

9th Annual Midwest/Great Lakes Undergraduate Research Symposium



September 30, 2017



Ohio Wesleyan University

Delaware, OH

Welcome!

We would like to extend a special “thank you” to all of the students, faculty, and schools participating in today’s event!



The University of Vermont



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Schedule

9:00 – 10:00am	Breakfast & Registration Merrick Hall, 1 st Floor
10:00 – 11:00am	Keynote Address: Dr. Robert West <i>"Adventures of the Medial Frontal Cortex: Personal Responsibility, Ethical Decision Making, and Self-Control"</i> Merrick Hall, 3 rd Floor
11:00 – 11:30am	Coffee Break Merrick Hall, 1 st Floor
11:30am – 12:15pm	Student Oral Presentations Merrick Hall, 3 rd Floor
12:15 – 1:15pm	Lunch Merrick Hall, 1 st & 2 nd Floors
1:30 – 3:30pm	Poster Session <i>All posters displayed throughout session. 1:30-2:30 Even-numbered posters presented 2:30-3:30 Odd-numbered posters presented</i> Science Center Atrium
3:30 – 4:30pm	Breakout Sessions Student Session: <i>Panel on Graduate Programs and Alternative Careers</i> Merrick Hall, 3 rd Floor Faculty Session: <i>What makes a good First Course in Neuroscience?</i> Merrick Hall, 2 nd Floor
4:45 – 5:00pm	Closing Remarks & Awards Merrick Hall, 3 rd Floor

Keynote

Robert West, Ph.D.

Elizabeth P. Allen Distinguished Professor
Department of Psychology & Neuroscience
DePauw University

Adventures of the Medial Frontal Cortex: Personal Responsibility, Ethical Decision Making, and Self-Control

The anterior cingulate cortex (ACC) is an intriguing neural structure that is widely involved in cognitive, social, and affective information processing. As examples, the ACC is active when we experience physical or social pain and when we observe someone else experiencing pain; when we experience negative outcomes, such as the loss of money or points in a game; and when there is conflict between our intentions and actions. In the talk, I will consider the results of studies using event-related brain potentials (ERPs) to explore the temporal dynamics (i.e., time course) of neural activity in the ACC and related brain structures in three domains including gambling, ethical decision making, and self-control. The results of these studies demonstrate that self-regulation across these domains arises from transient activity in the ACC followed by more sustained activity in the lateral frontal, parietal, and occipital cortices. Based upon these data, I propose that the pattern of transient ACC followed by sustained activity in other cortical structures represents a general property of neural interactions that cut across cognitive, social, and affective information processing, and that facilitate goal directed behavior across domains.

Career Panelists

Kristen Ambegaokar, Ph.D.

Medical Writer & Scientific Consultant
The Navicor Group

Mark Baccei, Ph.D.

Associate Professor of Anesthesiology
Director, Neuroscience Graduate Program
University of Cincinnati College of Medicine

Clare Edwards, M.P.H., C.P.H.

Community Education Supervisor
SourcePoint

Zach Ford

Ph.D. Graduate Student
Neuroscience Graduate Program
University of Cincinnati College of Medicine

Nwabi Makola

M.D./Ph.D. Graduate Student
University of Cincinnati College of Medicine

Jazmine Quinn

Research Specialist
Applied Genomics & Biology Division
Battelle Memorial Institute

Presentation & Poster Abstracts

Author Index can be found on page 53.

Robert D. White, Bridget Dames, Jack Frankel, Austin Nader, Olivia Ramsey, Drew Schleicher, Joshua Setters and Anna K. Radke.

Miami University

Striatal control of aversion resistant alcohol drinking in male and female mice

One characteristic of alcohol use disorder is persistent seeking and taking of alcohol despite negative consequences. This “compulsive-like,” “aversion-resistant,” or “inflexible” drinking is often modeled in rodents by adding the bitter tastant quinine to alcohol (Lesscher and Vanderschuren 2012). While this approach is commonly employed in both rats and mice, there have been few examinations of aversion-resistant alcohol drinking in female animals. This gap in the literature persists despite the fact that both human and animals studies suggest females are more vulnerable to addictive behaviors, including initiation, escalation, and relapse of drug-seeking (Anker and Carroll 2010). Here, we investigated aversion-resistant alcohol-drinking in male and female C57BL/6J mice. We used a version of the “drinking in the dark” procedure to model binge-like drinking (15% ethanol vs. water for 2 hours, 3 hours into start of the dark cycle) and tested for aversion-resistant drinking by adding quinine (100 or 250 μ M) to the ethanol solution (EtOH+Q). We also assessed the role of the nucleus accumbens (NAc) in aversion-resistant drinking with chemogenetic inhibition. The DREADD vector AAV-hSyn-hM4D(Gi)-mCherry (ADDGENE) was expressed in the NAc and mice were tested for aversion-resistant drinking following injections of the DREADD ligand clozapine-n-oxide (CNO, 1 mg/kg i.p.) or vehicle. Our results demonstrate increased alcohol consumption and escalation of drinking in female vs. male mice. There were no sex differences in consumption of EtOH+Q. Finally, NAc inhibition altered consumption of EtOH and EtOH+Q. Together, our results provide insight into the neural mechanisms of alcohol use despite negative consequences. Research supported by the Department of Psychology and College of Arts and Sciences at Miami University.

Emily Budde, Robert West and Kaitlyn Malley.

DePauw University

Neural Correlates of Ethical Decision Making: Why Do We Do Bad Things?

In today's world, information security has become a critical concern for governments, corporations, and private citizens. Roughly 50% of all breaches of information security come from individuals within the organization that is the target of cybercrime, and research reveals that current deterrence methods are generally ineffective in thwarting breaches of information security. The factors that give rise to violations of information security are not well understood, with some evidence indicating that individual differences in self-control and moral beliefs may moderate the likelihood of individuals committing a violation of information security. In addition, very little is known about the neural mechanisms underlying decision making processes in the context of information security. Given these limitations, our laboratory have been using event-related brain potentials (ERPs) to examine the neural correlates of decision making processes related to information security in the information security paradigm (i.e., IS paradigm). In this task there are 3 types of scenarios (i.e., control, minor, and major violations). Control scenarios involve decisions without an ethical consideration, while minor and major violation scenarios involve decisions that include an ethical component that varies in severity. In the current study we sought replicate and extend our previous research with this task by having individuals complete the IS paradigm while ERPs were recorded in addition to measures of self-control and moral potency. The behavioral data replicated previous results, with individuals being less likely to endorse ethical violations than control scenarios. The electrophysiological data revealed widespread ERP activity that differentiated control from minor and major scenarios beginning around 200 ms after stimulus onset. At 215 ms this reflected transient activity in fusiform gyrus, followed by sustained activity in right inferior frontal cortex, and anterior temporal cortex. For the right frontal region, this sustained ERP activity revealed a positive correlation with moral potency and negative correlations with impulsivity and risk taking. The comparison of major versus minor violations revealed activity in the left anterior frontal cortex and medial subcortical structures possibly including the posterior cingulate and thalamus between 400-800 ms. This ERP activity revealed a negative correlation with moral potency and positive correlations with impulsivity and risk taking. Together, our data reveal transient and sustained ERP activity related to decision making in the context of information security. This ERP activity may arise from neural structures that are commonly associated with moral reasoning, and is differentially related to moral potency and aspects of self-control, constructs known to be important in predicting violations of information security.

Anoohya Muppirala and Sarah C. Petersen

Department of Neuroscience, Kenyon College

A Novel zebrafish Mutant reveals reduced axon count in the CNS and hypomyelination in the PNS

Myelin is a critical component of proper neuronal function in vertebrate organisms. Produced by oligodendrocytes in the CNS and Schwann cells in the PNS, myelin wraps the axons of neurons, thus forming the myelin sheath in order to enhance signal propagation. However, damage to the myelin sheath can result in many debilitating neuropathies in humans, such as Multiple Sclerosis or Charcot-Marie tooth disease. Therefore, it is essential to investigate the underlying genetic mechanisms that govern glia development and myelination in order to advance our understanding of demyelinating conditions.

A large scale forward genetic screen conducted in zebrafish originally identified *stl93* as a CNS hypomyelin mutant. However, TEM analysis of the mutant has since revealed reduced axon count in the CNS and hypomyelination in the PNS. Because the function of oligodendrocytes and Schwann cells both depend on multiple neuronal factors, this combined phenotype suggests that the causative gene may regulate neuronal function. Subsequent whole-genome-sequencing analysis identified four candidate genes, three of which are indeed implicated in neuronal processes such as retrograde transport and signaling. Therefore, we hypothesize that impaired neuronal activity may indirectly influence the behavior of the surrounding myelinating glia, resulting in a novel CNS and PNS phenotype.

Central Michigan University

*Role of Inflammation and Gender on the Onset and Progression of
Alzheimer's Disease*

Accounting for 60-80 percent of all cases, Alzheimer's disease (AD) is the most prevalent form of dementia. Additionally, two thirds of the individuals affected with AD are females. Although there is currently no cure for AD, treatments are available to slow dementia symptoms and increase overall quality of life. The race for a cure continues as the prevalence of AD continues to rise. In our study, we will be using the NL-G-F mouse model of AD and crossing these mice with (neuronal) green fluorescent protein (GFP) transgenic animals to examine chronic inflammation in male and female mice. The NL-G-F model contains three mutations; Swedish, Arctic, and Beyreuther/Iberian (Saito et al 2014). Crossing the NL-G-F mice with GFP allows for examination of dendritic spine density and ramification of neurons in the hippocampus and cortex using 3D analysis software. We hypothesize that if chronic inflammation is induced at 12 weeks of age for six weeks, then by four months of age the females will accumulate overall more microglial cells and amyloid beta plaques compared to the males. Our hypothesis will be tested using weekly intraperitoneal injection of lipopolysaccharide (LPS) for 6 weeks. We will examine the extent of memory deficit in the mice and observe how the neuroinflammation affects the physiological parameters of AD progression in regard to genders. One week prior to the end of injections the mice will be tested using the six-arm radial water maze and the Y maze. At four months of age the mice will be euthanized with 4% paraformaldehyde (PFA) and their brains will be harvested for analysis. The brain tissue will be analyzed using immunohistochemistry and unbiased stereology to count amyloid beta ($A\beta$) plaques (4G8) and microglia (Iba1). Our numbers will be focused on the hippocampus and cortex as they are two of the primarily affected brain regions in AD. ELISA and Western Blots will be utilized for cytokine production in the peripheral and central nervous system examining pro-inflammatory and anti-inflammatory cytokines. We expect the mice with LPS doses to have a higher number of $A\beta$ plaques and microglia. We also predict these mice will exhibit more drastic changes in behavior. The goal of this study is to confirm the role of neuroinflammation as a potential risk factor in AD progression regarding gender. This study also has the potential to prevent or delay AD by timely targeting the disease's detrimental inflammatory process.

Work Cited:

Saito, T., Matsuba, Y., Mihira, N., Takano, J., Nilsson, P., Itohara, S.,... Saido, T. C. (2014). Single App knock-in mouse models of Alzheimer's disease. *Nature Neuroscience*, 17(5), 661-663.

¹ The College of Wooster

² Baylor College of Medicine

³ M.D. Anderson Cancer Center

Mechanisms of radiation therapy-induced neurocognitive impairments

Survivors of childhood brain cancer frequently experience negative changes in learning, memory, processing speed, and attention. Radiation therapy, the frontline treatment for brain cancer, is likely connected to these cognitive difficulties. Radiation is thought to cause changes at the level of the synapse, specifically at dendritic spines. Spines are structural representatives of synapses, and changes in them are tightly coordinated with changes in synapse size and strength. Decreases in dendritic spine density have been seen at 1 week, 10 days, and 30 days after exposure to radiation (Parihar, et al., 2013). In order to explain this long-term effect, our present research is looking at the mechanisms underlying it by examining acute effects of radiation injury.

Previously, we examined this in vivo, using transgenic YFP mice. Some mice received memantine, a NMDA receptor antagonist. Thirty minutes after exposure to 10 Gy radiation there was an increase in mouse hippocampal dendritic spine density. Interestingly, these were only significant in female mice, and memantine blocked these effects, indicating radiation injury may be sex-dependent and mediated through glutamate and NMDA receptor signaling.

Then, we used a Förster Resonance Energy Transfer (FRET) assay and fluorescence imaging to examine GTPase activation in acute radiation injury. We examined Rho family GTPases, whose activity was exhibited by the colors seen during imaging. Current, preliminary results indicate an increase in RhoA and a decrease in Rac1 GTPase activity one hour post-radiation, and a transient increase in the diameter of dendritic spines. Future experiments will focus on repeating this assay and obtaining more imaging data before the interpretation of results.

¹ Ohio State University

² Ecole Polytechnique Federale de Lausanne

Blood-Brain Barrier Gene Expression is Altered by Aging, Alzheimer's Disease, and Chemically-Induced Seizures

The functionality of the blood-brain barrier (BBB) is impaired in aging and Alzheimer's disease (AD). The two-hit vascular hypothesis proposes that BBB dysfunction combined with reduced cerebral blood flow trigger amyloid- β (A β) accumulation in the brain. We investigated whether age- and AD-associated BBB dysfunction is associated with changes to gene expression in the BBB. Total hippocampal RNA was extracted from wild type and APP/PS1 mice aged 3-18 months, and expression was analyzed by qRT-PCR. We observed enhanced inflammatory activation of endothelial cells with aging and APP/PS1 genotype, as evidenced by increased transcription of endothelin-1, vascular adhesion molecule-1, and inducible adhesion molecule-1, as well as reduced expression of nitric oxide synthase (NOS)-2. Expression of several A β efflux transporters decreased with normal aging. In the 18-month age group, APP/PS1 mice had increased expression of A β influx and efflux transporters compared to age-matched controls. Since neurons release A β following periods of activity, we investigated whether inducing seizures by intraperitoneal kainic acid (KA) injection would alter BBB gene expression. The KA-injected mice had reduced expression of advanced glycosylation end-product receptor (AGER), which is the primary influx transporter for A β , and increased NOS-3 expression. Our results lend support to the two-hit vascular hypothesis by showing that aging and AD could disrupt A β clearance through dysregulated transcription of BBB transporters and inflammatory modulators. Additionally, our KA experiments suggest neuronal activity can modulate BBB gene expression to influence A β clearance. Thus, the BBB may represent a promising therapeutic target for early intervention in AD.

Kenyon College

Characterization of a zebrafish peripheral myelin mutant

Myelin is the protective, fatty acid sheath that surrounds axons and allows for efficient electrical signaling throughout the central and peripheral nervous systems. While it is known that myelin is produced by oligodendrocytes in the CNS and Schwann cells in the PNS, the genetic and molecular mechanisms that regulate myelination are not fully understood. Illuminating these mechanisms may help researchers provide more effective therapeutics to currently incurable myelopathies such as Multiple Sclerosis and Charcot-Marie Tooth disease. A zebrafish forward genetic screen conducted at Washington University, St. Louis revealed a myelin mutant with reduced terminal Schwann cell differentiation in the peripheral nervous system. Whole-genome sequencing analysis of this mutant – referred to as *stl144* – revealed an autosomal recessive inheritance pattern and linkage to chromosome 8. A list of candidate genes was generated using whole-genome mapping data and heterozygous adult carriers of the mutation were identified using in situ hybridization staining of myelin basic protein (mbp) in embryos. These findings set the framework for further characterization of the *stl144* mutation, with the goal of identifying the causative gene and broadening our understanding of the genetic mechanisms that lead to myelination in the PNS.

Camille Hanes,¹ Binta Jalloh,² Chris Rounds,² Ken Moberg,² and Seth Kelly.¹



¹ The College of Wooster

² Emory University

Investigating the Potential of CaMKII to Rescue Lethality in dNab2 Homozygous Mutant Drosophila melanogaster

Intellectual disability (ID) is a neurodevelopmental disorder characterized by impaired intellectual functioning. Affecting around 1-3% of the population, patients with ID exhibit a lower IQ, as well as the inability to demonstrate adaptive behavior. Although intellectual disabilities vary in causative factors, loss of the ZC3H14 gene in humans has been linked to a nonsyndromic form of autosomal recessive intellectual disability. ZC3H14 encodes the Cys3His (CCCH) tandem zinc finger polyadenosine RNA binding protein. The post-transcriptional role of ZC3H14 involves polyadenylation of the mRNA transcript and regulation of poly(A) tail length. However, the target mRNAs of ZC3H14, as well as the overall role of ZC3H14 in gene expression remain unknown. Preliminary data suggests that ZC3H14 may bind to CaMKII mRNA and regulate its translation. Interestingly, CaMKII is a kinase prevalent in the brain and is critical for calcium signaling and learning and memory. To further study the function of ZC3H14 and its potential interaction with CaMKII, a *Drosophila melanogaster* model has been established using the ZC3H14 *Drosophila* ortholog, dNab2. Neuronal levels of CaMKII and dNab2 protein were quantified and rescue experiments were performed. The elevated CaMKII level in flies lacking dNab2 suggests an interaction does exist between these two genes and further supports the model that ZC3H14 regulates levels of CaMKII protein. Further studies investigating this potential interaction will offer insight into the underlying cellular and molecular mechanisms of human ID.

The Center for Brain and Spinal Cord Repair, The Ohio State University

Spinal Cord Injury Causes Chronic Liver Pathology in Mice

The typical goal of spinal cord injury (SCI) research is returning motor or sensory function. Less attention has been given to spinal regulation of visceral function via autonomic circuits after SCI. SCI disrupts the autonomic connections between the central nervous system and many peripheral organs. These altered connections cause chronic health complications that can result in significantly shorter life spans for SCI patients compared to the general population. One of these major health issues is metabolic dysfunction, and an organ critical for metabolic control is the liver. Studies from our lab have shown that a midthoracic contusion SCI in rats results in chronic liver inflammation and fat deposition, both symptoms of non-alcoholic fatty liver disease (NAFLD), a sign of metabolic syndrome in the liver. It is unknown if similar liver pathology occurs after SCI in mice. Moving into the mouse SCI model will provide us with more tools not widely available in the rat, such as transgenic mice with cell-specific alterations, that will help us further understand the mechanisms that drive the liver-spinal interactions after SCI. Thus, the purpose of this study was to investigate inflammatory and fatty changes to the mouse liver after SCI. We employed a moderate, midthoracic contusion SCI model and characterized the expression of liver Kupffer cell markers CD11b and CD68, liver fibrosis markers GFAP and PDGFRbeta, and liver fat deposition via Oil Red O histology across a span of acute and chronic time points. Liver CD68 expression on phagocytic Kupffer cells was significantly reduced at 18 days post injury (dpi) and then significantly increased at 180 dpi, compared to naïve levels. Conversely, liver CD11b expression on cytokine-producing Kupffer cells was significantly increased at 28 dpi and then returned to naïve levels by 180 dpi. Interestingly, GFAP and PDGFRbeta levels peaked at 18 dpi and returned to naïve levels by 180 dpi. Finally, Oil Red O+ fatty droplet staining was significantly increased at 28 dpi and remained elevated at 180 dpi. These results provide novel evidence that SCI causes chronic liver pathology in the mouse. This work provides us with a foundation for creating novel strategies that limit hepatic pathology after SCI and that improve the metabolic profile and overall recovery of SCI patients chronically.

Austin Waddell, Tomas Barrett, John Gallien, and Kevin Park.



Central Michigan University

The Effects of Early Neuronal Cell Cycle Dysregulation on Autism Spectrum Disorder Related Brain Morphology

Autism Spectrum Disorders (ASD) are common heterogeneous neurodevelopmental disorders preferentially affecting males. ASD is a group of neurodevelopmental abnormalities clinically characterized by impaired social interaction, communication, and repetitive stereotypic behavior. Phenotypic and etiological heterogeneity has made the genetic and cellular pathophysiology difficult to uncover. Despite a growing number of genetic studies linking dysregulated cell cycle and cell proliferation gene networks to those typically associated with ASD, the direct effects of prenatal cell cycle disruption on ASD phenotypes has not been explored. In this study, we directly examined the effects of early neuronal cell cycle dysregulation on brain morphology and ASD related behavior using our transgenic mouse model of cell cycle dysregulation. An analysis of brain weight showed that early neuronal cell cycle disruption led to a reduced brain weight (0.406g \pm 0.02g) compared to controls (0.427g \pm 0.02g). Behavioral analysis which included social interaction tests and grooming behavior observation indicated that cell cycle dysregulation leads to reduced sociability as well as increased grooming behavior representing repetitive stereotypic behavior in male mice but not females. These findings mirror stereotypical behavior and brain size changes observed in autism patients.

DePauw University

Impaired Attention in Patients with Early Phase Psychosis

The Human Connectome Project (HCP) initiated in 2009 was designed to map the structural and functional networks within and across the human brain. Stemming from this project, the HCP for Early Psychosis (HCP-EP) has focused on utilizing a multi-site clinical network in order to conduct diffusion and functional connectivity analyses to compare affective and non-affective psychosis (National Institutes of Health and Northwestern University, 2015).

Previous studies support the idea that the frontoparietal cortex plays a key role in attention cues that attend to spatial and feature priority (Liu & Hou, 2013). In addition, researchers discovered patients with both affective and non-affective psychosis have disruptions across networks with preferential reductions in functional connectivity within the frontoparietal network (Baker, Holmes, & Masters, 2014). HCP-EP researchers therefore hypothesized that fMRI scans will show frontoparietal network abnormalities in patients with non-affective and affective psychosis, while limbic abnormalities will be specific to affective psychosis, and ventral abnormalities will be specific to patients with non-affective psychosis.

While the images for HCP-EP have not yet been fully collected or analyzed, we believe that a cognitive assessment, the NIH Toolbox Flanker Inhibitory Control and Attention Test (NIH TFICA) will elucidate disruptions in attention for patients with psychosis, which could ultimately assist in determining if abnormalities within the frontoparietal network will be detected via neuroimaging (Slotkin et al., 2012). Specifically, we hypothesized that patients with non-affective psychosis would have worse performance than healthy controls on the NIH TFICA.

In the present study, data from one of the HCP-EP clinical sites, Indiana University Psychotic Disorders Program (IUPDP), was collected on eighteen subjects, including controls (N = 7) and patients with non-affective psychosis (N = 11).

Each subject participated in three study visits: one to confirm diagnosis or participation as a control, a cognitive assessment visit, and an fMRI-scanning visit. For the present project, only demographic information and a single paradigm of data collected during the testing visit was used: the NIH TFICA (Slotkin et al., 2012). The results indicated that patients with non-affective psychosis perform worse than healthy controls on attention tasks, which implicate the frontoparietal cortex. Further, these findings are consistent with the hypothesis that the fMRI images will show abnormalities within the frontoparietal cortex.

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Antibody Binding to Tau from an Alzheimer's Brain Predicts Therapeutic Efficacy

Alzheimer's disease has two major pathological hallmarks, amyloid- β plaques, and neurofibrillary tangles composed of tau. The tau protein is a microtubule-stabilizing protein that is primarily found in neurons. In Alzheimer's disease and related tauopathies, tau undergoes post-translational modifications, and forms intracellular paired helical filaments (PHF). Clearing pathological tau is one of the goals of therapeutic development, and immunotherapy is a promising approach. In this investigation, we characterized a novel monoclonal tau antibody, examining its binding, uptake and efficacy in preventing PHF induced pathology.

In AD brain, there are multiple forms of soluble tau monomers, small solubilized aggregates, and larger insoluble filaments. The ability of the antibody to bind to different forms of tau was assessed using two different ELISA assays. In the first, plates were coated with tau from each fraction, and the binding to each was measured. In the second, the antibody was pre-incubated with solubilized aggregates prior to plating. The tau antibody showed similar levels of binding to each of the fractions when added alone. However, when it was pre-incubated with solubilized PHF, binding was significantly reduced, indicating that it preferentially binds to soluble tau species.

Uptake and efficacy experiments were conducted in primary neurons cultured from newborn transgenic mice expressing human tau. Uptake of the antibody into the cells was evaluated using confocal imaging and western blotting. Using both methods, we observed that the antibody was readily taken up by neurons.

To assess efficacy, cells were incubated with 10 mg/ml human derived PHF tau, and 1 mg/ml of the antibody in one of three dosing methods, PHF alone, PHF and antibody added together (PHF + Ab), or PHF followed 24 hours later by antibody (PHF \rightarrow Ab). Samples were collected at 1 and 7 days. Toxicity was assessed using LDH levels. PHF alone significantly increased LDH when compared to the control at both time points ($p \leq 0.0001$). At day 1 PHF + Ab cells had significantly lower LDH levels compared to PHF alone ($p \leq 0.01$), while at day 7 both groups were significantly lower (LDH $p \leq 0.01, 0.05$). Similar results were seen when total tau levels were examined. Significantly lower tau levels compared

to PHF alone were seen in antibody treated cells in the PHF → Ab group at day 1 ($p \leq 0.01$), and both groups were significantly lower at day 7 ($p \leq 0.0001$).

Overall, the tau monoclonal antibody reduced the toxicity and seeding of tau pathology. It primarily bound soluble tau, associated with tau inside neurons, and was effective in both dosing conditions, indicating that it works both extra- and intracellularly. Understanding how these factors affect efficacy will be helpful in the development of immunotherapies for Alzheimer's disease and related tauopathies.

Wabash College

*Characterizing Spatial and Response Navigation in Rats in a Novel Maze:
Effects of Hippocampal Lesions*

A previous study by Packard and McGaugh (1996) examined the use of spatial and egocentric navigation strategies by training rats to obtain a food reward in a plus-shaped maze. With one arm closed off to form a T-shaped maze, rats were trained to find food placed in one arm of the T. During the training phase, the rat began and retrieved food from the same locations. On a probe trial, rats were placed in the arm opposite to the starting location. If the rat could accurately locate the reward, it was demonstrating a place strategy. Conversely, if the rat turned in the same direction as it did in the training phase, it was navigating via a response strategy. However, the experimental paradigm used by Packard and McGaugh may have its limitations. When conducting a probe trial in a plus-shaped maze, it is difficult to confirm that animals are relying specifically on either a place or response strategy, as any choice made by the animal, even a random search, counts as a strategy: when approaching the food reward, rats were only able to turn in two directions. As a result, the rats have no other choice than to exhibit either a response or place strategy. In the present study, we introduced a modified maze design that included other areas where the rat could travel during a probe trial, using a symmetrical maze with four starting locations and four goal locations. Using this new maze configuration, we aimed to replicate the findings of Packard and McGaugh. We hypothesized that rats will adopt a place strategy, and transition to a response strategy after extended training. We expected also that two rats with excitotoxic lesions of the hippocampus would show a deficit in the use of a place strategy. We also will examine other behavioral measures (reaction time, latency to reach the food reward, and consistency of the path taken to the reward) to identify other measures (beyond accuracy) that identify the use of place and response strategies. We expect that rats using a place strategy to take significantly longer reaction time, and will take a more variable path to the food reward.

Eastern Michigan University

Locating Brain-Lipid Binding Protein in the Mouse Olfactory Bulb

The mouse olfactory system has demonstrated plasticity when faced with environmental or physiological changes. One type of plasticity, is the generation of new sensory axons which are guided by olfactory ensheathing cells (OECs), a type of glia, to the olfactory bulb during neurogenesis. OECs are apparent in the olfactory nerve layer, but there has been discrepancy as to whether these glial can be found in the glomeruli neuropil layer. In our experiment, we located the OECs using a mouse-anti-brain-lipid binding protein (BLBP) antibody, a type of glial marker. BLBP was prevalent in the nerve layer as expected, but was also found in the glomeruli neuropil. This leads us to an unanswered question about pan-neurotrophin receptor 75 (p75NTR). Could p75NTR be expressed on a subtype of OECs in the glomeruli neuropil? In previous experiments within our lab, we were unable to localize p75NTR to TH and GAD65 expressing interneurons, olfactory sensory neurons, or GFAP expressing glial cells. Further colocalization experiments labeling with both p75 and BLBP antibodies are underway to provide an adequate conclusion to what cell type p75NTR is expressed on. Studying the location of p75NTR and OECs within the mouse olfactory bulb can help us better understand the mechanisms of neurogenesis and plasticity in both the sensory and central nervous system. This knowledge could be used to further advance treatments for neurological diseases/disorders such as strokes, spinal cord injuries, Parkinson's, or Alzheimer's disease.

Cindy Huynh and Mollie Marshall.

Ohio Wesleyan University



Look into the Effects of Maternal Methamphetamine Use and Psychiatric Disorders on Infant Mental Health.

Substance use is one of many risk factors that contribute to their infant's development, along with maternal depression, parental psychopathology, and the external environment. Women of childbearing age in the United States and New Zealand are among the increasing number of people who abuse methamphetamines (MA). Mothers who abuse MA are often also depressed, which is related to poor parenting behaviors and linked to changes in infant social behavior and temperament, a biologically based set of behavioral tendencies that influence how the infant will approach, respond to, and interact with the social world. Research has shown parental depression negatively impacts cognitive, motor, emotional, and social development. The current study further examines how a mother's history of substance abuse and/or psychopathology may impact infant health, temperament, and development. The study analyzes questionnaires and assessments from 234 mother- infant pairs in the NZ Infant Development, Environment, and Lifestyle Study. Of this sample, 106 mothers reported MA use during pregnancy and 115 denied MA use during pregnancy. Their substance use was compared with maternal report questionnaires—Brief Symptom Inventory, Beck Depression Inventory, and Infant Behavior Questionnaire—which measured maternal psychopathology, depression, and infant temperament. In addition, the results of the NICU Network Neurobehavioral Scale, Bayley Scales of Infant Development, and Strange Situation assessments were compared to standardized scales to determine infant behavior and attachment. The results from the maternal questionnaires were compared to the infant assessments to ascertain the effects of maternal substance abuse and/or psychopathology on infant mental health. The study points to the importance of early intervention and enhancing protective factors in multi-risk families.

Ohio University

Examining cerebellar development in Rbpj-related brain arteriovenous malformation

Arteriovenous malformations (AVM) are abnormalities of vascular organization, specifically in vessel connections. A defining feature of an AVM is AV shunting – delivery of blood through direct connections between arteries and veins, rather than through normal capillary networks. Blood flows rapidly through these abnormal connections; thus, these vessels are prone to rupture and can cause significant damage. AVMs can form throughout the body, but specifically, AVMs in the brain can have devastating outcomes. It has been previously shown that endothelial Notch signaling is involved in postnatal arteriovenous organization, and disruption of Notch signaling can result in the formation of AVM-like features in mice. We used an inducible Cre-loxP system to delete endothelial *Rbpj*, which is necessary for canonical Notch signaling, to model brain AVMs in mice. Deletion of endothelial *Rbpj* at birth leads to cerebellum defects by two weeks post-deletion; however, the exact cellular changes that occur are not fully understood. During this early postnatal period, granule cell migration and differentiation is vital for the formation of cerebellar cortical layers. Granule cell precursors exist in the external granule cell layer (EGL) and produce postmitotic granule cells that will migrate to the internal granule layer (IGL). To determine if granule cell migration was affected, we analyzed IGL granule cell density and EGL thickness. We hypothesized that granule cell migration would be affected when endothelial *Rbpj* was deleted. IGL granule cell density was unaffected when examining the whole cerebellum. After analyzing the lobules separately, we found that only the IGL granule cell density of lobule VII was significantly reduced. Additionally, cerebellar EGL thickness from whole cerebellum was not affected. Although, when examining the lobules separately, it was discovered that the EGL thickness of the distal region of lobule III was significantly increased. Interestingly, in four of six mutants, we found the EGLs of some adjacent lobules to be fused. Fusions were defined as two EGLs with no visible separation between granule cells of adjacent lobules. Our data suggest that, in the postnatal cerebellum, endothelial *Rbpj* is regionally required for proper IGL and EGL morphogenesis, and that endothelial *Rbpj* is necessary for the proper formation of distinct EGLs between lobules.

Anthony Maggard,¹ Vera Valakh,² and Sacha Nelson.²



¹ Earlham College

² Brandeis University

Persistent or Alternate Activation: Characterizing intrinsically excitable neuronal sub-population

Concerning homeostatic regulation of synapses, transcriptional responses to activity changes occur to incorporate individual strength differences of synapses while also maintaining network integrity. To further characterize these homeostatic properties, we observed how global silencing impacted activity-dependent transcription factors c-Fos and PhosphoCREB in the mammalian cortex via microscopic analysis of immunofluorescence. We found a sub-population of cortical excitatory cells that exhibit shifting markers for activity while network activity is silenced, indicating a means of alternative or persistent activation in such cells. The findings suggest these cells may function with some intrinsic properties to promote network integrity.

Abduselam Awol, Olivia Wallace, and Michelle Tong.



Earlham College

Effects of Odor Similarity on the Learning and Memory of Olfactory Discrimination

Olfactory discrimination is the ability to differentiate between odors. In this study, we examined the effects of odor similarity on the ability to both learn to discriminate between odors, and to form associative long-term memories (LTM) between an odor and reward. The effects of similar and dissimilar odors on olfactory memory and learning were examined with a specific focus on the olfactory function of discrimination through mice trials. All mice were conditioned to associate a reward (sugar pellet) with one of two odors, and were trained to discriminate between either two similar odors or two dissimilar odors. We compared the learning rate and 24-hour LTM between mice trained on the similar and dissimilar odors. We found that the mice experienced greater difficulty while trying to locate the pellets when presented with similar odors than dissimilar odors. These findings suggest that the mice had a higher learning rate for the dissimilar odors.

Kenyon College

Chemical screening in zebrafish to reveal small molecules that regulate myelination

Myelin is a multilayered sheath that incases axons to promote efficient conduction of action potentials and proper functioning of the nervous system. In vertebrates, Oligodendrocytes produce myelin in the central nervous system, and Schwann cells produce myelin in the peripheral nervous system. This distinction of cell types is evolutionarily conserved in zebrafish, which makes them an excellent model organism for studying the development of myelin. Gpr126 mutants, found in a forward genetic screen, are unable to correctly myelinate their PNS. The adhesion G-protein coupled receptor gpr126 is crucial for myelination of the peripheral nervous system (PNS). Phenotypically, this results in poor signaling through the peripheral nervous system. In a drug screen, we treated gpr126 mutant zebrafish with a 1600 pharmacophore library of small molecules during the critical period in their development for PNS myelination. Of those 1600 drugs, we found four that recovered myelination to some extent by analyzing myelin basic protein mRNA expression along their peripheral nervous system. We want to uncover the pathway by which each of those drugs recovers myelination. Our current efforts are focused on apomorphine hydrochloride, a dopamine agonist. Recently, we found that high concentrations of apomorphine can recover myelination in hypomorphic mutant zebrafish. Hypomorphic mutants have a fully intact GPR126; in contrast, amorphic mutants have a completely non-functional GPR126. Phenotypically, this means that hypomorphic mutants can have weak myelination, while amorphic mutants have no myelination. Going forward, we want to treat both hypomorphic and amorphic mutants with apomorphine hydrochloride. We hypothesize that an intact GPR126 is necessary for apomorphine hydrochloride to be effective, meaning that apomorphine is acting directly on the known pathway for PNS myelination.

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Neuroscience Department, Oberlin College

Detecting Oxidative Stress in Zebrafish Embryos

Autism Spectrum Disorder (ASD) is a category of diagnoses denoting cognitive, social, and behavioral abnormalities. Autism affects 1 out of every 68 children in the United States and is considered a neurodevelopmental disorder, mainly detected in childhood. The molecular mechanisms underlying ASD are not well known. Genetic analyses have recently associated oxidative stress pathways with ASD. To test oxidative stress in the context of neurodevelopment, we developed an assay to reliably measure levels of oxidative stress in zebrafish embryos. Oxidative stress was measured using a fluorescent dye, H2DCFDA, which fluoresces only after reacting with reactive oxygen species. Embryos were treated with the dye and then homogenized into lysates by sonication; oxidative stress in the samples were then quantified using a fluorescence plate reader. We optimized the assay for a number of variables, including dye concentration, time of exposure, and embryos per sample. The assay demonstrates a positive linear relationships between fluorescence output and dye concentration. This assay can assess the oxidative stress pathway in neurodevelopment *in vivo*, including the effects of environmental and genetic factors associated with disorders such as ASD.

The effects of coloring mandalas on electroencephalography (EEG) asymmetry and self-reported mood

Coloring mandalas is touted as a mind-body technique that can relieve stress and elevate mood. We sought to test this claim by studying whether coloring mandalas would improve mood as indicated by self-report and greater electroencephalographic (EEG) left-hemispheric activity; a proposed global indicator of positive mood. Participants were randomly assigned to color a mandala template or engage in free form drawing, with both tasks lasting 20 minutes. Self-reported positive and negative affect were measured before and after the tasks, while EEG asymmetry was measured before, during and after the experiment. Due to muscle artifacts, it is difficult to measure EEG while participants engage in tasks that require movement. To address this challenge, we used the b alert EEG system and artifact rejection strategies to measure EEG while participants engaged in the tasks. As predicted, there was more left hemispheric activity during coloring as compared to during drawing. Contrary to our predictions, there were no differences in EEG asymmetry or self-reported mood post task. The overall pattern of results suggest that the positive effects of 20 minutes of coloring on mood, as measured by EEG asymmetry are manifest during coloring, but may quickly dissipate after task completion.

Oberlin College

Investigating environmental factors associated with autism spectrum disorder; effects of methylene chloride on neurodevelopment in zebrafish

Recent epidemiological research reports that increased exposure of pregnant mothers to methylene chloride correlates with increased prevalence of Autism Spectrum Disorder (ASD) in their children. Methylene chloride is a volatile organic compound commonly used as a solvent in paints, in the production of pharmaceuticals, and as a propellant for insecticides. However, it is unknown how this chemical impacts brain development or if it regulates known ASD physiology. The purpose of this research is to determine how methylene chloride affects neural development in zebrafish. Embryos were exposed on the day of fertilization to various concentrations of methylene chloride; body and brain morphology was observed via brightfield microscopy, and mRNA expression of genes associated with different cell types were measured using quantitative PCR. Transgenic reporter zebrafish lines were then used to further characterize neuron changes in terms of temporal and anatomical specificity via fluorescence microscopy. Initial results indicate methylene chloride exposure can (1) induce dose-dependent changes in overall development of zebrafish embryos, (2) downregulate gene expression of neuron cell markers, and (3) result in abnormal GABA neuron anatomy. Long-term, characterizing the molecular impact of methylene chloride on brain development may contribute to the improvement of ASD therapeutic treatments and inform data-driven environmental regulations.

Exploring relationships between electroencephalography (EEG) oscillations and empathy

By establishing relationships between resting electroencephalography (EEG) measures and psychological disorders, researchers may be able to develop diagnostic markers and objective measures of treatment outcomes. The aim of this research was to explore correlates between EEG slow wave/fast wave (SW/FW) ratios and empathy. While there are disagreements regarding the definition of empathy, most researchers agree that empathy is a multidimensional construct with both cognitive and affective components. Essentially, empathy involves affective and cognitive responses to another person's plight or situation. Importantly, empathy is related to emotional and moral development; with empathy deficits manifest in a number of psychological disorders and some criminal behavior. Historically, EEG SW/FW ratios were explored in relationship to attention. Our pilot study is part of a growing line of research extending the study of EEG SW/FW ratios to affect and personality. Relationships between baseline EEG SW/FW ratios and self-reported empathy were explored among eleven healthy female participants' ages 18 to 25 using the interpersonal reactivity scale to measure the cognitive and affective dimensions of empathy. Drawing from the theory that high SW/FW ratios reflect cortical under arousal resulting in limbic system dysregulation, we hypothesized that higher EEG SW/FW ratios would be linked to lower levels of both the cognitive and affective dimensions of empathy. Our hypotheses were partially supported. As predicted, higher EEG SW/FW ratios were correlated with lower levels of the affective dimension of empathy. A different pattern, inconsistent with our hypothesis, however emerged for the cognitive dimension of empathy. Specifically, we found that higher EEG SW/FW ratios were linked to higher levels of the cognitive dimension of empathy. While our hypotheses were only partially supported, the overall pattern of results from this pilot study support the link between EEG SW/FW ratios and affect.

Slow Wave Activity as a Marker of Proactive Control

The Dual Mechanisms of Control theory proposes that individuals flexibly alternate between two modes of control – reactive and proactive – based on current task demands (Braver, 2012). In imaging studies, reactive control is correlated with transient activity in the lateral prefrontal cortex (PFC) and the anterior cingulate cortex, while proactive control is associated with sustained activity in the lateral PFC. Research using event-related potentials (ERPs) has identified the frontal slow wave (FSW) as a neural marker of proactive control (Bailey, West, & Anderson, 2010; West & Bailey, 2012; West et al., 2012). The current study aimed to replicate and extend previous work by examining slow wave activity in three different tasks: the counting Stroop, N-back, and flanker tasks. Participants completed the three tasks while EEG was recorded. Stimulus and response-locked slow waves were present across all three tasks, further supporting its association with the implementation of proactive control.

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The Relationship between Productivity, Equality, and Wellbeing as Modulated by Social Dominance

The purpose of this study was to investigate whether the relationship between inequality and both productivity and wellbeing was modulated by social dominance orientation (SDO). Groups of 5 participants completed 60 trials of math problems for which they were rewarded with a certain piece rate per correctly solved problem. This piece rate changed every trial and it was shown to participants what piece rates the other players were earning before they were asked to solve as many or as little math problems as they wished. Previously, a questionnaire assessing their SDO was completed. Findings showed that participants scoring high on SDO, thus believing in systems of inequality, reported higher wellbeing and showed higher productivity than those with lower SDO scores. This implies that inequality is only beneficial for those believing in a system of inequality.

Oberlin College

Creating an Effective Set-Up for Assessing Larval Zebrafish Movement

Understanding the functional impact of environmental factors on neurodevelopment is an important but challenging topic to study. One prospective way to investigate these questions in vivo is to utilize model organisms useful for studying neurodevelopment, such as zebrafish. Analysis of larval zebrafish swimming patterns following embryonic exposure to neurotoxins may provide key insights into any associated functional changes in neurodevelopment and mirror effects of these toxins in human neurodevelopmental disorders. Here, we sought to implement a protocol for the tracking and analysis of larval zebrafish using established MATLAB programs, LSRtrack and LSRanalyze. By loading fish into multi-well plates and recording swimming behavior over a predetermined period of time, we can track fish movement and quantify neurobehavioral phenotypes. Additionally, the program records errors in its ability to track fish, such as its inability to detect fish in their respective wells or the presence of well walls. We report here the results of optimization experiments used to determine the most accurate and replicable set-up for movement recording and analysis. Variables such as imaging orientation (camera above vs. inverted), well solution volume, and video software filters were examined. Unexpectedly, imaging with the camera above the plate was problematic due to issues with solution meniscus and well wall visibility. An inverted set-up was found to be the best arrangement, where the camera rests at the base of a platform and captures the bottom of the well plate lit from above by a light box.

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Potential for glutamate excitotoxicity: The interplay between surgical stress and anesthetic exposure in a clinically relevant animal model

Background and significance: In an unprecedented move, the Food and Drug Administration will soon require a warning to be placed on the label of all commonly used anesthetic drugs stating that they may be neurotoxic in young children. Though a number of preclinical and clinical studies have investigated the potential neurotoxic effects of anesthetics, few have attempted to elucidate the mechanisms thereof. Most studies focus on the effects of anesthesia alone and overlook the contribution of surgical stress. Glutamate, the brain's primary excitatory neurotransmitter, is known to be involved in many different neuropathologic conditions when its activity is dysregulated. The goal of the present study is to evaluate hippocampal glutamate dysregulation in response to surgery as a potential mechanism of anesthetic neurotoxicity.

Central hypothesis: We hypothesize that surgical stress under sevoflurane anesthesia significantly increases extracellular glutamate concentration in the hippocampus, potentially leading to excitotoxicity. If present, glutamate dysregulation is a potential mechanism of neuronal apoptosis in the context anesthesia and/or surgery.

Materials and methods: Enzyme-based microelectrode arrays (MEAs) provide a direct, linear relationship between the amount of electrical current generated and the extracellular glutamate concentration. MEAs were calibrated *in vitro* to ensure fidelity and proper function. Piglets are well recognized as an excellent animal model for translational developmental neuroscience research. Seven neonatal piglets (3 - 4 days old, 1.5 - 3kg) were randomized to one of two experimental groups: Group A (3% or 1 MAC sevoflurane anesthesia alone, $n = 4$) and Group B (3% or 1 MAC sevoflurane anesthesia plus femoral osteotomy, $n = 3$) for three hours (clinically relevant exposure). An anesthesia workstation and infusion pump were used to monitor and maintain physiological homeostasis of piglets to prevent potential confounds (hypoxia, hypothermia, hypoglycemia) throughout experimentation. Following animal placement in a custom-designed stereotaxic apparatus, stereotaxic coordinates were used to create a craniotomy window (0.25 cm²) overlying the hippocampus. An MEA was then carefully lowered via

stereotaxic guidance into the hippocampus for glutamate measurements. Animals in Group A received no further intervention. After 30-minutes, piglets in Group B underwent a femoral osteotomy while continuous *in vivo* amperometric measurements of extracellular glutamate concentrations were recorded. After 210-minutes, all piglets were ethically sacrificed via transcardiac perfusion for verification of MEA placement and further investigations.

Results: The mean glutamate level in the surgical stress group ($11.14 \pm 0.03 \mu\text{M}$) was significantly higher than the mean glutamate level in the sevoflurane alone group ($5.99 \pm 0.02 \mu\text{M}$). A two-sample *t*-test for unequal variances generated a *p*-value < 0.001 . Surgical intervention correlates to a 1.8-fold increase in extracellular glutamate levels in the hippocampus under sevoflurane anesthesia.

Conclusions: MEA technology represents a novel methodology for the investigation of mechanisms of anesthesia-induced neurotoxicity (AIN). Our study demonstrated a significant and potentially excitotoxic increase in extracellular glutamate concentrations as a result of surgical stress and sevoflurane exposure. Exacerbated glutamate dysregulation along with MEA position verification inside the extracellular space of the hippocampus may be extremely valuable for long-term implantation investigations in awake, freely moving piglets.

Miami University

Oxytocin as a Potential Treatment for Gambling Disorders

Gambling disorders are characterized by an increase in risky behavior due to progressive loss of impulse control, even in the presence of negative financial and social consequences. Despite the overwhelming evidence of these disorders and their prevalence in society, few targeted pharmacological therapies exist to combat them, and current treatments are not widely effective. Oxytocin (OT) is a hormone implicated in social behaviors such as trust. This relationship suggests that OT will play a role in decision-making tasks, although this has yet to be established. The purpose of this study is to determine if oxytocin administration influences decision-making in rats using two operant paradigms: delayed and probabilistic discounting. We predict that enhanced oxytocin signaling will yield significant decreases in impulsivity across both behavioral contexts. One group of rats, who were to be run on these paradigms using a food reward, were implanted with a cannula targeting the lateral ventricle, allowing direct administration of the drug to the brain. OT is known to influence appetite control in rats, and so to control for this, a second cohort of rats was implanted with both a cannula targeting the lateral ventricle and a stimulating electrode targeting the Medial Forebrain Bundle (MFB). This stimulating electrode can be used to deliver rewards that are not food-dependent and do not require food-restriction, circumventing the confounds inherent to the food-based tasks. Prior to testing, all animals received intracerebroventricular infusions of either the OT agonist Carbetocin (0.1µg/5µl) or vehicle. Animals were then tested on a probabilistic discounting task in which they chose between a certain small reward or a risky large reward, which paid off at declining probabilities across days of testing. After completion, animals were tested on a delayed discounting task, in which the animal could have a small immediate reward or a large reward after an increasingly longer delay. Regardless of reinforcement type, animals that received Carbetocin exhibited no statistically significant difference in task performance when compared with control rats for either delayed or probabilistic discounting. Oxytocin does not appear to have a notable effect on impulsive decision-making, and thus may not be an effective treatment for gambling disorders. These results have important implications for both previous and future studies surrounding OT and decision-making.

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Eastern Michigan University

Development of a Cost-Effective Movement Assay for An Insect Sensory Neuroscience Lab

Cost effective and intuitive experiments for undergraduate neuroscience laboratory courses are not in use at many universities. There are several reasons for this including: time, cost, and ease of use. Thus, there is a basic conundrum in undergraduate laboratory equipment: good equipment can be cost prohibitive, while inexpensive equipment can be unreliable or not easy to use. Fortunately, new hardware companies have emerged to create inexpensive equipment that can be utilized in undergraduate neuroscience laboratory courses. Here we describe using such equipment to detect insect movement. Specifically, a Backyard Brains SpikerBox amplifier was paired with a piezoelectric and the signal was digitized and visualized using three different hardware-software combinations. The Backyard Brains amplifier can be connected directly into a smartphone. Then using a smartphone sound card, and the Spike Recorder phone app the voltage created by the piezoelectric is digitally converted and visualized. The Backyard Brains amplifier can also be connected directly to a computer through the sound card. The sound card converted the voltage and Audacity sound recording software was used for visualization. Lastly, the amplifier was connected to the teaching-grade iWorx TA data recorder. The iWorx TA converted the voltage and was then visualized using LabScribe software. The ability to use these three recording set-ups with one cheap piezo sensor means that we have found a cost-efficient and intuitive way to measure movement in an undergraduate neuroscience lab setting. Insects can be connected to the piezo sensor in a way that, once movement is performed, will manipulate the sensor into creating a voltage; thus, recording spontaneous movement. Sample recordings from bumblebee and cricket are presented. We are combining this setup with a homemade light board controlled via Arduino. The goal is to measure insect movement stimulated by different wavelengths and amplitudes. Thus, sensory perception and thresholds can easily be scientifically distinguished and measured. Future experiments will not be limited to light. Any number of sensory stimuli can be manipulated and reactions can be recorded.

Miami University

Influence of Reward Type on Impulsive Decision-Making

Decision-making is a crucial cognitive process that underlies almost every aspect of our daily lives. When presented with potential choices, we discount the value of a delayed or probabilistic reward, avoiding it when compared to immediate or certain rewards of equal inherent value. This decision-making process is known as discounting. Delay and probability discounting are known to be disrupted after drug-use and in a variety of psychiatric conditions, including ADHD, addiction, and gambling disorders. In rodents, these behaviors are typically assessed using food rewards; however, food reward is associated with numerous confounds, including satiety, and requires food-restriction for maximal effectiveness, leading to hunger-based brain changes. Alternative methods of reinforcement may avoid these confounds and provide greater clarity on the neurocognitive systems associated with decision-making. An alternative reinforcement method is brain stimulation reward (BSR), the direct stimulation of the reward pathway in the brain, thus eliminating the neurological and physiological confounds associated with food-reward. Additionally, BSR allows reward magnitude to be catered to each individual, compensating for natural variations in sensitivity to the reward. The purpose of this project is to evaluate the use of BSR in decision-making paradigms, comparing this new method of reward with the more traditional food reward. For this study, decision-making was assessed in two decision-making tasks using both food reward and BSR. Animals were first tested on a probabilistic discounting task in which they chose between a certain small reward or a risky large reward, which paid off at declining probabilities across days of testing. After completion, animals were tested on a delayed discounting task, in which the animal could have a small reward immediately or a large reward at an increasingly longer delay across days of testing. Our hypothesis was that BSR would result in improved behavioral performance on both tasks, as indicated by reduced behavioral variability and improved discounting. The results indicated that decision-making based on food reward had a similar pattern to BSR-motivated behavior, yet BSR was associated with preferences for larger reward-types later (slower discounting), and reduced behavioral variability. These results indicate that BSR may be an effective motivator of performance in delay and probability discounting tasks. Continued research on this concept will pave the way for more reliable and accurate research on decision-making in rodents. Such improvements in experimental designs will enhance the reliability and translational validity of rodent research on decision-making.

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BNST PAC1 Receptor Activation May Reinstates Cocaine Seeking Behavior in Rats

Nearly 2/3 of all patients relapse within three months of drug addiction treatment. Previous studies have demonstrated that in rats, stressors such as a footshock lead to the reinstatement of cocaine seeking. However, the behavioral and neural mechanisms that underlie this reinstatement are not well understood. Our previous work suggests that BNST PACAP-38 receptor activation is a possible mediator in stress induced reinstatement. However, we do not know which receptor mediates this response as PACAP-38 binds three receptor types PAC1, VPAC1 and VPAC2. To determine whether PAC1 activation might mediate reinstatement, in experiment 1, a PAC1 specific agonist (maxadilan) was infused bilaterally into the BNST after animals received 10 days of acquisition training and 9-15 days of extinction training. Preliminary data suggests that bilateral maxadilan infusions into the BNST may cause reinstatement of cocaine seeking. To determine if PACAP is likely released during stress induced reinstatement, in experiment 2, extracellular signal-regulated kinase (pERK), a downstream target of PAC1 receptor activation, was assessed after footshock or no footshock paired with PACAP6-38 or vehicle infusions. If footshock leads to PACAP receptor activation, we predict that there will be increased activation of ERK in the BNST after footshock but not after PACAP6-38 infusion. Preliminary data shows that pERK staining can be seen in the BNST but further data analysis is needed to see whether footshock or PACAP6-38 effect this activation.

Miami University

Effect of Adolescent Binge Drinking Context on the Rewarding Properties of Alcohol

Binge drinking is a pervasive problem for adolescents, particularly on college campuses. Because the prefrontal cortex is still developing in adolescence, the brain is vulnerable to enduring damage and alteration due to excessive alcohol use. These alterations persist into adulthood and produce lasting biological and behavioral consequences. Knowledge of the motivators of drinking allows for the invention and implementation of relevant and accurate education and prevention strategies. There are strong social and physiological motivators for adolescent binge drinking. For example, the decision to drink alcohol can result from both its euphoric effects and its socially facilitating effects. However, it is unknown whether the indirect social rewards or the direct physiological rewards derived from the alcohol are most influential on drinking patterns. The purpose of this study was to determine if the physiological effects of alcohol or the social rewards are greater, and if these motivators potentially contribute to the development of addiction. This was tested through the rats' self-administration of alcohol, in the form of gelatin. Rats received alcohol access either with social partners or in isolation, every night throughout adolescence. To assess the relative preference for social interaction versus alcohol, rats were then tested in a Conditioned Place Preference (CPP) apparatus to determine if rats established CPP for either a social conspecific or access to alcohol. Our hypothesis was that rats who ingested alcohol in a social context would prefer a social reward, whereas, the rats who ingested alcohol in isolation would prefer the alcohol reward. Preliminary results determined that the context of drinking (social or isolation) did not have an effect on the preference for a social partner compared with alcohol, although methodological issues may have confounded these results. Because of this, it is still unknown which motivating factor of drinking, social vs physiological, is greater, and if this is contingent upon the context of drinking. Methodological changes have been made for future cohorts to improve the parameters of CPP. Development of new treatments and alcohol education tactics, particularly within a high school or college setting is a necessity, and future studies will work towards this goal.

Impact of early life trauma and adolescent alcohol exposure on adult PTSD- and AUD-like phenotypes

The latest estimates using the criteria of the DSM-V suggest post-traumatic stress disorder (PTSD) has a lifetime prevalence rate of 8.3%. Individuals suffering from PTSD show a heightened vulnerability to alcohol use disorder (AUD), possibly because the neural substrates mediating vulnerability in PTSD and AUD are similar. Individuals with comorbid PTSD/AUD are also more likely to have suffered childhood adversities than those with only one of these conditions, pointing to childhood trauma as an important vulnerability factor for comorbid PTSD/AUD. The present experiment addresses whether infant stress exposure (footshock) on postnatal day 17 increases alcohol consumption in adolescence and then in adulthood, using an intermittent two-bottle choice procedure. In addition, we address whether the stress exposure results in faster progression to compulsive alcohol drinking. Finally, we are addressing whether the enhanced adult fear conditioning that is observed in animals previously exposed to the infant footshock stress correlates with the amount of alcohol consumed during adolescence and/or adulthood. The results of this experiment will help to determine further whether our infant stress model of PTSD is valid for investigations of comorbid PTSD/AUD.

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Miami University

Increased fear memory expression following early life stress exposure: An animal model of PTSD

Early life stress exposure yields increased vulnerability for the development of posttraumatic stress disorder (PTSD) in adulthood. This has been modeled in rodents by showing that adult fear learning with mild to moderate aversive stimuli is enhanced in adulthood following early life stress exposure. In patients experiencing symptoms of PTSD, fear memory is resistant to extinction during exposure therapy, producing sustained exaggerated fear responses. However, these patients do not demonstrate symptoms of increased general anxiety. The present experiment addresses whether early life trauma exposure produces enhanced fear learning in adulthood (stress enhanced of fear learning; SEFL) that is resistant to extinction. In a separate experiment, we assessed whether an identical early life trauma experience yields incubation of fear in adulthood. Rats were exposed to zero or 15 footshocks on postnatal day 17 (PND17). In adulthood (approximately PND90), rats underwent fear conditioning in a novel context using 0, 1 or 3 footshocks at 0.5, 0.75 or 1mA. Rats were tested for enhanced fear learning 1 or 30 days following fear conditioning. A subset of these rats was subjected to an additional 4 days of extinction training. Rats fear conditioned with a single 0.5mA footshock showed incubation of fear over time, from 1 day to 30 days following fear conditioning. On day 1 of extinction, trauma-exposed rats extinguished to control levels across the 10-minute session. However, on day 2 of extinction, trauma-exposed rats froze significantly higher than controls. Thus, within-session extinction looked comparable between trauma-exposed and control animals. However, between-session extinction was impaired in trauma-exposed rats. These data demonstrate pathological fear memory regulation in adulthood following early life stress exposure and provide additional strong construct validity for the stress-enhanced fear learning (SEFL) model in the study of PTSD.

Oberlin College

Effects of autism-associated environmental factors on locomotive behavior of zebrafish larvae

Autism Spectrum Disorders (ASD) involve changes in motor development, and those with ASD often experience behavioral differences such as repetitive motion (e.g. arm flapping, rocking) or difficulties initiating movement. Recent epidemiological research indicates that exposure of pregnant mothers to certain environmental compounds has been linked to an increased risk of developing ASD in their children. How these chemicals affect development of motor pathways to result in behavioral changes is unknown. The purpose of this research is to assess motor behavior of zebrafish larvae following exposure to chemicals implicated in ASD risk. Embryos are exposed on the day of fertilization to concentrations of chemicals that have been found to affect the morphology and gene regulation of zebrafish larvae. At 5 days post fertilization (dpf), these exposed larvae are transferred into 96-well-plates in a controlled environment and their swimming behavior is digitally recorded. The swimming behavior is analyzed through open-source MATLAB software to identify changes in swimming velocity and total distance moved. Changes in swimming behavior following exposure to chemicals may result from changes to neural development and spinal morphology. Results gained from this experiment will provide further information on the developmental effects of prenatal chemical exposure relevant to understanding ASD mechanisms.

Earlham College

Changes in excitability protein expression during the first week after spinal cord injury in lamprey

Spinal cord injury (SCI) is damage to any part of the spinal cord or the nerves at the end of the spinal cord canal. Since human spinal cord neurons do not regenerate after injury, SCI and the resulting pain and paralysis are major medical problems worldwide. We studied the molecular mechanisms of spinal cord regeneration using lamprey, primitive vertebrate fish that regenerate their spinal cord neurons after injury. Neuronal regeneration in lamprey could be due to decreased neuronal excitability and changes in astrocyte activity, causing decreased excitotoxicity after injury. Thus, we hypothesized that we would observe decreased voltage-gated sodium channel (Nav) and calcium channel (Cav) expression, increased potassium channel (Kv) expression, changes in glutamate localization, and altered astrocyte morphology after SCI. Lamprey were given complete spinal cord transections using small-animal surgical techniques. They recovered for one day or one week, and their spinal cords and brains were dissected. Tissues were cryoprotected, cryosectioned, labeled with primary and fluorescently-tagged secondary antibodies, and imaged with fluorescence microscopy. As expected, we observed decreased immunoreactivity of Nav and Cav channels and increased immunoreactivity of Kv channels. We also observed distinct changes in astrocyte morphology, and unexpected increases in glutamate immunoreactivity. These findings may help lead to novel forms of treatment for human spinal cord injury in the future.

Miami University

Playing the Odds: Examining Decision- Making Differences Under Restricted Resources

Restricted access to resources, such as educational, nutritional, and financial, is known to elicit change in human behavior, often leading to suboptimal decisions. Much of our current understanding of the neurocognitive factors that control decision-making has been established through rodent models, relying on food-based reward. In such models, subjects are food restricted to motivate them to complete behavioral tasks; however, the degree of food restriction and methods used in its execution vary amongst researchers in the field. Prior research has been focused on the effects of food restriction on the biology of the brain with little literature on the influence various methods of food restriction have on behavior and decision-making. The purpose of this study is to determine the impact of food restriction amounts and methods on decision-making and impulsive behaviors. We hypothesized that animals on a more restricted diet would make more impulsive decisions during behavioral tasks. Adult rats were assigned to four treatment groups to determine the amount of food they were given each day: percent of body weight (2.5% and 3.3%), fixed amount (14 g), and temporal (2 hr) access. Subjects were tested in two behavioral tasks, probability discounting and delay discounting. During the probability discounting task, animals chose between a guaranteed small reward (1 sugar pellet) or an uncertain large reward (3 sugar pellets). In the delay discounting task, the large reward was delivered after an increasing delay, while the small reward delay stayed constant. We found that subjects receiving 2.5% food restriction made more impulsive choices during the delay discounting task, but performed normally during probability discounting. Additionally, animals in the temporal and fixed amount groups showed significant differences in task performance compared to the weight-based restricted groups. Temporal and fixed amounts also showed differences during the delay discounting task. These results suggest that the type of food restriction may uniquely impact areas of the brain involved in different decision-making tasks. In addition, future studies using the delay discounting task should give special attention to the degree of food restriction to avoid influencing results. These data support previous conclusions on the effect of limited access to resources on changing behaviors.

Effects of physiological stress on hnRNP K gene expression in the Drosophila brain

GSK-3b is an enzymatic regulator of cell metabolism and cell survival in nearly all eukaryotic cell types. Neurons likely use different mechanisms for survival than other cell types, given that neurons are post-mitotic and most are not replaced if they are damaged or die. We hypothesize that one way neurons may mitigate harmful effects is by regulating GSK-3b activity differentially than is regulated in other cell types. We have previously shown that changes in *hnRNP K* expression alter GSK-3b activity via the insulin signaling pathway. We hypothesized that physiological insults would lead to changes in *hnRNP K* signaling in the brain in order to modulate GSK-3b signaling. To test this hypothesis, we used heat shock as a physiological stress and analyzed gene expression at various time points in brain and non-brain tissues for comparison. Our results show that physiological stress does not have a substantial effect in *hnRNP K* gene expression in non-brain tissue, however there are variable effects of expression in the brain. These results, while preliminary, support our hypothesis of differential modulation of GSK-3b activity in neuronal tissue in response to cellular stress, and that *hnRNP K* may be a key protein required for this differential effect in neurons.

Baldwin Wallace University

Assessing the impact of 4th ventricular administration of the dopamine D2 receptor antagonist raclopride on sucrose-induced c-fos expression in the rostral nucleus of the solitary tract

This study served as a continuation of a pilot study which suggested a potential neural mechanism underlying the diminishing effects of raclopride on taste sensitivity. We hypothesized that these effects were the result of antagonistic action of raclopride on the dopamine D2 receptors present in the rostral nucleus of the solitary tract (rNST). The inhibition of these receptors could block the transmission of gustatory information from the rNST to the gustatory cortex and other neural regions associated with taste perception. This mechanism was tested through the 4th intracerebroventricular (4icv) administration of raclopride followed by intraoral (IO) infusion of sucrose. Neural activity resulting from this combination of inhibitory and stimulating infusions was measured via FOS activation using immunohistochemistry. The data collected from this experiment were largely inconclusive, contrasting with the preliminary data from the pilot study. The low number of usable tissue samples collected from subjects as well as technical difficulties with the antibody used during the immunohistochemical analysis may have led to these inconclusive data. A further replication study is necessary to assess the validity of the hypothesized neural mechanism underlying the effects of raclopride.

Ohio University

Determining neural disruptions in the cerebellum following endothelial deletion of Rbpj

Cerebrovascular disease, including stroke and arteriovenous malformation, often leads to neurological dysfunction and compromised quality of life. Brain arteriovenous malformation (BAVM) is a severe cerebrovascular disease characterized by enlarged, tortuous vessels and arteriovenous shunting, which connects arteries directly to veins and may lead to tissue hypoxia, stroke, and neurological deficit. Existing treatment options for BAVM include high-risk treatments that may not be applicable or available to all BAVM patients. Even with successful resection or shrinking of the vascular malformations, damage to the neural compartment may accompany and present long-term neurological complications. Currently, little is known about the mechanisms underlying neural disruptions in BAVM – our goal is to uncover such mechanisms. Our previous work has shown that deletion of *Rbpj*, a transcriptional regulator of Notch signaling, from postnatal endothelium leads to features of BAVM in mice. Published data indicates gross- and histo-pathological neural abnormalities in 100% of *Rbpj*-mutant mice. Given that the cerebellum undergoes extensive morphogenesis – of both its vascular and neural compartments – during this early postnatal period, we sought to define how the neuronal migration, lamination, and circuitry formation are affected alongside vascular abnormalities in *Rbpj* mediated BAVM. Specifically, we tested the hypothesis that deletion of *Rbpj* from early postnatal vascular endothelium disrupts postnatal cerebellar morphogenesis and gross motor behavior in mice. We found that, following endothelial deletion of *Rbpj* at birth, the stereotypical foliation pattern, lobule outgrowth, and neuronal lamination of the cerebellum are affected within two weeks of postnatal development. Interestingly, a subset of lobules was preferentially affected, suggesting regional regulation of *Rbpj*. These results suggest that endothelial *Rbpj* influences the neural cerebellar compartment and is required for proper cerebellar morphogenesis during early postnatal life.

Baldwin Wallace University

Determining Proestrus Estradiol Concentrations and Testing the Efficacy of Hormone Replacement Treatment on Gonadectomized Sprague-Dawley Rats

Previously, our laboratory demonstrated that female rats in the proestrus stage discriminated sucrose from water less accurately than did male rats across all sucrose concentrations. Before investigating further whether or not estrogen played a role in this detection difference, we sought to determine the circulating levels of estrogen present in the female rats during proestrus, and whether or not this level could be recapitulated in male rats through hormone replacement treatment. Blood serum samples were taken from anesthetized female rats during the proestrus and diestrus stages of estrus and from intact male rats (n=6/grp). The female rats and 6 male rats were then bilaterally gonadectomized. Following recovery, female rats, intact male rats, and GDX male rats (n=6/grp) underwent hormone replacement treatment, during which estrogen dissolved in DMSO (2micrograms/.1ml sc) was injected every fourth day for a total of 16 days. Blood serum samples were taken 6hr, 18hr, and 48hr after the fourth and final injection. All of the collected serum samples were then analyzed using a competitive enzyme-linked immunosorbent assay (ELISA). High absorbance readings precluded quantifiable measures of estrogen concentrations. Analysis of absorbance levels did not reveal any significant differences between groups, potentially due to hemolyzed samples. Future studies are needed to provide a clear estrogen level reading and delineate an exogenous regimen that would mimic the estrogen levels across estrous.

Grand Valley State University

The Effect of CRF₂ Receptor Regulation on the Depressive – Like Behavior Experienced During Protracted Ethanol Withdrawal

There are many times in which stress plays an influential role in alcohol addiction, but it is most influential during the time of relapse, in which there are cycles of heightened anxiety, depressive mood, and negative affect (Logrip, et al., 2011). Clinical studies have suggested that the most common reason for relapse is the ability for alcohol to relieve these negative mood symptoms experienced during withdrawal (Hershon, 1977; Cloninger, 1987). The target of this study is the corticotropin-releasing factor (CRF) system, which is understood to play a central role in the regulating the behavioral stress response within the body (Dunn and Barridge, 1990). It is our hope that by regulating the depression experienced, we can begin the foundation for long-term strategies in the prevention of relapse following protracted abstinence. Urocortin 3 (Ucn 3) is the highly selective CRF₂ receptor agonist (Lewis et al., 2001), and studies have shown that Ucn 3 has the ability to reduce behaviors associated with depression in animal models (Tanaka and Telegdy, 2008). However, the ability Ucn 3 to diminish depressive-like behaviors during protracted withdrawal has yet to be studied. In order to examine the effects of Ucn 3 following protracted abstinence, male and female Wistar rats (n=24) were given a liquid diet consisting of a chocolate nutritional shake, vitamins, minerals, and 10% ethanol as their sole source of nutrition for 25 days. Those in the control group were given sugar as a caloric substitute for the ethanol and were fed the average amount of the liquid diet that the ethanol group consumed the previous day. The results showed that there were no statistical differences found between diet and the body weight or fluid intake for either male or female rats. This ensures that any potential behavioral differences cannot be attributed to nutritional deficiency or dehydration. The amount of ethanol consumed by the rats has been previously shown to produce blood ethanol levels of 150-225 mg/dl (Macey, 1996). One day after removal of the liquid diet, rats were observed for physical signs of alcohol withdrawal. The results from this assessment showed that ethanol liquid diet fed rats had significantly higher withdrawal scores, confirming physical dependence to alcohol. After five weeks during which rats were left undisturbed with the exception of routine husbandry, the rats were examined in the forced swim test. The results from the test did not show a statistically significant interaction between diet, drug, and time spent immobile. However, post hoc analysis did show a trend of less time spent immobile when injected with Ucn 3 during ethanol withdrawal that approached statistical significance. Further studies would be need to be conducted in order to confirm the hypothesis that Ucn 3 has the ability to alleviate depression experienced during withdrawal, but the trend shows that there is reason to be

cautiously optimistic about the future pharmaceutical opportunity to help those going through long term withdrawal in their path to remaining abstinent.

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This image shows a full page of blank handwriting practice paper. It features approximately 28 evenly spaced horizontal black lines across the entire page, providing a guide for letter height and placement. The background is plain white, and there are no margins, text, or other markings present.

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This image shows a single sheet of white paper with horizontal ruling lines. The lines are evenly spaced and run across the width of the page. There are no margins, text, or other markings on the paper.

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again next year when
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