, the interplay between them is understudied.

Methods: Previous research from the lab suggested that order of fear conditioning (FC) and cocaine self-administration (SA) can affect cue-induced reinstatement. To test this, rats underwent FC before SA, SA before FC, or no FC. Additionally, to explore the brain regions contributing to expression of these memories, following SA, cocaine, fear, or both memories were reactivated and then rats were euthanized to examine cFos expression in reward and learning-related brain regions.

Results: Rats that underwent FC after cocaine SA had attenuated cue-induced reinstatement compared to the other groups. Moreover, there were differences in cFos expression following retrieval of single or multiple memories. For example, in rats that underwent FC then SA, in the infralimbic medial prefrontal cortex, there was a decrease in cFos density between rats that underwent reactivation of both memories compared to controls. Tissue from rats that had FC before SA is still being processed.

Conclusion: We found that the addition of FC and when it occurred can affect cue-induced reinstatement, a model of relapse. Moreover, we found that reactivation of both memories led to different patterns of neural activation compared to each memory alone, suggesting that rats receiving both conditioning paradigms have altered circuitry. Examining the mechanisms underlying multiple memories, and how they might interact, is not well explored. Determining unique or overlapping mechanisms could lead to novel therapeutic options.

Presenter Name/Degree(s): Andrea Hall, MD
Current Position: Psychiatry Resident

Primary Mentor in Psychiatry: Eydie Moses-Kolko, MD

Title: **Simplifying the risk discussion of antidepressant use in pregnancy, a prototype web-based learning module**

Author(s): Hall A[1], Moses-Kolko E[1], Senko K[2], and Gopalan P[1]
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]University of Pittsburgh School of Medicine

Introduction: Psychiatry trainees need effective ways to learn about antidepressant (AD) risk in pregnancy. Extant resources lack easily accessible, up-to-date, quantitatively specific data for risk-benefit decision-making with patients. To address this, we assessed the feasibility of developing a 30-minute self-paced learning module on AD use and birth defect risk to enhance trainee knowledge and counselling ability.

Methods: We reviewed literature for up-to-date, quantitatively specific data on AD use and birth defects. The module was developed in Articulate 360 by integrating risk data and selecting topics, patient cases, and presentation formats in collaboration with expert scientists and instructional designers to optimize user agency, interactivity, and feedback.

Results: The module incorporates quantitative data from a study under review including relative risk values of AD use and birth defects. Content includes video and text on teratology, study design, relative risk, and provider-patient risk discussions. The module is self-paced, 30 minutes in duration, interactive with multiple-choice questions, and includes pre- and post-tests with quantitative data stored for future analysis.

Conclusion: The prototype is operational, effectively conveys information on AD use and birth defect risk, integrates a novel risk tool and models nuanced provider-patient risk discussions. The self-paced, interactive, 30-minute format engages learners, provides valuable feedback, and supports flexible learning. A significant challenge was selecting a focus from numerous psychotropic exposures and outcomes. We conclude a thorough approach requires expertise in perinatal psychiatry and input from specialists. Further development will include a pilot trial of 10-20 senior psychiatry residents with preliminary results expected by November.

Presenter Name/Degree(s): Nicholas Harris, MD, PhD
Current Position: Postdoctoral Scholar

***Primary Mentor in Psychiatry*:** Brant Hasler, PhD

Title: **Positive child experiences may mitigate associations between early life adversity and neural stress reactivity in the central visceral circuit**

Author(s): Harris N[1,2], Hasler B[1], Culyba A[2], and Banihashemi L[2]
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]UPMC Children's Hospital of Pittsburgh

Introduction: Childhood threat (CT) impacts neurodevelopment and stress reactivity, increasing risk for mental and physical health problems. Positive childhood experiences (PCEs) can buffer the adverse effects of CT on health outcomes. Here, we quantify PCEs in an abuse-enriched sample to evaluate their independent and interactive effects with trauma on neural stress reactivity within the central visceral circuit (CVC), a network implicated in stress and emotion regulation.

Methods: Participants (n=97, ages 21-35) were recruited with graded exposure to childhood physical abuse. CT severity was measured via the Trauma History Questionnaire (THQ). PCEs were quantified by aggregating scores from separate questionnaires, individually assessing positive interpersonal relationships, community connectedness, and direct support. Participants completed fMRI scans during a mild cognitive stress task, the Multi-Source Interference Task (MSIT). Linear mixed-effect models assessed neural activity differences between stress and control task conditions in the amygdala and other CVC regions. Moderation analyses using linear regression assessed the impact of CT (THQ score) on MSIT-associated neural reactivity with PCE score as moderator, controlling for age, sex, and racial/ethnic identity.

Results: PCE scores varied broadly (mean=31.21+/-8.4, range 14-46) and were negatively correlated with CT severity ($r=-0.457$, $p<0.001$). Alone, PCEs were not associated with regional CVC activation but were significantly associated with greater anti-correlated stressor-evoked connectivity between the bed nucleus of the stria terminalis (BNST) and the amygdala ($\beta=-0.21$, $p=0.011$). PCEs, however, moderated the relationship between CT and amygdala activation ($\beta=0.70$, $p=0.01$): higher trauma exposure was associated with greater amygdala stressor-evoked activity in participants with fewer PCEs but less stressor-evoked activity among those with higher PCEs.

Conclusion: These findings highlight the buffering role of positive childhood experiences (PCEs) in neural responses to stress following adversity, revealing interactions not detectable through unidimensional analyses. Future work will extend this framework to examine effects on sleep and substance use, informing targeted interventions that promote resilience and healthy trajectories.

Presenter Name/Degree(s): V. Blair Harvie, BS
Current Position: Research Specialist

Primary Mentor in Psychiatry: Andrea Goldschmidt, PhD

Title: **Breakfast skipping among children: Associations with body mass index, food insecurity, and cognitive function**

Author(s): ***Harvie B, Neiser A, Christian C, Hudson C, Hammer M, Bell V, Kinkel-Ram S, Kolko-Conlon R, and Goldschmidt A***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Skipping breakfast is a maladaptive health behavior associated with higher body mass index (BMI), elevated psychiatric symptoms, and poorer executive functioning which may be driven by intentional efforts to control body shape or weight, or by socioeconomic factors such as limited food access. This study aimed to replicate previous findings on breakfast skipping and further examine associations with food insecurity and eating-related psychopathology in a community-based sample of children.

Methods: Participants were 95 children (aged 9-12y) enrolled in a longitudinal study of eating behavior and cognitive functioning. Baseline data on self- or parent-reported eating-related psychopathology, depressive symptoms, and food insecurity, and task-based measures of working memory and planning were entered into regression analyses as predictors of breakfast skipping patterns over the week prior to assessment.

Results: More frequent breakfast skipping was positively associated with BMI percentile and negatively related to food insecurity and depressive symptoms. Lower performance on a working memory task was independently associated with more frequent breakfast skipping, but this effect disappeared when accounting for other variables. Conversely, better performance on a planning task was associated with more frequent breakfast skipping.

Conclusion: Results were consistent with previous findings showing a positive association between breakfast skipping and BMI. Contrary to previous findings, breakfast skipping was not significantly associated with food insecurity, depressive symptoms, or executive functioning. Further research is needed to better understand the association between chronic breakfast skipping, mood, and cognitive functioning, particularly in families struggling with food insecurity. Identifying the long-term benefits of breakfast consumption may better support development and implementation of programs designed to improve breakfast patterns for overall health in children.

Presenter Name/Degree(s): Kirsten S. Ho, BS
Current Position: Research Associate

Primary Mentor in Psychiatry: Benjamin L. Handen, PhD

Title: Depressive symptoms and global cognitive functioning in adults with Down syndrome

Author(s): ***Ho K[1], Peven J[2], and Handen B[1]***
Affiliation(s): ***[1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]VA Pittsburgh Medical Center***

Introduction: Depressive symptoms are linked to cognitive impairment in the aging population and may be a risk factor for and concurrent symptom of dementia. The nature of this relationship is less clear in adults with Down syndrome (DS) who face elevated risk for developing Alzheimer's disease. We investigated associations between depressive symptoms and global cognition in adults with DS at baseline and longitudinally. We hypothesized that higher caregiver-reported depression would be related to worse cognitive functioning.

Methods: Participants included 490 adults with DS aged 25-81 years enrolled in the Alzheimer Biomarker Consortium–DS (ABC-DS) study. 133 participants had up to 4 data timepoints for longitudinal analyses. The Reiss Screen for Maladaptive Behavior (RSMB) and the DS Mental Status Examination (DS-MSE) evaluate behavioral and cognitive functioning. RSMB depressive symptom subscales were combined to create an overall depression score (RSMB-D). The DS-MSE includes subtests that measure verbal abilities, memory, apraxia, orientation, and visuospatial construction. Baseline cross-sectional and longitudinal analyses used linear mixed effects models controlling for age, sex, premorbid intellectual disability, and race.

Results: Participants were, on average, 44 years old, 54.3% male, 91.2% Caucasian, and 45.1% had mild intellectual disability. After controlling for covariates, each one-point increase in baseline RSMB-D was associated with a 1.15-point decrease in baseline DS-MSE scores ($r=-0.04$, $p<0.001$). Across four timepoints (up to 64 months), DS-MSE scores declined by 1.15 points per visit ($r=-0.05$, $p<0.001$). Higher RSMB-D scores were also associated with steeper DS-MSE score declines over time, with each one-point increase associated with an additional 1.29-point reduction in DS-MSE score ($r=-0.02$, $p<0.001$).

Conclusion: Heightened caregiver-reported depressive symptoms are associated with lower baseline global cognitive function and greater declines in abilities over time in adults with DS. These findings highlight the importance of monitoring depressive symptoms, as they may help detect early cognitive decline in this high-risk group.

Presenter Name/Degree(s): Nicole Horan BS
Current Position: Graduate Student

Primary Mentor in Psychiatry: Marianne Seney, PhD

Title: **The barrier has a bedtime: Circadian control of the blood brain barrier in the nucleus accumbens**

Author(s): ***Horan N, Peterson K, Scott M, DePoy L, Ketchesin K, McClung C, and Seney M***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: The blood-brain barrier (BBB) maintains central nervous system homeostasis and protects against peripheral insults. Emerging evidence suggests BBB permeability is under circadian control, with increased permeability during the inactive phase for waste clearance and decreased permeability during the active phase to limit toxin exposure. While both circadian disruption and BBB dysfunction have been independently implicated in psychiatric disorders, their potential interaction remains poorly understood**.**

Methods: To explore circadian regulation of BBB function, we mined RNA-sequencing datasets from mouse and human nucleus accumbens (NAc), a brain region implicated in reward and mood regulation. We assessed rhythmicity of tight junction transcripts and effects of adolescent circadian disruption in mice. Additionally, we analyzed human postmortem bulk RNA-seq data from individuals with psychosis to assess changes in tight-junction rhythmicity. BBB permeability across time of day was assessed by perfusing wild-type mice with Evans Blue dye at two Zeitgeber time points (ZT1 and ZT13) and quantifying dye permeation into the NAc.

Results: Claudin-5 (***Cldn5***), a tight junction protein and marker of BBB integrity, exhibited robust circadian rhythmicity in the mouse NAc under control conditions ($p<0.0001$). This rhythmicity was significantly dampened following adolescent circadian disruption, with reduced amplitude and weaker model fit as well as a loss of rhythmicity in males($p=0.08$). Another tight-junction transcript, ***Pecam1,*** exhibited sex-specific vulnerability: females showed initial rhythmicity($p<0.05$) that was lost after disruption ($p=0.73$), whereas males lacked baseline rhythmicity($p=0.45$) but show a trend towards rhythmicity post-disruption ($p=0.09$). Similarly, analysis of human postmortem RNA-seq data from individuals with psychosis revealed reduced ***CLDN5*** rhythm amplitude ($p<0.05$).

Conclusion: These findings suggest circadian disruption impairs rhythmic expression of tight junction genes, potentially altering BBB permeability at times when the brain is normally protected, increasing vulnerability to peripheral stressors. Ongoing in vivo experiments are quantifying time-of-day differences in BBB permeability and whether circadian gene knockout disrupts temporal dynamics.

Presenter Name/Degree(s): Chloe Horter, BS
Current Position: Research Specialist

Primary Mentor in Psychiatry: Erika Forbes PhD

Title: **Exploring dopamine availability and smartphone derived GPS patterns in young people with depression**

Author(s): ***Horter C[1], Brodnick Z[1], Jones N[1,2], Calabro FJ[1], Luna B[1,2], Ryan ND[1], Seah THS[1], Medich C[1], and Forbes EE[1,2]***
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Psychology, University of Pittsburgh

Introduction: Dopamine availability (DA) is a key factor in frontostriatal function and motor activity, likely playing a role in the pathophysiology of depression and anhedonia, a symptom involving reduced motivation for and enjoyment of rewards. Similarly, smartphone-derived GPS data, which track mobility patterns, including extent of movement and time spent at home, could capture DA-mediated aspects of movement and offer insight into depression-related disruptions. Exploring how DA and mobility patterns are related is essential for improving our understanding of the neurological mechanisms and motor aspects of depression.

Methods: Young people aged 15-25 years old (***N*** = 77) who met criteria for a current DSM-5 depressive disorder completed MRI protocols measuring basal ganglia tissue iron and neuromelanin (NM), as well as questionnaires assessing depression (QIDS) and anhedonia (SHAPS). After the initial MRI session, participants began a 6-month, phone-based collection of continuous passive sensor data, including GPS location, using the EARS app. Regression analyses examined associations between DA function proxy variables and GPS variables, while controlling for age, sex, and depressive symptoms.

Results: Higher nucleus accumbens tissue iron was associated with greater distance traveled (β = 1.41, $\boldsymbol{t}(40) = 2.58$, $\boldsymbol{p} = .01$). Higher caudate tissue iron was modestly but non-significantly related to less time at home ($\boldsymbol{r}(56) = -0.21$, $\boldsymbol{p} = .11$). Higher severity of anhedonia was associated with greater time at home ($\beta = 9.50$, $\boldsymbol{t}(72) = 2.18$, $\boldsymbol{p} = .03$).

Conclusion: Higher DA function, as captured by basal ganglia tissue iron, was associated with greater extent of movement. This association suggests clinical implications of this measure of DA for exploratory and reward-driven behavior in depression. This non-invasive MRI technique has potential for understanding real-world mobility patterns in young people. Our results highlight a potential neurobiological marker for behavioral aspects of depression and anhedonia. This phenotyping could inform targeted interventions.

Presenter Name/Degree(s): Emma C. Hudson, MA
Current Position: Medical Student

Primary Mentor in Psychiatry: Helmet T. Karim, PhD

Title: **Greater stress response network (SRN) connectivity is associated with higher worry in men and women in late life**

Author(s): Hudson EC[1], Chae C[1], Karim HT[1,2], Tudorascu DL[1,3], Butters MA[1], and Andreescu C[1]
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Bioengineering, University of Pittsburgh; [3]Department of Biostatistics, University of Pittsburgh

Introduction: Anxiety disorders are among the most prevalent psychiatric conditions in the United States. However, the neural mechanisms underlying distinct anxiety phenotypes, particularly worry, and their interaction with stress regulatory networks remain underexplored. Research indicates that the Stress Response Network (SRN) is implicated in fear and stress modulation, while Executive Control Network (ECN) is implicated in emotional regulation. ECN dysfunction have been associated with persistent worry and stress reactivity in anxiety disorders. In this study, we investigated age-related differences in SRN connectivity and SRN-ECN connectivity between men and women, examining their roles in worry severity, emotional regulation, and psychological well-being.

Methods: We recruited 131 participants who were 50 years or older, normally distributed on worry severity measured by Penn State Worry Questionnaire (PSWQ). We measured resting state functional connectivity of the SRN as well as the SRN to ECN. We evaluated associations with anxiety phenotypes including worry, anxiety, and rumination as well as their correlates with sex and age.

Results: We found that SRN functional connectivity was lower with age but greater with worry. We found no sex differences in SRN functional connectivity. An exploratory analysis showed that age moderated the association between SRN and worry – we found that older participants with greater SRN connectivity had greater worry.

Conclusion: We found that SRN connectivity was lower with age, but participants with high SRN intra-network connectivity in older age reported more severe worry. We did not observe a statistically significant correlation between worry and SRN-ECN connectivity, underscoring the need for further research into the interaction between these two networks.

Presenter Name/Degree(s): Karolina Ilina, BS
Current Position: Research Assistant

Primary Mentor in Psychiatry: Cėsar Escobar-Viera, MD, PhD and Ana Radovic, MD, MSc

Title: **A social support online intervention for sexual and gender minority youth to increase help-seeking for anxiety and/or depression: Pilot randomized controlled trial**

Author(s): Ilina K[1], Escobar-Viera C[2], Smith C[1], Porta G[2], and Radovic A[1,3]
Affiliation(s): [1]Department of Pediatrics, University of Pittsburgh School of Medicine; [2]Department of Psychiatry, University of Pittsburgh School of Medicine; [3]UPMC Children's Hospital of Pittsburgh

Introduction: Sexual and gender minority youth (SGMY) are at an increased risk for depression and anxiety, while also less likely to seek mental health services. SGMY utilize online environments for social support and sharing mental health symptom experiences, making them ideal for reaching this group.

Methods: A single-blind, factorial randomized controlled trial involving SGMY aged 14-19 with at least mild anxiety and/or depression and not in psychotherapy, was conducted. After completing a baseline, participants were randomized into one of 16 groups, each receiving 0-4 intervention principles (IPs) over four weeks. IPs focused on (1) Intersectionality, (2) Confidentiality and Privacy in Healthcare Settings, (3) Educating Others, and (4) Finding Affirming Caregivers delivered on separate moderated Discord channels. One-month follow-up surveys were completed.

Results: Of the seventy-six participants who completed the trial, 76.5% identified as Black and 52.9% as gender-diverse. Usability scored 76.7 (range: 0-100). Most said their sexuality/gender was affirmed (97.3%), their privacy and confidentiality maintained (98%), and found the tools helpful (95.9%). Participants receiving IPs 3 and 4 were more likely to report intention to seek help from a mental health counselor at follow-up (***d***=0.59, ***p***=0.04). In the full group at follow-up, 31 (41%) participants sought help from a mental health professional. A participant on IP 4 commented, "I learned that its really important to advocate for things you want in a therapist...Coming in with a list of questions asking how the therapist feels and identifies with things that are important to you can help your overall experience."

Conclusion: A diverse group of SGMY experiencing depression or anxiety symptoms, but not in psychotherapy, rated the usability and acceptability of a brief, moderated Discord-based discussion forum highly after being recruited online. Specifically, those exposed to content on educating supportive adults about SGMY health and finding affirming caregivers showed increased help-seeking intention.

Presenter Name/Degree(s): Alexandra M. Izydorczak, PhD
Current Position: Postdoctoral Associate

Primary Mentor in Psychiatry: Thomas K. Karikari, PhD

Title: **Development of an immunoprecipitation mass spectrometry method for tau peptides in plasma for Alzheimer's disease diagnosis**

Author(s): ***Izydorczak AM, Zeng X, and Karikari TK***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Alzheimer's disease (AD) is a progressive neurodegenerative disease. Traditional diagnosis of AD relies on cognitive tests, which when used alone have low accuracy. Recent advancements in neuroimaging and cerebrospinal fluid biomarkers have enabled the in vivo detection of amyloid-beta (Aβ) plaque and neurofibrillary tangle pathologies, allowing for accurate biological diagnosis of AD. However, these methods are expensive and invasive, making them unsuitable for widespread use. This prompts the need of a less invasive diagnosis method using plasma. While there are some effective plasma biomarkers such as p-tau217 for Aβ pathology, there is still a lack of effective blood-based biomarkers for tau aggregate pathology. We aimed to develop a mass spectrometry-based assay to identify novel plasma tau forms indicative of tau pathology.

Methods: To enrich pan-tau peptides, we coupled a cocktail of antibodies targeting various tau regions to Dynabeads-280 sheep anti-mouse IgG beads for immunoprecipitation (IP). Recombinant tau was spiked into pooled plasma sample to evaluate IP efficiency. IPed peptides were enriched by perchloric acid (PCA) precipitation, trypsin-digested and desalted using C18 spin cartridges. Mass spectrometry was performed on an Orbitrap Exploris 480 using both data-dependent acquisition and parallel reaction monitoring (PRM). Data were analyzed using PEAKS Studio12 and Skyline V24.1.0.414.

Results: Pan-tau IP efficiently enriched spiked-in recombinant tau441. However, high-abundant plasma proteins such as albumin, IgG, and fibrinogen remained prevalent after the IP. Addition of post-IP PCA precipitation reduced the interference of high abundant plasma proteins, improving the sequence coverage of tau441. Further optimization is needed to enable detection of the endogenous plasma tau. The optimized method will be applied to clinically relevant cohorts to associate identified tau forms with Tau Positron Emission Tomography (PET) to identify tau pathology-specific tau forms.

Conclusion: Preliminary results indicate that the IPMS method effectively enriches and quantifies recombinant tau spiked into plasma samples. However, further optimization is needed to profile endogenous plasma tau and identify tau pathology-specific forms.

Presenter Name/Degree(s): Michael Janecek, BA
Current Position: PhD Student

Primary Mentor in Psychiatry: Rui Peixoto, PhD

Title: **Elevated dopamine signaling in the NAc of Shank3B$^{-/-}$ pups during maternal interaction**

Author(s): ***Janecek M[1] and Peixoto R[2]***
Affiliation(s): [1]Center for Neuroscience; [2]Department of Psychiatry, University of Pittsburgh School of Medicine

Introduction: Disrupted social reward processing is a hallmark of autism spectrum disorders (ASD). Loss of function in the Shank3 gene (Shank3B$^{-/-}$) represents a penetrant risk factor for ASD, and in adult mice disrupts the activity of dopamine neurons and social preference. Dopamine (DA) deficits could thus play a developmental role in the establishment of abnormal social behavior. Whether downstream DA release is reduced in male Shank3B$^{-/-}$ pups has not been directly measured due to technical challenges of implanting probes into rapidly expanding postnatal tissue.

Methods: To overcome these challenges, we optimized minimally-invasive tapered fiber photometry and for the first time ever recorded sub-second release of DA ($GRAB_{DA}$) in freely-moving pups aged 14-15 postnatal (P) days. We specifically quantified DA dynamics in a brain region implicated in social reward processing, the nucleus accumbens (NAc), across self-paced and social contexts.

Results: We discover that maternal interactions drive subcortical DA signals as early as P15. Exploiting the maternal salience, we uncover that Shank3B$^{-/-}$ pups exhibit elevated DA release compared to wildtype controls at the onset of pup-dam interactions. DA release adapted over the course of maternal interactions in all mice, but during self-paced behavior the Shank3B$^{-/-}$ pups exhibited a paradoxically reduced rate of DA release in the NAc.

Conclusion: We demonstrate the feasibility of directly measuring DA dynamics in P15 pups and find that NAc DA signaling is already present and robust at this early stage. Remarkably, our findings indicate that the DA system may be hyperactive in juvenile Shank3B$^{-/-}$ animals, but this phenotype emerges only in specific social contexts, indicating that an early disruption of specific DA dynamics might underlie abnormal reinforcement of social behavior. Our study highlights the need for ethologically-relevant measurements of early postnatal behavior and brain activity in order to reveal nuanced developmental trajectories.

Presenter Name/Degree(s): Sabine Janssen, BS
Current Position: Research Manager

***Primary Mentor in Psychiatry*:** Fabio Ferrarelli, MD, PhD

Title: **Descrying phasic and tonic REM sleep, from healthy control's EEG oscillatory activity, with no significant cognitive correlations**

Author(s): Janssen SA, Sanguineti C, Moore C, Mayeli A, and Ferrarelli F
Affiliation(s): Department of Psychiatry, University of Pittsburgh School of Medicine

Introduction: Delta-theta frequency oscillations, also called sawtooth waves, are important components of rapid eye movement (REM) sleep. Sawtooth waves are more commonly coupled with eye movements during phasic REM. However, the occurrence and other characteristics of these waves during phasic and tonic REM sleep remain largely undetermined. In this study, we characterize the properties of sawtooth waves during both phasic and tonic REM; we also explored their correlations with cognition.

Methods: Twenty healthy individuals completed high density sleep EEG recordings with 62 channels. REM sleep was visually identified and divided into 6-second epochs. These REM epochs were then further scored as phasic or tonic based on the presence or absence of rapid eye movements, respectively. Next, we automatically extracted several sawtooth wave parameters (density, integrated activity, positive peak amplitude, average upslope, average downslope, and duration) with different period (1-2, 1-4, 1-6, 2-4, 2-5, 2-6 Hz frequency ranges) and amplitude (0, 5,10, 20 μV) thresholds. Using an FDR-corrected paired t-test and Pearson's rho, we then examined correlations between the MATRICS™ Consensus Cognitive Battery (MCCB) cognitive tests and sawtooth wave parameters.

Results: Higher sawtooth integrated activity and wave density were found in fronto-central areas in phasic REM when compared with tonic REM. This effect was observed only when waves of higher frequencies (5-6 Hz) and lower amplitudes (0 or 5 μV) were included. Furthermore, positive peak amplitude for phasic REM was lower than during tonic REM in centro-parieto-occipital regions across all amplitude thresholds and frequency ranges > 2Hz. No significant correlations were found between phasic and tonic parameters and cognitive performance.

Conclusion: Our data shows that phasic and tonic sawtooth REM waves have different characteristics, especially in fronto-central regions, in healthy individuals. Future research should assess and compare these characteristics across tonic and phasic REM between non-clinical and clinical populations.

Presenter Name/Degree(s): Alex Jo, BS
Current Position: Student Research Assistant

Primary Mentor in Psychiatry: Rebecca Thurston, PhD

Title: **Perceived barriers and facilitators to an integrated treatment for insomnia and PTSD symptoms in women: A qualitative analysis**

Author(s): Jo A[1], Cameron FA[2], Thurston RC[1], and Doyle C[1]
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Medicine, University of Pittsburgh

Introduction: Women are ~50% more likely to develop post-traumatic stress disorder (PTSD) after trauma exposure, and 58% more likely to report insomnia compared to men, making women a higher risk group for comorbid PTSD and insomnia. Treating both insomnia and PTSD is critical, as treating one disorder often fails to resolve the other. However, few integrated PTSD-insomnia treatments exist. Brief Behavioral Therapy for Insomnia (BBTI) and Written Exposure Therapy (WET) are ideal for integration due to their brevity and effectiveness. However, it is unknown whether women with insomnia and PTSD would be receptive to an integrated insomnia – PTSD treatment approach. The aim of this study was to assess integrated intervention facilitators and barriers among women with PTSD and insomnia symptoms.

Methods: Participants were 34 women across 5 virtual 90-minute focus groups and 2 interviews who met criteria for insomnia and PTSD through the Posttraumatic Checklist 5 (PCL-5) and Insomnia Severity Index (ISI). Participants were asked about 1) perceived barriers to treatment 2) interest in and perceived benefits of an integrated treatment and 3) treatment structure preferences. Focus group transcripts were analyzed using thematic analysis.

Results: Thematic analysis of transcripts revealed: (1) women preferred a flexible, holistic treatment approach that addressed both PTSD and insomnia symptoms; (2) they favored a digital treatment format (i.e., virtual sessions, digital sleep diaries) to accommodate busy schedules; and (3) some participants welcomed a written approach to PTSD treatment.

Conclusion: Women with PTSD and insomnia symptoms expressed interest in a tailored, holistic integrated treatment. Preferences for virtual sessions and digital diary options reflected perceived barriers to treatment such as time constraints and difficulty maintaining paper diaries. These insights support the development of a customizable treatment manual integrating BBTI and WET, with options for delivery format, frequency, and diary type to accommodate individual preferences and promote engagement.

Presenter Name/Degree(s): Amanda L. Johnston, BS, BA
Current Position: Research Project Coordinator

Primary Mentor in Psychiatry: Greg Siegle, PhD

Title: **Public assistance as a risk factor for brain sequelae of chronic trauma**
Author(s): ***Johnston A[1], D'Andrea W[2], and Siegle GJ[1]***
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]The New School for Social Research

Introduction: Chronic trauma (CT) exposure and low socioeconomic status (SES) have independently been linked to alterations in resting-state brain activity, particularly in the default, executive, and salience networks—regions associated with self-processing, executive control, and threat reactivity, respectively. Public assistance (PA) use, often a marker of low SES, is also associated with increased trauma exposure, yet the combined effects of CT and PA use on neural function remain unclear. Understanding whether CT and PA exert additive effects on brain function could enhance early identification and intervention strategies for trauma-exposed individuals.

Methods: Seventy-four adults (ages 18–65) from the Blunted and Discordant Affect (BADA) study with a history of psychopathology were stratified into groups based on trauma exposure (acute vs. chronic) and PA use. Trauma duration was measured via the Trauma History Questionnaire (THQ). Resting-state brain activity was assessed using Amplitude of Low Frequency Fluctuations (ALFF) in the default, executive, and salience networks via fMRI. A univariate ANOVA was conducted to compare ALFF across trauma and PA groups, followed by LSD post hoc tests.

Results: Significant group differences emerged in ALFF measures across all three networks of interest ($\boldsymbol{p} < .05$). Post hoc comparisons using the LSD test revealed that individuals with both CT exposure and PA use exhibited significantly lower ALFF activity than those with either acute trauma or chronic trauma alone, across all networks of interest ($\boldsymbol{d} < -2.3$, $\boldsymbol{p} < .05$).

Conclusion: The combination of chronic trauma and PA use is associated with compounded reductions in resting-state brain activity within critical functional networks. These additive effects suggest that individuals experiencing both risk factors may be particularly vulnerable to trauma-related neural dysfunction. These findings underscore the importance of targeted, trauma-informed interventions for PA-using populations with chronic trauma exposure.

Presenter Name/Degree(s): Ila Abhijeet Joshi
Current Position: Undergraduate Student

Primary Mentor in Psychiatry: Marta Peciňa, MD, PhD

Title: **μ-Opioid modulation of expectancy-mood dynamics during acute antidepressant placebo effects**

Author(s): ***Joshi I, Kosterine A, Snyder I, Strohecker E, Badham G, Karim H, Ferrarelli F, Price R, Dombrovski A, and Pecina M***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Major Depressive Disorder (MDD) affects over 280 million people globally and is a leading cause of disability. Despite advances in treatment, many antidepressants fail to outperform placebo, underscoring the need to understand the neurobiology of placebo effects. Endogenous opioids are implicated in placebo analgesia and emerging evidence suggests they play a similar role in depression. Prior work from our lab showed that placebo-induced opioid release predicts antidepressant placebo response and can be blocked by the μ-opioid antagonist naltrexone. However, it remains unknown whether opioid modulators can both enhance and diminish these effects.

Methods: We randomized 101 unmedicated individuals with MDD to receive the μ-opioid receptor agonist buprenorphine, the μ-opioid antagonist naltrexone, or placebo. After administration, participants completed an fMRI task designed to manipulate placebo-associated expectancies and their reinforcement while measuring both expected and actual mood improvements.

Results: Consistent with prior findings, expectancy and reinforcement conditions significantly enhanced participants' expectancy and mood ratings. Naltrexone reduced the effect of expectancy on positive expectancy ratings compared to placebo. In contrast, buprenorphine amplified expectancy effects on expectancy ratings. Neither drug moderated the effects of the task on mood ratings. Neuroimaging revealed that naltrexone decreased activation in dorsal attention and salience networks—regions involved in processing expectations—while placebo and buprenorphine conditions showed a linear increase in activation, with buprenorphine producing the strongest response.

Conclusion: These findings confirm that antidepressant placebo effects are mediated, at least in part, by the endogenous opioid system. While naltrexone attenuated expectancy-induced responses, buprenorphine enhanced them, providing compelling evidence that μ-opioid signaling modulates antidepressant placebo mechanisms. This work advances our understanding of the molecular pathways underlying placebo effects in depression and highlights potential targets for enhancing treatment outcomes.

Presenter Name/Degree(s): Riley J. Jouppi, M.S.
Current Position: Ph.D. Student

Primary Mentor in Psychiatry: Michele D. Levine, Ph.D.

Title: **Exploring descriptives and correlates of distress associated with loss of control eating across the perinatal period**

Author(s): Jouppi RJ[1], Call CC[2], Kolko Conlon RP[2], and Levine MD[1,2]
Affiliation(s): [1]Department of Psychology, University of Pittsburgh; [2]Department of Psychiatry, University of Pittsburgh School of Medicine

Introduction: Loss of control eating (LOC), feeling unable to control the type/amount of food consumed, is highly prevalent during pregnancy and linked to adverse health outcomes. LOC is central to psychological disturbance in nonpregnant individuals, but it is unknown if LOC-related distress differs in pregnancy. This study explored LOC distress and correlates of its change across the perinatal period.

Methods: In a longitudinal community sample of pregnant participants with prepregnancy BMI≥25, those who endorsed LOC at any timepoint (***n*** range=14-61) reported LOC distress (1=Not at all; 5=Extremely) and eating disorder symptoms on the Eating Disorder Examination-Pregnancy Version (EDE-PV) in the 3 months prior to pregnancy (PTP); monthly across pregnancy (T0-5); and at 6 months postpartum (T6). Differences in LOC distress between PTP-T0, T0-T5, and T5-T6 were examined with ***t***-tests. Correlations between changes in LOC distress and EDE-PV subscale scores were estimated.

Results: LOC distress decreased from PTP to T0 (***M***=2.8 vs. 2.2; ***p***=.01), was stable across pregnancy (***M***=2.2 vs. 1.8; ***p***=.24), and increased from T5 to T6 (***M***=1.8 vs. 2.7; ***p***=.01). Changes in LOC distress were not associated with EDE-PV scores (***p***s>.10).

Conclusion: Despite documented increases in LOC prevalence during pregnancy, LOC distress was lower in pregnancy vs. prepregnancy and postpartum. Future research should replicate these findings and determine factors underlying changes in LOC distress across the perinatal period.

Presenter Name/Degree(s): Karla Joyce, MSW, LCSW
Current Position: Research Operations Coordinator

Primary Mentor in Psychiatry: David Brent, MD

Title: **Benefits of comprehensive clinical internship programs**
Author(s): ***Joyce K, Stahler J, Milford-George B, Monteverde C, Bigley K, and Porta G***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Research indicates that internship programs play a vital role in the education and professional development of students pursuing degrees in mental health related fields. Comprehensive internship programs allow students to apply theoretical knowledge learned in the classroom, assist students in making informed decisions regarding career pathways, and provide opportunities for entry into the workforce.

Methods: In 2018, the STAR Research Department introduced a comprehensive graduate student internship program. This program primarily focused on master's level students pursuing degrees in psychology, counseling, and social work. The STAR internship program was designed to expose students to the field of mental health research, contribute to the development of clinical skills, and provide mentorship and career development opportunities through weekly supervision meetings with licensed field supervisors. Students who participated in the STAR internship program were provided with comprehensive training that included an orientation period focused on the fundamentals of research, a rigorous clinical training program that prepared them to work with teens at risk for suicide, and additional training opportunities centered on general career development.

Results: To date, 35 graduate students have participated in the STAR Research Department's program. Of these students, 27 were master's or doctoral level students in psychology/counseling, and 8 were master's level students in social work. All 35 of these students fully completed the internship requirements.

Conclusion: Graduate internship programs allow students to gain clinical skills and experience, improve career readiness, and provide motivation to pursue careers in the mental health field upon graduation. An additional benefit of well-developed internship programs is the reciprocal relationship that develops between the internship placement site and the student. As interns gain clinical skills and experience, they take on various responsibilities within the department, which benefits both the student and the internship placement site.

Presenter Name/Degree(s): Megan Julien, BHA
Current Position: Research Coordinator

Primary Mentor in Psychiatry: Erika Forbes, PhD

Title: **Changes in anhedonia and depression with a single ketamine infusion in youth with depression**

Author(s): Julien M, Seah THS, Price R, Pogue AM, Ryan N, Eckstrand K, Vanderschelden B, Rengasamy M, Jones N, and Forbes EE
Affiliation(s): Department of Psychiatry, University of Pittsburgh School of Medicine

Introduction: Anhedonia is a key symptom of depression associated with poorer treatment response and suicidality, particularly among youth. Targeting anhedonia is thus critical for improving symptom course. Recent research suggests that ketamine exerts rapid-acting antidepressant effects on anhedonia by altering the function of frontostriatal circuitry. We examined the short-term effects of ketamine on anhedonia and depressive symptoms in young adults with treatment-resistant depression. The developmental focus enables early intervention of depression, with potential to mitigate its chronicity and severity.

Methods: In a larger longitudinal study involving the multimodal assessment of anhedonia (e.g., digital evaluations, behavioral tasks, fMRI), ketamine was administered to participants who did not respond to a 2-week trial theta burst stimulation (TBS). Twenty-three participants (78% female; Mage=20.5 years; range: 19–25 years) completed a single-session ketamine infusion. Participants met DSM-5 criteria for a current depressive disorder, had failed one antidepressant trial (≥6 weeks at adequate dose), and had no history of bipolar disorder, psychosis, lifetime ketamine or PCP use, or moderate-to-severe substance use within the past 6-months. Anhedonia and depression symptoms were assessed at pre- and post-infusion, using the Snaith-Hamilton Pleasure Scale (SHAPS; Snaith et al., 1995) and the Quick Inventory of Depressive Symptomatology (QIDS; Rush et al., 2003).

Results: The mean time between pre- and post-infusion was 24 hours (range: 0-72 hours). Before ketamine infusion, both the mean SHAPS and QIDS scores were in moderate range. Paired samples t-tests indicated that anhedonia decreased pre-post ketamine (M = 34.863→ 33.00, SDs = 7.357, 7.856), t (22) =2.273, p=.034. There was no significant change in depression (p=.072).

Conclusion: Results suggest that a single ketamine infusion could potentially reduce anhedonia more than depression in youth with treatment-resistant depression, including those who did not show a meaningful response to TBS. These results support claims that ketamine acts through neural reward systems.

Presenter Name/Degree(s): Mariya Kaminsky, PhD
Current Position: Postdoctoral Associate

Primary Mentor in Psychiatry: Colleen McClung, PhD

Title: **Ketogenic diet as potential Treatment for bipolar disorder**

Author(s): ***Kaminsky M[1, 2], Fairbanks N[1, 2], Inzano J[1, 2], and McClung C[1,2]***
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine;
[2]Translational Neuroscience Program, University of Pittsburgh

Introduction: Bipolar disorder is a common and debilitating mood disorder. It is characterized by aberrant GABAergic and dopaminergic signaling, as well as mitochondrial dysfunction and oxidative stress. There is recent interest in the ketogenic diet as a treatment for bipolar disorder with small case study reports of efficacy. However, the neurobiological mechanisms by which ketone bodies might ameliorate symptoms of bipolar disorder are yet to be determined. Here we investigate whether ketogenic diet rescues manic-like behavior in ClockΔ19 mice and whether it leads to changes in gene expression in the nucleus accumbens and ventral tegmental area of the brain.

Methods: Homozygous ClockΔ19 and wild type (WT) male and female mice (n = 8-10/treatment and genotype group) were treated with a control chow (Research Diets D19082304) or a ketogenic diet (Research Diets D10070801) for four weeks. Then mice went through behavioral tests in the following order: locomotor activity, open field, dark/light box, elevated plus maze and forced swim test. Following testing, blood ketone levels were measured using the ketone monitoring system (Abbott); mice were sacrificed, brains were rapidly extracted and flash frozen, punches from the NAc and VTA were taken and RNA isolated with RNeasy Plus Micro Kits (Qiagen) followed by CDNA synthesis (Invitrogen) for quantitative PCR analysis.

Results: Our results show that the ketogenic diet increased levels of β-hydroxybutyrate in the blood of both homozygous ClockΔ19 and WT mice. The ketogenic diet normalized the abnormally high novelty seeking behavior in female ***Clock***Δ19 mice ($p<0.05$), with no effect in the males in the light dark box. In comparison, the diet produced an antidepressant-like response in both males ($p<0.05$) and females ($p<0.001$) in the forced swim test. Interestingly ketogenic diet decreased locomotor activity in female WT mice ($p<0.05$) only. No differences were observed in the time spent in the center in the open field test or in the time spent in the open arms in the elevated plus maze following the ketogenic diet. We have also found that keto diet led to a significant decrease in tyrosine hydroxylase (a rate-limiting enzyme in dopamine synthesis) in ClockΔ19 mice, suggesting that the diet reduces their aberrant hyper dopaminergic transmission.

Conclusion: Our results suggest that the ketogenic diet affects mouse behavior relevant to bipolar disorder and reduces hyper dopaminergic transmission in the VTA.

Presenter Name/Degree(s): Judah Kass
Current Position: Undergraduate Student

Primary Mentor in Psychiatry: Rachel Vaughn-Coaxum, PhD

Title: **Parental acceptance and rejection: Examining its impact on sexual and gender minority youth depression severity**

Author(s): *Kass J, Johnston K, Mills B, Day S, and Vaughn-Coaxum R*
Affiliation(s): *University of Pittsburgh, Department of Psychiatry, Department of Psychology*

Introduction: Sexual and Gender Minority youth, (those who identify as non-heterosexual, or non-cisgendered; SGM) face a disparity in internalizing problems. The experience of acceptance in the parent-child relationship has been shown to be a significant protective factor for SGM youth's mental health. Conversely, parental rejection has been found to significantly exacerbate negative mental health outcomes. Given the high rate of rejection reported by SGM youth, research exploring these associations is critical, particularly when youth are already experiencing mental health difficulties. This study explores effects of parental acceptance and rejection on depression symptom severity for SGM and non-SGM youth experiencing clinically elevated depression symptoms.

Methods: Participants were youth ages 12-15 with clinically elevated depression symptoms, and a parent (N = 110 youth/parent dyads). Youth and parents completed a clinical diagnostic interview and self-report surveys where youth reported their sexual orientation and gender identity. Both youth and parents completed a measure of parents' acceptance and rejection behaviors (Parental Acceptance and Rejection Questionnaire; PARQ), and the youth's depression symptom severity (Mood and Feelings Questionnaire).

Results: There were no mean differences in youth depression symptoms or PARQ scores (by youth or parent report) for SGM youth and non-SGM youth. However, PARQ scores were correlated with symptoms among SGM youth. Interaction effects tested via linear regression analyses revealed that higher levels of parent-reported rejection ($F=5.35$, $p=.02$) and lower warmth ($F=6.75$, $p=.01$) were associated with higher symptoms reported by SGM youth than non-SGM youth. Despite significant interactions, F-tests suggest parent behaviors were not particularly strong predictors of symptom severity in this sample.

Conclusion: While there is some indication that parental acceptance and rejection is associated with greater depression symptoms in SGM youth, more research is needed to understand how parent behaviors (both in general and specific to youths' identity) impact the course of youth depression.

Presenter Name/Degree(s): Megan "Memphis" Kastner, BS
Current Position: Peer Specialist I

Primary Mentor in Psychiatry: Danella Hafeman, MD, PhD

Title: **Mental health trends and demographic insights in STEAM peer support**
Author(s): ***Kastner M, Mazlo A, Belback E, Goldstein T, and Hafeman D***
Affiliation(s): ***Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Teens with mood disorders often have difficulties with emotional regulation, social engagement, and overall mental health stability. The STEAM Peer Support Program aims to remediate these barriers by connecting participants with peer support specialists with lived experience in managing mood disorders. This study will examine the demographic characteristics of enrolled teens, and identify differences in self-reported mental health from intake to the follow up period of 3 months.

Methods: Subjects included patients (16-25 y.o.) who were diagnosed with a mood disorder and were patients of the CABS, STAR and CoSTAR clinics. Participants were given peer support through trained individuals who had lived experience. Peer support specialists provided mentorship through structured psychoeducational sessions, goal-setting frameworks, and socialization opportunities, helping participants navigate their mental health challenges. Standardized behavioral health screenings were conducted at intake, 3 months, and 12 months to monitor changes in overall mental health; given that enrollment and follow-up is still in progress, we focus on 3-month follow-ups.

Results: Thus far, we have enrolled 55 participants (n=27, 49.1% female); recruitment has met or exceeded targets. This has been a racially and ethnically diverse sample, with 12 (21.8%) identifying as multiracial and 5 (9.1%) as African American. Of these, 27 (49.1%) have completed 3-month follow-ups so far. Peer specialists have successfully integrated into the clinics, as evidenced by high referral rates and qualitative program feedback. Over the course of three months, the percentage of patients that categorized their overall mental health as "Very Good" or "Excellent" increased from 3.3% to 22.25%.

Conclusion: Results identify the benefits of lived-experience peer mentorship in supporting the well-being of adolescents and the improvement of self-reported mental health over time. Structured peer-led interventions may improve emotional resilience and social functioning and should be further developed to improve access and support adolescents across varying demographics.

Presenter Name/Degree(s): Jack Kavanagh, M.Phil
Current Position: Research Specialist

Primary Mentor in Psychiatry: Brian Coffman, PhD

Title: **Investigating auditory segmentation deficits in the cingulate motor area of first episode psychosis**

Author(s): ***Kavanagh J, Rhorer H, Seebold D, Fowler L and Coffman B***
Affiliation(s): ***Clinical Neurophysiology Research Laboratory, Department of Psychiatry, University of Pittsburgh School of Medicine***

Introduction: Auditory Segmentation Potentials (ASPs) are event-related potentials (ERPs) that are thought to reflect the brain's classification of an acoustic pattern from multiple auditory stimuli. Previous EEG data from our lab has shown reduced ASPs in the Cingulate Motor Area (CMA) for individuals within 1 year of treatment for first episode psychosis (FEP). This present study aims to further investigate our previous EEG results by recording ASPs with Magnetoencephalography (MEG) while attending or ignoring to acoustic patterns.

Methods: 9 FEP and 14 matched healthy controls (HC) completed two tasks of ignoring or attending to standard musical notes while watching a silent video. ASP's were recorded from 200ms-900ms periods after onset of the first tone within CMA and bilateral Auditory cortex, and subcortical ROI's. MEG was recorded using a MEGIN Triux.

Results: Statistical analysis of MEG data showed several significant ASP power differences during both attend and ignore tasks for FEP and HC groups. Specifically, within the right Lateral belt ($p=0.01$) and left and right dorsomedial thalamus ($p=0.02$). There were also significant differences when comparing between ignoring and attending, with a significant lack in activity in FEP's specifically in left A1 region ($p=0.03$) and bilateral dorsomedial thalamus ($p= 0.02$).

Conclusion: These results indicate aberrant ASP amplitude in multiple areas of the brain, including the CMA (SCEF), the Lateral Belt, the A1, and the dorsomedial thalamus, which are key areas of the cortico-striatal-thalamo-cortical and cerebello-thalamo-cortical circuit, thus showing deficits in the CSTC circuit for first episode psychosis.

Presenter Name/Degree(s): Lauren Keller, BS
Current Position: Research Coordinator

Primary Mentor in Psychiatry: Adriane M. Soehner, PhD

Title: **Melanopsin-driven light responsivity and reward motivation in young people at risk for mania**

Author(s): ***Keller L[1], Kuzemchak M[1], Wallace ML[1], Sollie B[1], Elia N[1], Caswell A[1], Chan S[1], Hasler BP[1], Roecklein KA[2], and Soehner AM[1]***
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Psychology, University of Pittsburgh

Introduction: Mania is associated with circadian and reward dysregulation, and these systems share strong reciprocal relationships. To better detect mania risk, it is essential to characterize abnormal biobehavioral relationships between circadian and reward systems. Light signals conveyed through melanopsin-containing retinal ganglion cells influence the biological clock. We examined whether melanopsin-driven light responsivity (via pupillometry) is associated with reward dysregulation in young people at-risk for mania.

Methods: Ninety-five participants aged 16-24yr (***M***=21.82, ***SD***=2.02) spanning a spectrum of mania vulnerability (MOODS-SR-Lifetime, MOODS) completed a 24-hr lab visit. Testing included melanopsin-driven pupil responsivity (post-illumination pupil response, PIPR), reward-based aggression (Point Subtraction Aggression Paradigm, PSAP), and reward motivation (Effort Expenditure for Rewards Task, EEfRT). PIPR was estimated at 10-40sec (PIPR30) post light stimulus, calculated as percent of baseline. Poisson regression models assessed PIPR's associations with reward outcomes and moderation by lifetime mania risk (MOODS mania score), adjusting for age, sex assigned at birth, past-week mood symptoms, psychotropic medication use, time awake, photoperiod, and MOODS depression score. Johnson-Neyman intervals examined the range of significant moderation (interaction) effects.

Results: There was a significant interaction (RR=0.31. p<0.001) between melanopsin-driven light sensitivity (PIPR30) and mania risk (MOODS-Mania) for reward-related aggression (PSAP percent steals). A Johnson-Neyman test showed that lower PIPR30 was associated with greater PSAP percent steals at low/moderate mania vulnerability (MOODS-Mania values <16), but this relationship was reversed for individuals with higher levels of mania vulnerability (MOODS-Mania> 23). There was not a significant association between PIPR30 and reward motivation (EEfRT % hard choices; RR=0.11, p=0.586) nor a significant moderating effect of mania risk (RR=1.00 p=0.368).

Conclusion: Interim findings suggest that, among individuals with high mania risk, lower melanopsin-driven pupil responsivity is associated with greater reward-related aggression. This may reflect abnormal circadian-reward system interactions in individuals with elevated mania vulnerability, contributing to irritability and aggression in mania/hypomania.

Presenter Name/Degree(s): Ameya Kharade
Current Position: Student Researcher

Primary Mentor in Psychiatry: Helmet T. Karim, PhD

Title: **Predicting age using resting state connectomes with deep curriculum based learning**

Author(s): ***Kharade A[1] and Karim HT[1,2]***
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Bioengineering, University of Pittsburgh

Introduction: Aging significantly alters the brain's functional organization but identifying robust lifespan markers using functional brain connectivity has proven to be challenging. Accurate functional age estimation may enable early detection of pathological decline, yet standard single-stage models often lack precision and interpretability. We utilized a curriculum deep learning framework to address this gap.

Methods: We trained a machine learning model using a multi-stage curriculum learning framework, progressively tackling more difficult tasks: (1) classifying age into quartiles, (2) refining into decile bins, and (3) predicting continuous age. This approach encourages gradual feature abstraction. We used a ResNet-18 architecture trained on functional connectivity matrices derived from the Dallas Lifespan Brain Study (n=315), preprocessed and mapped using the Schaefer400 and Shen atlases. Connectivity matrices from varying resting-state time windows were included for data augmentation. We used a 75%-15%-15% train, validation, and test split. We conducted ablation studies to assess the value of each training stage and used permutation testing to identify key brain networks. Generalizability was tested via sex classification using the same architecture.

Results: Our model predicted age with a mean absolute error (MAE) of 4.83 years ($p < 0.001$) and a correlation of 0.948. Removing the decile stage increased MAE to 7.58 ($p < 0.001$). Permutation testing highlighted the Default Mode, Somatomotor, and Visual networks as key contributors to aging. For sex classification, the model achieved 93% accuracy and an AUC of 0.971.

Conclusion: Curriculum learning enables a powerful and interpretable approach for modeling brain aging using functional connectivity and generalizes well to other prediction tasks like sex classification. We propose extending this model to detect early cognitive dysfunction in Alzheimer's Disease and stage functional brain changes over time.

Presenter Name/Degree(s): Shruti Kinkel-Ram, MA
Current Position: Clinical Psychology Intern

Primary Mentor in Psychiatry: Brian Thoma, PhD

Title: **An intersectional examination of weight and gender identity-based minority stress on depression symptoms among gender minority youth**

Author(s): Kinkel-Ram SS[1], Thoma B[1], Roberts S[2], Choukas-Bradley S[2], Puhl R[3], and Watson R[3]
Affiliation(s): [1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Psychology, University of Pittsburgh; [3]Department of Human Development and Family Sciences, University of Connecticut

Introduction: Gender minority (GM) youth are at increased risk for experiencing gender minority stress as well as weight stigma, but the LGBTQ+ research field currently lacks studies examining intersectional identities and related mental health outcomes. Hence, the aim of our study was to examine how GM stress and weight-based bullying relate to depression symptoms among GM youth across two large-scale cross-sectional studies.

Methods: Study 1 included data from the 2017 LGBTQ Teen Study (***n*** = 5637) whereas study 2 included data from the 2022 LGBTQ National Teen Survey (***n*** = 10,964). All participants were between the ages of 13 and 18 and completed online. Multiple regression analyses were used to test if GM stress and WS related to participants' depression scores. Missing data were handled using listwise deletion. All analyses were conducted across both studies after accounting for age, race/ethnicity, BMI, sex assigned at birth, gender identity, and sexual orientation as covariates.

Results: In Study 1, stress related to revealing LGBTQIA+ identity, gender identity-based bullying at school, weight-based bullying from family, and weight-based bullying at school each significantly predicted depression symptoms (β = .11-.21, all ***ps***<.001). Together, GM stress, WS, and covariates explained 19.3% of the variance in depression symptoms (***F*** (10, 3234) = 77.29, ***p*** < .001).

In Study 2, online gender-based bullying, gender identity-based bullying at school, online weight-based bullying, weight-based bullying from family, and weight-based bullying at school each significantly predicted depression symptoms (β = .05-.12, all ***ps***<.001). Together, GM stress, WS, and covariates explained 12.1% of the variance in depression symptoms (***F*** (12, 9777) = 111.85, ***p*** < .001. Bivariate correlations also revealed significant associations across these study variables for both studies.

Conclusion: GM youth experience minority stress across multiple domains, contributing to deleterious consequences of mental health, including increased risk for depression and suicide symptoms.

Presenter Name/Degree(s): Mei-Chuan Ko, PhD
Current Position: Professor

Title: **Does a highly G protein-biased mu opioid receptor agonist have an improved therapeutic profile?**

Author(s): ***Ko MC[1], Ding H[2], and Zhang Y[3]***
Affiliation(s): ***[1]Department of Psychiatry, University of Pittsburgh School of Medicine; [2]Department of Translational Neuroscience, Wake Forest University School of Medicine; [3]Research Triangle Institute, Research Triangle Park***

Introduction: SR-17018 was identified as a G protein-biased mu opioid peptide (MOP) receptor agonist with the highest bias factor and lacked MOP agonist-associated adverse effects in mice. The aim of this study was to determine the functional profile of spinal and systemic delivery of SR-17018 in non-human primates.

Methods: In vivo effects of SR-17018 were compared with those of MOP agonists in different intrinsic efficacies, DAMGO, morphine, heroin, and buprenorphine, in a series of behavioral assays established in rhesus monkeys (***Macaca mutatta***) (n=4 per study endpoint). Nociceptive, itch-scratching, and operant behavior were measured by experimenters blinded to the dosing conditions.

Results: Following intrathecal administration, SR-17018 (30-300 ug), buprenorphine (3-10 ug), morphine (10-30 ug), and DAMGO (1-3 ug), dose-dependently attenuated capsaicin-induced thermal allodynia ($p < 0.05$). However, unlike DAMGO and morphine eliciting robust itch-scratching activities, intrathecal SR-17018 and buprenorphine only elicited mild scratching activities, indicating that SR-17018 has low efficacy for activating MOP receptors. In the intravenous drug self-administration assay, heroin (0.3-10 ug/kg/infusion) produced a higher reinforcing strength (abuse liability) as compared to lower reinforcing strengths by SR-17018 (3-30 ug/kg/infusion) and buprenorphine (1-10 ug/kg/infusion) in primates under the progressive-ratio schedule of reinforcement ($p < 0.05$).

Conclusion: The intrathecal opioid-induced itch-scratching and intravenous drug self-administration have been documented to distinguish MOP receptor agonists with different intrinsic efficacies in primates. Our findings reveal that ***in vivo*** apparent low efficacy of SR-17018 is similar to that of a MOP ***partial*** agonist buprenorphine measured by the non-human primate assays with translation relevance. Such a low intrinsic efficacy explains its improved side-effect profile of a highly G protein-biased MOP agonist, SR-17018, in primates.