6th Annual mGluRs
Midwest/Great Lakes
Undergraduate Research
Symposium in Neuroscience
Hosted by Wabash College
September 20, 2014

Generously sponsored by:

UNIVERSITY OF Cincinnati
Neuroscience Graduate Program

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THE INTERNATIONAL HONOR SOCIETY IN PSYCHOLOGY
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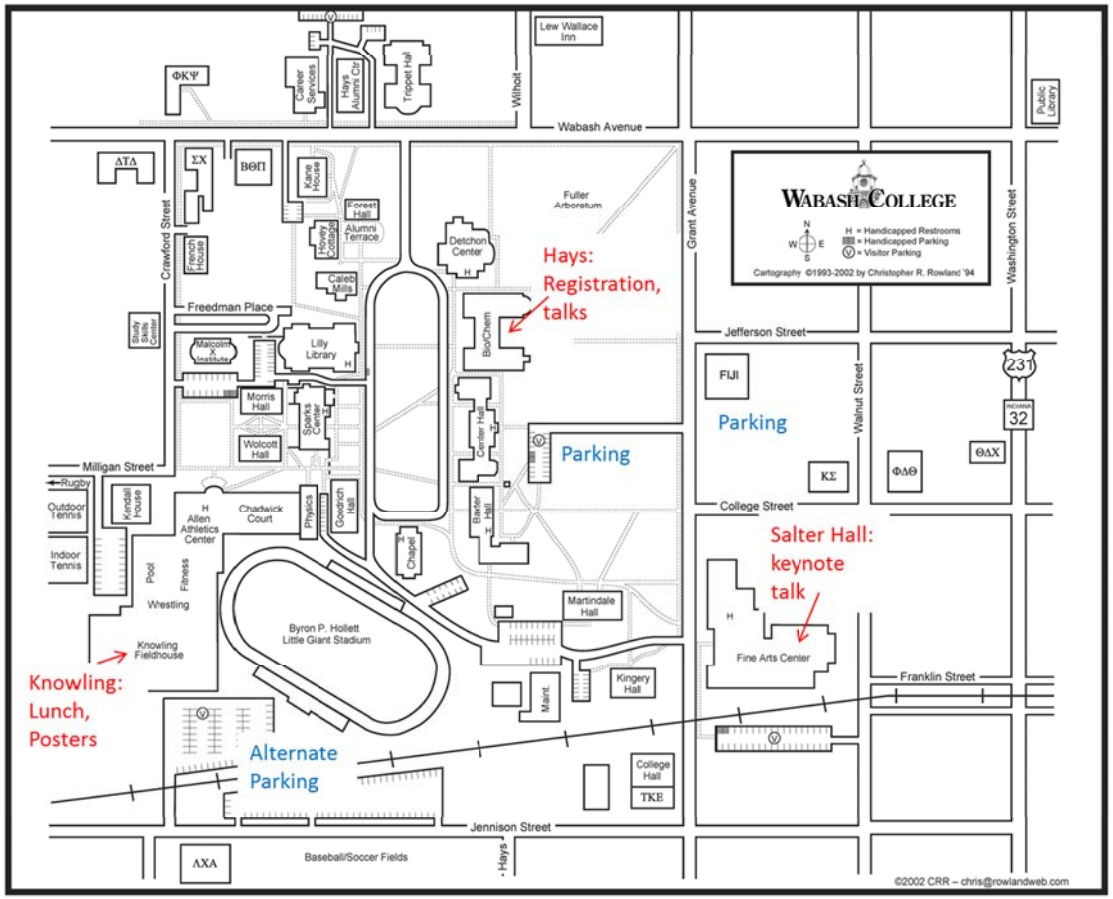
Wabash College
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H = Handicapped Restrooms
= Handicapped Parking
V = Visitor Parking
Hays: Registration, talks
Parking
Salter Hall: keynote talk
Knowling: Lunch, Posters
Alternate Parking
Wabash Avenue
Jefferson Street
College Street
Milligan Street
Jennison Street
Franklin Street
Crawford Street
Grant Avenue
Washington Street
Wilhoit
Walnut Street
Freedman Place
Lew Wallace Inn
Public Library
Career Services
Hays Alumni Ctr
Trippet Hall
Kane House
Forest Hall
Alumni Terrace
Hovey Cottage
French House
Caleb Mills
Fuller Arboretum
Detchon Center
Bus/Chem
Center Hall
Baxter Hall
Chapel
Martindale Hall
Kingery Hall
Maint.
College Hall
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Study Skills Center
Malcolm X Institute
Lilly Library
Morris Hall
Spanks Center
Wolcott Hall
Rugby
Outdoor Tennis
Indoor Tennis
Kindall House
Allen Athletics Center
Chadwick Court
Physics
Goodrich Hall
Pool
Fitness
Wrestling
Knowling Fieldhouse
Byron P. Hollett Little Giant Stadium
Baseball/Soccer Fields
Fine Arts Center
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mGluRs 2014 Schedule

Saturday, September 20, 2014

Time	Activity	Location
8:30-9:00 am	Registration & Continental Breakfast	Hays Lobby
9:00 am	Welcome by Dean Scott Feller Brief overview of the day's schedule	Hays 104
9:30-10:15 am	Student Presentations **VISUAL SEARCH REVEALS POORER PERFORMANCE OF NON-CARDINAL COLORS AT VARYING STIMULUS SIZES** *Colin Downey* *Psychology Department,* *Wabash College* **MORPHOLOGICAL ALTERATIONS IN MICROGLIA ARE DELAYED COMPARED TO THE INDUCTION OF GLIAL CYTOKINE EXPRESSION AND SICKNESS BEHAVIOR AFTER AN ACUTE IMMUNE CHALLENGE** *Paige Trojanowski* *Neuroscience Department,* *Institute for Behavioral Medicine Research,* *The Ohio State University* **PREPULSE INHIBITION: ARE WE INHIBITING OUR RESULTS, TOO?** *Rachel A. Zacharias* *Department of Psychology & Neuroscience Program,* *Baldwin Wallace University*	Hays 104
10:15-10:30	NuRhoPsi, Neuroscience Honors Society Zoe Hesp, The Ohio State University	
10:30-11:00 am	Coffee break	Hays Lobby
11:00-12:00 pm	Breakout Session I	
	Graduate & Medical School Panel Kim Seroogy & Mark Baccei Neuroscience Graduate Program, University of Cincinnati Stephen Burns School of Optometry, Indiana University David Braitman, W'09 4th Year Medical Student, Indiana University	Hays 104

Time	Activity	Location
	Faculty Discussion Immersion Learning Neil Schmitzer-Torbert, Wabash College Bob Rosenberg, Earlham College	Hays 319
12:00-1:00 pm	Buffet Lunch	Knowling
1:00-1:45 pm	Odd posters present	Knowling
1:45-2:30 pm	Even posters present	Knowling
2:30-2:45 pm	Coffee break	Salter Lobby
2:45-3:45 pm	Keynote Address Dr. Eric Nauman Professor of Biomedical Engineering, Mechanical Engineering, & Basic Medical Sciences Director, College of Engineering Honors Program, Purdue University *Neurophysiological Consequences of Repetitive Sub-Concussive Trauma* KEYNOTE CO-SPONSORED BY THE WABASH CHAPTER OF PSICHI	Salter Hall
3:45-4:45 pm	Breakout Session II	
	Alternative Careers in Science Jill Rogers, genetic counselor Kathleen Novak, pharmacist Shannon Hudson, middle school science teacher Jim Leuck, W'09, neurophysiology technologist	Hays 104
	Faculty Discussion What components should be in a neuroscience major or minor? Karen Gunther, Wabash College	Hays 319
4:45-5:00 pm	Closing Remarks Welcome to next year by Charlie Campbell, OSU host.	Hays 104

We hope to see you again at The Ohio State University (Mershon Auditorium) on Saturday, Oct. 3, 2015!

1. VISUAL SEARCH REVEALS POORER PERFORMANCE OF NON-CARDINAL COLORS AT VARYING STIMULUS SIZES

Colin Downey & Karen Gunther
Department of Psychology, Wabash College

The current experiment seeks to understand the effect of stimulus size on non-cardinal color mechanisms in all three planes of color space. Multiple studies have shown subjects to perform better on cardinal colors than non-cardinal colors, especially in the Red-Green (RG)/Black-White (Luminance a.k.a. LUM) and Blue-Yellow (BY)/LUM color planes. Solomon, Peirce, and Lennie (2004) showed that stimulus size affects the strength of the luminance suppressive surround, which might make non-cardinal mechanisms in the RG/LUM and BY/LUM planes more sensitive to stimulus size. We therefore tested 10 color-normal subjects on visual search at four dot sizes (0.5, 1, 2, & 3°) in each of the three color planes (RG/BY, RG/LUM, & BY/LUM). A two-way ANOVA on the RG/BY color plane yielded a significant main effect of color axis (p=0.024, η^2=0.45), a significant main effect of dot size (p=<0.001, η^2=0.52), but no interaction (p=0.365). The RG/LUM color plane also yielded a significant main effect of color axis (p=0.020, η^2=0.47), a significant main effect of dot size (p=<0.001, η^2=0.65), but no interaction (p=0.282). Similarly, the BY/LUM color plane yielded a significant main effect of color axis (p=0.020, η^2=0.47), a significant main effect of dot size (p=<0.001, η^2=0.48), but again no interaction (p=0.424). These results suggest that non-cardinal mechanisms in the RG/LUM color plane may be more sensitive to stimulus size (due to a larger effect size) than the isoluminant plane. Testing is underway with smaller dot sizes (0.25, 0.5, 1, & 2°), to see if we can increase the effect size of dot size within the BY/LUM plane.

2. MORPHOLOGICAL ALTERATIONS IN MICROGLIA ARE DELAYED COMPARED TO THE INDUCTION OF GLIAL CYTOKINE EXPRESSION AND SICKNESS BEHAVIOR AFTER AN ACUTE IMMUNE CHALLENGE

Trojanowski, P.J., Villanueva, E., Norden, D.M., & Godbout, J.P.
Neuroscience Department, Institute for Behavioral Medicine Research, The Ohio State University

Microglia and astrocytes interpret and propagate inflammatory signals initiated in the periphery, coordinating communication between the immune system and the brain. These biochemical, physiological, and morphological changes in activation influence behavior and cognition. One issue in glial biology is that analysis of morphology alone is used to interpret the activation state of glia. Therefore, the purpose of this study was to provide a detailed time course of inflammatory markers in enriched microglia and astrocytes compared to their

morphological profiles. Adult BALB/c mice received an intraperitoneal injection of lipopolysaccharide (LPS) (10 μg), which evoked a pro-inflammatory CNS response and induced sickness behavior. One acute LPS injection sufficiently activated both glia, causing time dependent increases in mRNA expression of IL-1β, IL-6, CCL2, and TNFα. Specifically, the height of cytokine mRNA expression after LPS was at 4 h in microglia and 12 h in astrocytes. Moreover, the induction of IL-6 mRNA preceded that of IL-1β and TNFα in both microglia and astrocytes. Body weight, locomotion, and social exploratory behavior also decreased following LPS. Behavioral sickness peaked between 2-12 h and was resolved within 24 h. Microglial morphological changes with increased Iba-1 immunoreactivity were detected at 24 and 48 h, inconsistent with pro-inflammatory cytokine expression. Changes in IL-1β, IL-6, CCL2, and TNFα expression temporally paralleled sickness behavior in microglia but the de-ramified microglial morphology did not. These data indicate that cytokine mRNA expression precedes microglial morphological alterations and that cytokine up-regulation does not correspond directly to de-ramified microglial morphology.

3. PREPULSE INHIBITION: ARE WE INHIBITING OUR RESULTS, TOO?

Rachel A. Zacharias, Janace J. Gifford, Christopher P. Turner, Brian L. Thomas
Department of Psychology & Neuroscience Program, Baldwin Wallace University

Prepulse inhibition (PPI) is a task that is often employed to measure the acoustic startle response in animals. Deficits in PPI have been linked to schizophrenia, Alzheimer's disease, and impaired auditory processing. Evidence suggests that anesthetic compounds can impair normal brain development and impair auditory processing during infancy. In the present study, neonatal rats were exposed to the anesthetic drugs MK801 (1 mg/kg; n = 13) or ketamine (20 mg/kg; n = 15) at P7 and were then compared with saline control animals (n = 13) once per week from P8 to P70 on a PPI task. Results indicated that ketamine decreased peak and average startle amplitude (over trials) on low and high prepulse and pulse only trials at P21. MK801 also decreased the startle response at P21, but to a lesser degree than ketamine. Thus, neonatal exposure to NMDA receptor antagonists can cause deficits in prepulse inhibition at P21. Considerations for data processing will also be presented. PPI is typically determined using an equation that indicates if the startle response changes when a prepulse (warning) is provided prior to the startle stimulus; however, one may also determine PPI by analyzing prepulse trials without transforming them via a percent PPI equation. Differences in data processing may influence the results of PPI measures.

KEYNOTE ABSTRACT – 2:45-3:45pm, Salter Hall

Neurophysiological Consequences of Repetitive Sub-Concussive Trauma

Dr. Eric Nauman
Professor of Biomedical Engineering, Mechanical Engineering, & Basic Medical Sciences
Director, College of Engineering Honors Program,
Purdue University

KEYNOTE CO-SPONSORED BY THE WABASH CHAPTER OF PSICHI

Conservative estimates indicate that, in 2003, there were 1.5 million cases of traumatic brain injury (TBI) in the United States, resulting in 51,000 deaths, 290,000 hospitalizations, and over 1 million emergency room visits. The incidence of TBI is especially high in children under the age of 14, many of whom participate in contact sports such as football, hockey, and lacrosse. Today, approximately 3.2 million individuals are living with long-term disabilities as a result of TBI-related injuries. Based on recent work by the Purdue Neurotrauma Group (PNG), it has become clear that these numbers are only the tip of the iceberg. Chronic neurophysiological changes resulting from head trauma affect more than 70% of a typical high school football team. As a result, the Centers for Disease Control noted that TBI is no longer a silent epidemic, but something that has the potential to affect people of all ages and all walks of life. In order to prevent these injuries and improve treatment outcomes it is necessary to develop (1) methods for the early identification of neurophysiological changes, and (2) the ability to identify which anatomical structures and cell types are injured. This talk will focus on the quantification of injuries and their neurophysiological consequences in an effort to begin the discussion of how best to synthesize contributions from engineering, medical imaging, athletic training and biology.

1. THE EFFECTS OF KETANSERIN ON CHRONIC NEUROINFLAMMATION

Crockett, A. M. [1,2], D'Angelo, H.M.[1], Hopp, S.C.[2], Royer, S.[2], Adzovic, L.[1], Wenk, G.L.[1,2]
[1]Psychology Department, The Ohio State University; [2]Neuroscience Department, The Ohio State University

Chronic neuroinflammation may be an important aspect of depression and its symptoms. Protracted neuroinflammation induces excessive microglial activation and increases in pro-inflammatory cytokines, as well as spatial memory impairments. Previous antidepressants have been shown to have anti-inflammatory properties, however much of their success has been attributed to their ability to inhibit serotonin reuptake. The current study looks at the anti-inflammatory effects of Ketanserin, a serotonin receptor antagonist. Rats received an intraventricular infusion of lipopolysaccharide (LPS) for 28 days, and were treated with injections of ketanserin or the vehicle. The animals given the sucrose test to measure their depressive symptoms. The study also quantified microglial activation in the hippocampus as well as pro-and anti-inflammatory cytokine protein and mRNA expression. Our preliminary results suggest that ketanserin has anti-inflammatory proclivities in the brain, which may be the basis of its anti-depressant actions.

2. GESTATIONAL STRESS ALTERS MATERNAL MOTIVATION AND INDUCES ANHEDONIA, ROLE OF DOPAMINE SIGNALING IN THE NUCLEUS ACCUMBENS SHELL

Morgan L. Sherer, Achikam Haim, Chris Albin-Brooks, Emily Mills & Benedetta Leuner
Neuroscience Department, The Ohio State University

Postpartum depression (PPD) is a common complication following childbirth experienced by 20% of all new mothers. Mothers diagnosed with PPD often experience anhedonia and impaired mother-infant interactions and yet the underlying neural mechanisms mediating these symptoms of PPD remain unspecified. Maternal care is a goal-oriented, highly motivated behavior, which in conjunction with increased anhedonia suggests that deficits in reward and motivation processing might play a role. Here, we investigated the effects of gestational stress, a risk factor for PPD, on maternal motivation and anhedonia in rats using the conditioned place preference test and sucrose preference test respectively. We also investigated the underlying mechanism of this phenomenon by examining tyrosine hydroxylase (TH) as a marker of DA signaling in the nucleus accumbens (NAc) shell, a critical component of the brain's reward circuit. Our results demonstrate that gestational stress induces anhedonia and deficits in maternal motivation. We also found decreased TH in mothers that experienced gestational stress. Overall, our results show that chronic gestational stress is a valid and useful animal model to investigate the

underlying neural mechanisms of PPD which may involve a reduction in DA signaling in the NAc.

3. POSITIVE ALLOSTERIC MODULATION OF THE A7 NICOTINIC ACETYLCHOLINE RECEPTOR INTERACTS WITH MESOLIMBIC STIMULATION IN EVOKED PREFRONTAL GLUTAMATE RELEASE

Brian Upton, David Bortz, and John Bruno
Neuroscience Program, The Ohio State University

Cognitive deficits in schizophrenia are thought to be caused, in part, by disrupted prefrontal cholinergic and glutamatergic transmission. Activation of the α7 nicotinic acetylcholine receptor (α7nAChR) has been shown to increase prefrontal glutamate, as well as rescue failing performance in cognitive tasks in rodents and primates. Intra-accumbens stimulation with NMDA dose-dependently increases prefrontal acetylcholine, which in turn increases prefrontal glutamate via α7nAChR activity. This assay was used to determine if two α7nAChR positive allosteric modulators (PAMs), AVL-3288 and PNU-120596, are able to potentiate glutamate release as a function of the amount of acetylcholine release in the PFC. Second-by-second measurements with a glutamate-sensitive microelectrode in the PFC of awake rats reveal that only an appropriate combination of the dose of NMDA and dose of PAM consistently potentiates prefrontal glutamate release. However, at other concentrations of NMDA and PAM, the effect on mesolimbic stimulation varied greatly from significant potentiation to a reduction of glutamate release. Furthermore, the type-I PAM, AVL-3288, led to desensitization over several days, whereas the type-II PAM, PNU-120596, did not. Overall, these results demonstrate the importance of dose of α7nAChR PAMs in modulating neurotransmitter systems, and PAMs as a potential therapeutic in treating cognitive deficits of schizophrenia.

4. CHRONIC GESTATIONAL STRESS LEADS TO DEPRESSIVE-LIKE BEHAVIOR AND COMPROMISES MEDIAL PREFRONTAL CORTEX STRUCTURE AND FUNCTION DURING THE POSTPARTUM PERIOD

Nealer C, Leuner B, Fredricks PJ, Albin-Brooks C
Departments of Neuroscience and Psychology, The Ohio State University

Postpartum depression, which affects approximately 15% of new mothers, is associated with impaired mother-infant interactions and deficits in cognitive function. Exposure to stress during pregnancy is a major risk factor for postpartum depression. However, little is known about the neural consequences of gestational stress. The medial prefrontal cortex (mPFC) is a brain region that has been linked to stress, cognition, maternal care, and mood disorders including postpartum depression. Here we examined the effects of chronic gestational stress on mPFC function and whether these effects might be linked to structural modifications in the mPFC. We found that in postpartum rats, chronic gestational stress resulted in maternal care deficits, increased

depressive-like behavior on a forced-swim test, and impaired performance on an attentional set shifting task that relies on the mPFC. Furthermore, exposure to chronic stress during pregnancy reduced dendritic spine density on mPFC pyramidal neurons and altered spine morphology. Taken together, these findings suggest that pregnancy stress may contribute to postpartum mental illness and its associated symptoms by compromising structural plasticity in the mPFC.

5. AAV9 TRANSDUCTION OF MYENTERIC NEURONS BY INTRAVENOUS INJECTION

Christopher Cowley, Kevin Foust, Sara Gombash-Lampe
Department of Neuroscience, the Ohio State University

It has recently been shown that adeno-associated virus type 9 (AAV9) can cross the blood brain barrier (BBB), thus making it a promising technique for the treatment of global neurological disease. Intravenous injection produces widespread vector distribution, allowing for treatment of neurological disease with peripheral involvement. Gastrointestinal (GI) symptoms are often reported by patients with neurologic and neuromuscular disease, with these symptoms impairing their quality of life. These symptoms can be a direct result of impaired neuromuscular transmission from the enteric nervous system (ENS). The purpose of the study was to characterize AAV transduction in the ENS of neonatal and juvenile mice. Newborn mice (postnatal day 1) and juvenile (postnatal day 21) were injected intravenously with AAV9 expressing green fluorescent protein (GFP) and euthanatized as adults, after which their GI tracts were dissected. Additional cohorts of neonatal mice were injected with AAV serotypes 1, 5, 6, and 8 expressing GFP determining if a BBB penetrating AAV was necessary for ENS transduction. Immunohistochemical labeling showed GFP transduction of 25-57% of enteric neurons in AAV9 injected neonatal or juvenile mice with prominent expression in choline acetyl transferase positive cells, but not in vasoactive intestinal peptide or neuronal nitric oxide synthase cells, suggesting a bias toward excitatory neurons. Furthermore, AAV8 mediated transduction was similar to AAV9 with AAV1, 5 and 6 transduction being less efficient. These data show that through intravenous injection, AAV9 can be used in the development of therapies for the ENS as well as provide further insight in ENS functioning.

6. ROLE OF PAR-1B KINASE IN SPINE MORPHOGENESIS, LEARNING, AND MEMORY

Anna Damato[1], Huaye Zhang[2], Amy Jo Stavnezer[1]
[1]Dept of Neuroscience, The College of Wooster
[2]Dept of Neuroscience and Cell Biology, Rutgers Robert Wood Johnson Medical School

Dendritic spine plasticity is thought to play a key role in cognitive function, as changes in spine density and structure have been observed in neurological disorders. It was previously established that a decrease in Par-1b kinase activity

leads to immature spine formation *in vitro*. The objective of this research was to determine the effect of Par-1b kinase on spine morphogenesis *in vivo.* Quantification of dendritic spines in coronal sections of Par-1b/Thy-1-YFP mice showed that homozygous (Par-1b -/-) mice have significantly higher spine density (number of spines/ 100 µm) and spines that are longer and thinner than those in wild type mice. The Morris Water Maze and Active Avoidance Test were carried out using Par-1b knockout mice and show learning and memory deficits in knockout mice. In the Morris Water Maze, knockout mice took significantly more time to reach the platform than the wild type mice and in the Active Avoidance Test, knockout mice took significantly more time to reach the shock free zone and escaped the shock, instead of avoiding it. These results suggest that Par-1b knockout mice have deficits in associative and spatial learning and memory. Multi-layer immunohistochemical staining resulted in the visualization of a thinner neocortex in knockout mice compared to wild type and heterozygous mice. This research has exposed many new and exciting questions and topics for research into the causes and effects of abnormal spine morphogenesis *in vivo.*

7. Decreased Sodium Channel Expression during Spinal Cord Regeneration in Lamprey

J. Anna Juras, Avalokiteswari A. Kurup*, Ruth Y. Lewis*, Rebecca C. Palmarini*, Elizabeth S. Richards*, Yim J. Rodriguez†, Robert L. Rosenberg*†#*
**Biochemistry Program, †Neuroscience Program, and #Biology Department, Earlham College*

Around 273,000 people live with spinal cord injury (SCI) in the US, with ~12,000 new cases per year. Decreased quality of life and health care costs of up to $1million/person/year make SCI a devastating condition.

Lampreys are a well-characterized vertebrate model for SCI. Unlike higher vertebrates, lampreys exhibit spinal cord regeneration; 10-12 weeks after complete spinal transection they swim almost normally. New knowledge on lamprey spinal cord regeneration could help identify mechanisms for improved recovery from SCI in humans.

Voltage-gated sodium channels (NaV) allow neurons to create and propagate action potentials but excessive NaV activity after injury could cause hyper-excitability that can kill neurons. Lampreys recovering from SCI are resistant to NaV blockers, suggesting that their expression of NaV is decreased. Thus, decreased NaV expression may be an important for the survival and regeneration of spinal neurons following SCI.

This study assesses NaV expression in normal and transected lamprey spinal cords. We use immunofluorescence microscopy to visualize NaV in spinal nerve axons. To quantify expression, we asked an observer, uninformed about the experiment, to count the NaV-labeled axons in the micrographs. There was

a significant decrease after injury that started to recover after 11 weeks. We also performed behavioral studies to measure swimming ability and the effect of a NaV blocker, during recovery. The NaV blocker allowed slightly faster recovery. Thus, our preliminary data provide evidence of decreased expression of voltage-gated sodium channels in regenerating lamprey axons and a functional benefit of this decreased expression.

8. AN INVESTIGATION OF PERIRHINAL CORTEX FUNCTION IN FETAL ALCOHOL SPECTRUM DISORDER MODEL RODENTS

Nicole MacIlvane
Psychology Department, The Ohio State University

Fetal Alcohol Spectrum Disorder (FASD) results from maternal alcohol consumption during pregnancy. Offspring may exhibit growth disturbances and cognitive deficits. Our laboratory employs a FASD rat model in which neonate pups are exposed to ethanol across a time period comparable to the third trimester in humans (Bayer et al., 1993). We are currently interested in the neurotoxic effects of FASD on the perirhinal cortex (PR) and ventral hippocampus (VH) across development. As anxiety is mediated, in part, by the VH (Engin & Treit 2007), control and ethanol rats will first be exposed to the open field anxiety task during adolescence and adulthood. Control and ethanol adult rats will then be trained in the PR-dependent Novel Object Recognition (NOR) task, in which rats are exposed to two identical objects and then tested 20min or 4h later representing long term and short term memory, respectively. At test, rats must discriminate between the familiar and a novel object. Previous research from our lab has demonstrated FASD animals show impairments in long term memory (LTM) consolidation (Goodfellow & Lindquist, 2014). Therefore, we hypothesize FASD rats will show deficiencies when tested with the 4h, but not 20min, training-test interval.

9. NO DIFFERENCE IN AMYGDALA VOLUME IN YOUNG DEPRESSIVE WOMEN

Christina Gruenwald, Kelly McLean, & Cathy L. Pederson
Department of Biology, Wittenberg University

Depressive personality disorder is defined as a gloomy, negative, pessimist personality. The amygdala is the brain area associated with emotion and fear. We hypothesized that participants with depressive personality disorder will have a significantly larger amygdala volume than control participants. Participants were all right handed women (average age=27) with no serious medical complications. There was no difference between groups for age, alcoholic drinks per year, packs of cigarettes smoked per year, total joints of marijuana, and Wonderlic scores ($p>.05$). There was a significant difference of average sexual, physical, and emotional abuse scores between groups, $F(1,22)= 5.381$; $p=.03$. By design, there was a significant difference of MCMI Depressive

Scale scores between groups, $F(1,22)= 929.5$, $p<.001$. After tracing horizontal MRIs from a database on MIPAV, SPSS was used to analyze the data. Also, there was no significant difference of right amygdala volume between depressive and control participants with the covariate of average abuse, $F(2)=1.059$, $p>.05$. Although amygdala volume does not differ between groups, further research could be done on the activity of the amygdala between groups. A logical next step in this research would be to look at amygdala volume in a larger sample of women after controlling for the use of antidepressant medication.

10. DYNAMICAL SYSTEMS MODELS OF ODOR-TRACKING BEHAVIOR

Alexander J. Riordan[3,4,5], Peter W. Jones[2,5], Nathan N. Urban[2,5], G. Bard Ermentrout[1,5]
[1]Department of Mathematics, University of Pittsburgh; [2]Department of Biological Sciences, Carnegie Mellon University; [3]Department of Mathematics, Oberlin College; [4]Department of Neuroscience, Oberlin College; [5]The Center for the Neural Basis of Cognition, Pittsburgh, PA

The ability to sense and track noisy environmental chemical cues is essential for survival in a multitude of diverse species, from bacteria to mammals. Despite the biological importance of this capability, the mechanisms used by most organisms, and especially rodents, to follow chemical trails remain poorly understood. Hence, we developed precise, testable computational models of odor-tracking behavior to investigate the mechanisms that underlie this widespread behavior. We created models simulating tracking mechanisms based on spatial (internostril) and temporal (sniff-to-sniff) gradients of odor concentration. Models were simulated via numerical integration using XPP/XPPAUT, and their parameters were tuned to represent mice given an odor trail tracking task. When odor trails were represented in an idealized, noiseless form, simple spatial comparison models accurately tracked the trails, yet temporal models were only successful with additional, more sophisticated features. Models incorporating both spatial and temporal comparisons also accurately tracked the odor trail. To test how well each mechanism performed in more realistic, noisy odor-signal conditions, we quantified the behavior of each model using a broad range of initial conditions when locating and following noisy odor trails. A combination of spatial and temporal comparison strategies produced the best odor-tracking behavior tested. Notably, models relying on only spatial comparison were unsuccessful in tracking the odor trail in these noisy conditions. These investigations suggest that a combination of temporal and spatial odor-signal comparison mechanisms may be an essential physiological component of biological scent-tracking behavior.

11. POSTPARTUM ADMINISTRATION OF CITALOPRAM REVERSES GESTATIONAL STRESS-INDUCED DEPRESSIVE-LIKE BEHAVIOR AND STRUCTURAL MODIFICATIONS IN THE REWARD PATHWAY

Emily Mills, Achikam Haim, Chris Albin-Brooks, Morgan L. Sherer & Benedetta Leuner
The Ohio State University, Department of Psychology

Postpartum depression (PPD) is a common complication following childbirth experienced by approximately 20% of new mothers. Mothers diagnosed with PPD are often prescribed selective serotonin reuptake inhibitors (SSRI) antidepressants to ameliorate mood and other deleterious effects of PPD. We have previously shown that gestational stress, a risk factor for PPD, induces depressive-like behavior in rats and causes structural changes on neurons in the medial prefrontal cortex (mPFC) and nucleus accumbens (NAc), brain regions implicated in mood regulation. Here, we investigated whether the SSRI Citalopram can reverse the behavioral and morphological effects of gestational stress. In addition, we investigated the effects of gestational stress on structural plasticity in the basolateral amygdala (BLA), also implicated in mood regulation. Pregnant rats were stressed daily from gestational day 7 (GD7) to GD20 or handled daily for 5 min. Mother rats were subjected to the forced swim test on PD23 to assess depressive-like behavior and brains were collected 24 h later for Golgi staining and neural morphology analysis. Our results show that gestational stress induced depressive-like behavior during the late postpartum period and structural modifications in the mPFC, NAc, and BLA. Postpartum administration of Citalopram significantly reversed the depressive-like behavior and the structural modifications in the NAc and in the mPFC but not in the BLA. Overall, our results expand the validity of our gestational stress model by showing responsiveness to antidepressant treatment. Further, our results suggest that structural plasticity in the mPFC-NAc pathway may play a critical role in mediating depressive-like behavior in PPD.

12. HIPPOCAMPAL VOLUME NOT SIGNIFICANTLY INFLUENCED BY BODY MASS INDEX IN WOMEN

Keith S. George, Jessica Coggins, Cathy L. Pederson
Department of Biology, Wittenberg University

Obesity occurs when an individual weighs more than the target weight for his or her height. Obese individuals have been found to exhibit less N-acetyl-aspartate, an important indicator for neuronal health found in the hippocampus, than normal individuals, suggesting a link between obesity and hippocampal volume could exist. To investigate if individuals with high BMIs have smaller hippocampal volumes than people with low BMIs, a group of 24 right handed women with no history of serious medical complications was divided into to a high (BMI ≥ 30) or low (BMI ≤ 21) BMI group $F(1, 22) = 358.743$, $p=0.001$. Groups were homogenous when comparing average abuse

scores, pack X years smoking, Wonderlic personnel scores, drinks per year, and age ($p>0.05$). Every other horizontal MRI image containing a portion of the left hippocampus was traced twice using the MIPAV computer software and averaged to give the final area. The areas of all the tracings from a single brain were added and then multiplied by 2 to give the final hippocampal volume. A univariate ANOVA was performed in SPSS to indicate that there is no significant difference between hippocampal volume for the two groups $F(1,22) = 0.008$, $p=0.928$. Therefore, no evidence was found suggesting that a relationship between BMI and hippocampal volume existed in this small sample of women.

13. Navigating the Adolescent Maze: The Impact of a High Fat Diet on Brain Chemistry in Rats, a Pilot Study

Kyle A Johnston & Steven L Neese
Psychology and Neuroscience, Baldwin Wallace University

Adolescent obesity rates have more than tripled in the past 30 years, yet little is know about the effects obesity has on the developing brain. This study examined the effects of exposure to a high fat diet (HFD) or a low fat diet (LFD) during adolescence in male rats. Dietary exposure began on postnatal day (PND) 21 and spatial memory was assessed 4 weeks later in the Morris water maze (MWM). Following MWM testing, rats were humanely sacrificed and hippocampi were extracted (PND 56). Tyrosine hydroxylase (TH) levels were detected using a western blot procedure. A t-test found a marginally significant difference in TH/β-actin ratio between HFD and LFD groups, $t(5)=4.87$, $p=0.06$. TH/Actin expression was decreased in the HFD group. Analysis of the MWM data failed to uncover a difference in performance between the HFD and LFD groups. These findings contribute to the understanding of the effects of exposure to a diet high in fat on the developing adolescent brain.

14. Oxytocin in the Prelimbic Medial Prefrontal Cortex Reduces Anxiety-Like Behavior in Female and Male Rats

Jared Krein, Benedetta Leuner
Department of Psychology, The Ohio State University

The neuropeptide oxytocin (OT) has anxiolytic effects in rodents and humans. However, the specific brain regions where OT acts to regulate anxiety requires further investigation. The medial prefrontal cortex (mPFC) has been shown to play a role in the modulation of anxiety-related behavior. In addition, the mPFC expresses OT receptors and receives long range axonal projections from OT-producing neurons in the hypothalamus, suggesting that the mPFC may be a target where OT acts to diminish anxiety. To investigate this possibility, females rats were administered OT bilaterally into the prelimbic (PL) region of the mPFC and anxiety-like behavior assessed. To determine if the effects of OT on anxiety-like behavior are sex dependent and to evaluate the specificity of

OT, male and female anxiety-like behavior was tested following delivery of either OT or the closely related neuropeptide arginine vasopressin (AVP) into the PL mPFC. Finally, the importance of endogenous OT in the regulation of anxiety-like behavior was examined in male and female rats that received PL infusions of an OT receptor antagonist (OTR-A). Overall, even though males and females showed some differences in baseline levels of anxiety-like behavior, OT in the PL region of the mPFC decreased anxiety regardless of sex. In contrast, neither AVP nor an OTR-A affected anxiety-like behavior in males or females. Together, these findings suggest that although endogenous OT in the PL region of the mPFC does not influence anxiety, the PL mPFC is a site where exogenous OT may act to attenuate anxiety-related behavior independent of sex.

15. IMPAIRED PAVLOVIAN CONDITIONING AND ALTERED FOREBRAIN N-METHYL-D-ASPARTATE RECEPTOR SUBUNIT COMPOSITION IN A RAT MODEL OF FETAL ALCOHOL SPECTRUM DISORDER

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Fetal Alcohol Spectrum Disorder (FASD), resulting from gestational alcohol consumption, is marked by physical and mental abnormalities, including persistent impairments in learning and memory. In this study, rat pups were intragastrically intubated with ethanol (5E rats; 5g/kg/day) or sham intubated across postnatal days 4 to 9. Adult 5E rats were significantly impaired in trace fear conditioning (TFC), a Pavlovian conditioning paradigm in which the conditioned stimulus (tone) and the unconditioned stimulus (footshock) are separated by a stimulus-free interval. TFC requires forebrain *N*-methyl-D-aspartate receptor (NMDAR) activation. NMDARs contain four subunits: two mandatory NR1 subunits and two regulatory NR2 subunits. When activated, NR2B-containing NMDARs gate more calcium than NR2A-containing NMDARs. Calcium acts as a second messenger in a molecular signaling cascade that contributes to the maintenance of long-term potentiation (LTP), a learning-mediated enhancement of neural signaling. Thus, NR2B subunits are proposed to enhance learning-dependent synaptic plasticity more so than NR2A, and ethanol-induced alterations to NMDAR subunit composition are proposed to disrupt LTP maintenance. In support, whole cell lysate immunoblotting revealed an elevated NR2A/NR2B ratio in dorsal hippocampus, but not ventral hippocampus or medial prefrontal cortex of 5E rats. Utilizing subcellular fractionation, synaptic subunit immunoblotting showed a significant reduction in NR2B subunits in dorsal hippocampus of 5E rats, which is proposed to impede the synaptic plasticity required for successful TFC. Results are expected to provide new and valuable knowledge regarding the etiology of FASD, and

may lead to the use of novel pharmacological therapies targeting NMDARs to ameliorate cognitive deficits in FASD individuals.

16. AN INVESTIGATION INTO LEARNING-DEPENDENT ALTERATIONS IN NMDA RECEPTOR DOWNSTREAM SIGNAL TRANSDUCTION IN A RAT MODEL OF FETAL ALCOHOL SPECTRUM DISORDER

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Fetal alcohol spectrum disorder (FASD) is an umbrella term encompassing several illnesses caused by prenatal alcohol exposure. We use a rat model of 3rd trimester-equivalent exposure in which rats are intragastrically intubated with 5 g/kg/day ethanol (5E) or sham intubated (SI) across postnatal days 4-9. 5E rats have been shown to be impaired in trace fear conditioning (TFC) (DuPont et al., 2014; Hunt et al., 2009), a dorsal hippocampus (DH)-dependent Pavlovian conditioning paradigm (Kopp et al., 2007). This impairment could be caused, in part, by altered N-methyl-D-aspartate receptor (NMDAR) subunit composition which may make the induction of long-term potentiation (LTP), a form of synaptic plasticity, more difficult (Shouval et al., 2002). NMDAR activation contributes to LTP through a signal transduction pathway that, early on, involves the activation of calcium/calmodulin-dependent protein kinase II (CaMKII) causing it to be autophosphorylated and therefore able to be continually activated even in the absence of further excitation. Later, this pathway results in the insertion and phosphorylation of GluR1 (pGluR1), an α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor (AMPARs) subunit involved in long-term memory consolidation. We intend to measure pGluR1 and phosphorylated αCaMKII in 5E and SI rats using immunofluorescent (IF) detection in DH. This investigation will provide valuable information regarding the signaling pathways that could play a role in the learning impairments observed in alcohol-exposed rats. This research is expected to contribute to the development of more effective pharmacotherapies for fetal alcohol related cognitive disorders.

17. PHYSICAL AND EMOTIONAL ABUSE VICTIMS SHOWED NO DIFFERENCE IN AMYGDALA VOLUMES COMPARED WITH CONTROL

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The amygdala is a region in the brain that regulates emotions. Researchers have found that victims of abuse have reduced amygdala volumes compared to non-abused participants. We conducted a study to investigate the change in amygdala volume of young females between the age of 20 and 38 years who were victims of physical and emotional abuse. We hypothesized that the amygdala would be smaller for the abused compared with the non-abused

participants. There was no difference for potentially confounding variables such as age, drinks per year, pack years smoking and the Wonderlic Personnel test, ($p>0.05$). By design, the level of abuse differed significantly among groups $F(1,23)=228.73$; ($p<.001$). Magnetic Resonance Images (MRI) were examined to measure the right amygdala area for each participant, which were then averaged, summed into a total volume, and compared between participants through an univariate analysis of variance. Amygdala volumes were smaller but not significantly different in abuse victims compared with controls, ($p>0.05$). However, this research indicates that people with a history of physical and emotional abuse may have decreased amygdala volumes. Future studies will include a larger population in order to determine whether there is indeed a trend toward smaller amygdala volumes in abused patients, as has been found in other studies.

18. Effect of Perinatal Fluoxetine Exposure on Autism-Related Social Behavior in Mice

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Autism Spectrum Disorder (ASD) is characterized by social deficits and repetitive behaviors. Modification of serotonin levels early in life has been implicated to produce symptoms typical of the disorder. Recent studies have found evidence suggesting maternal SSRI use during pregnancy has resulted in an increased risk of ASD in their children. We examined the effects of chronic maternal fluoxetine treatment on offspring using a three-chamber test for sociability and social novelty. Open field tests for rearing events and total motor activity were also used. When exposed perinatally to fluoxetine, mice displayed social impairments as well as deficits in non-selective attention. These results support previous human population studies finding an association between mothers on antidepressants during pregnancy and children with ASD. These results implicate a risk when pregnant women seek treatment for depression. Women should be informed of this risk and given other treatment options to ensure the health of their children.

19. Rock out with you Proboscis Out: Bringing Red-Shifted Optogenetic Control of Drosophila Behavior to the Classroom

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Optogenetics enables the manipulation of neural activity in free moving organisms with millisecond precision by making modified ion channels sensitive to a particular wavelength of light. Unfortunately today, most optogenetics methods are expensive and out of reach beyond well funded institutions. This

is regretfully common within the field of neuroscience and most students aren't exposed to any neuroscience concepts until graduate education.

Making neuroscience methods such as optogenetics available in grade-school classrooms is important for introducing students to the excitement of neuroscience. Neuroscience is a rapidly growing field and bringing the most cutting edge methodologies, such as optogenetics, to the classroom will play a role in expanding the field of neuroscience in the future.

Using a red-shifted opsin (ReaChR) that has been developed to allow light to penetrate through the exoskeleton of insects and activate target cells, I will develop protocol and experiments using affordable tools and materials to observe which neural pathways are involved in the Proboscis Extension Response(PER).

Creating new tools for understanding the systems behind animal behaviors is important not only because it can inspire interest in neuroscience and encourage critical inquiry in youths, but to eventually gain a further understanding of the mechanisms of neural substrates similar to those of humans via such animal models.

20. Binge-like exposure of ethanol in a Fetal Alcohol Spectrum Disorder rat model effects medial Prefrontal Cortex dependent Context Object Recognition task

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Fetal Alcohol Spectrum Disorder (FASD) yields mental and physical abnormalities in offspring caused by maternal consumption of alcohol during fetus development. Our FASD rat model mimics binge-like ethanol exposure during a human third trimester-equivalent period, postnatal days 4-9. Via intragastric intubation, rats receive either 5 g/kg/day ethanol (5E) or a sham intubation (SI); unhandled control (UC) rats had no exposure to intubations. This model is known to impair cognition, particularly in tasks requiring the hippocampus. Less is known regarding the function of the medial prefrontal cortex (mPFC), which we test using a mPFC-dependent context-object recognition (COR) task. On days 1 and 2, rats were habituated to both Context A and Context B for 10 minutes each with no objects present. On Day 3, animals were trained in Context A with an Object A pair present, and again in Context B with an Object B pair. Twenty minutes later animals began COR testing—one each of Object A and Object B were presented in a context and time spent exploring each object was recorded. An animal free of deficit would show enhanced exploration of the context-mismatched object relative to the context-matched. As expected, context-mismatched object exploration was greater in all test groups relative to matched objects. SI performance in context-mismatched object exploration after 5 minutes was low relative to the

other test groups, however, 5E and UC context-mismatched object exploration after 5 minutes showed no significant differences, suggesting ethanol exposure did not impair the mPFC.

21. NPAS4 AND VULNERABILITY TO PRENATAL STRESS: IMPLICATIONS FOR IMPAIRED MATURATION OF THE GABAERGIC SYSTEM

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Delayed maturation of the GABAergic system is implicated in the etiology of several neuropsychiatric disorders. Maturation processes of GABA involve the chloride co-transporter KCC2. Prenatal immune activation such as that induced by lipopolysaccharide (LPS) is known to alter KCC2 expression, resulting in social and cognitive impairments. The mechanisms behind such changes remain unknown. LPS is also known to alter adult expression of Npas4, an activity-dependent transcription factor highly involved in GABA transmission. We suggest that Npas4 mediates the effects of LPS on GABAergic maturation. To test this idea, LPS or vehicle is administered to C57/Bl6 female mice on gestation day 16. KCC2 and Npas4 expression are assessed in offspring during early postnatal development. We expect to observe a correlation between LPS-induced reduction of Npas4 and KCC2 expression. To confirm the role of Npas4 in LPS-induced GABAergic impairment, Npas4 heterozygous (HET) pregnant mice will be injected with either LPS or vehicle. Offspring will be tested for social and cognitive functions and KCC2 expression will be assessed. We expect HET offspring to have more severe behavioral deficits and KCC2 alteration than wild type mice. With this study we aim to advance our understanding of the etiology of neuropsychiatric disorders.

22. SHOCKS AND STINGS: MICROSTIMULATION OF SCORPION DEFENSIVE BEHAVIOR

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A scorpions? experience of the world is primarily dependent upon its sense of touch, much of which is localized to sensory organs on their legs. Previous studies on scorpions have indicated that receiving direct tactile stimulation to their legs induces a defensive behavior-either stinging, or snapping with their pedipalps, in the perceived direction of the stimulation, or movement directly away from the perceived stimulus. In this study, different species of scorpions had their defensive responses evoked and examined. The scorpion surgery includes interfacing implanted wire electrodes with the leg nerves, which are then paired with an external function generator. The scorpions received 2.5 V of 55 Hz AC for 150 ms, at differing legs, and their resultant behavior relative to the point of stimulation was recorded. The prediction is that those scorpions with thicker pedipalps will reliably sting or claw to the side the electrical

stimulation is provided, and those with thinner pedipalps. Little is understood about scorpion defensive behavior and neurophysiology, and this is being used as a means to explore both subjects.

23. THE EFFECTS OF PERMANENT DAMAGE TO THE RETROSPLENIAL CORTEX ON CONTEXTUAL LEARNING IN RATS

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The retrosplenial cortex (RSC) provides sensory information to the hippocampal memory system in rodents and primates and is thought to be important for the formation of memories pertaining to a particular environment. This experiment tests the hypothesis that RSC may be involved in the formation of stimulus-stimulus associations; a component of hippocampal-dependent learning. In this experiment, rats received either permanent electrolytic lesions of the RSC or sham surgery and were subsequently tested for the ability to form stimulus-stimulus associations using an aversive learning and memory paradigm. During the first training phase of the procedure one auditory stimulus was presented in Context A whereas a second auditory stimulus was presented in Context B. In the second training phase, one auditory stimulus was paired with footshock in Context C whereas the second auditory stimulus was presented in Context C, but was not paired with footshock. During the context test phase, rats were exposed to Context A and B and freezing levels were evaluated. During a fourth and final phase, freezing behavior in response to the auditory stimuli was evaluated while rats were in Context C. Control rats exhibited more freezing behavior in the context where they heard the auditory cue that was paired with footshock compared to the context where they heard the unpaired cue, indicating that stimulus-stimulus associations had been formed during the first phase of training. In contrast, RSC-lesioned rats failed to show context discrimination, but maintained the ability to discriminate between the individual auditory stimuli. These data support the notion that RSC is involved in forming associations between neutral stimuli even in the absence of reinforcement.

24. ESTRADIOL AND LUTEINIZING HORMONE AFFECT BRAIN-DERIVED NEUROTROPHIC FACTOR LEVELS IN OVARIECTOMIZED FEMALE RATS

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As women age, estradiol levels decrease and Luteinizing Hormone (LH) levels increase. Age-related memory loss has been associated with these changing levels of hormones yet the way in which these hormones exert their effects on the brain is still unclear. It was hypothesized that low-LH, high-estradiol conditions lead to cognitive health, specifically higher levels of neurotrophic

factors and cell proliferation. To test this hypothesis, female rats were ovariectomized and implanted with either an estradiol or blank capsule. After four days of recovery, the estradiol animals were injected with the LH homologue hCG or vehicle, and the blank animals with the LH blocker Antide or vehicle. Immunocytochemistry was used to detect levels of brain-derived neurotrophic factor (BDNF: Alomone Labs anti-BDNF antibody 1:500 dilution) and Ki67 (anti-Ki67 antibody 1:750 dilution), a nuclear marker of proliferation. Significantly higher levels of BDNF were found in the dentate gyrus of estradiol-implanted/vehicle females compared to blank/vehicle. Levels of BDNF were not significantly different in estradiol/vehicle and estradiol/hCG females. Significantly higher levels of BDNF were found in the blank/Antide-injected females compared to blank/vehicle, suggesting higher levels of BDNF in low-LH conditions. No significant difference in Ki67 cell counts was found between estradiol females and blank. These results suggest that low-LH, high-estradiol conditions may improve cognitive health by affecting neurotrophic factors rather than the proliferation of new cells.

25. THE EFFECTS OF KETAMINE ON LEARNING AND SPATIAL MEMORY IN JUVENILE RATS

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During early childhood, the brain is susceptible to environmental changes which may result in cell death. Ketamine is an anesthetic agent used in children (< 4 years), which has shown to cause increased apoptotic cell death in the hippocampus in neonatal rats (P7). Previous studies indicate that the Ketamine-induced apoptosis occurs with binding of Ketamine to N-methyl-D-aspartate (NMDA) receptors which disrupts the neural uptake of calcium. The aim of this study was to determine if ketamine adversely effects spatial memory or reasoning in neonatal rats. A prospective trial was conducted utilizing 25 Sprague-Dawley rats divided into a control and experimental group. On P7 animals were administered four subcutaneous doses of ketamine (20 mg/kg) or an equivalent amount of sterile saline. At P25, both groups were tested using the Morris Water Maze to assess their spatial learning and memory. During training days, each rat had four trials to locate the escape platform before the final probe trial when the platform was removed from the pool. The average time spent in the platform-containing quadrant once the platform was removed was 30.74 seconds for the control and 28.30 seconds for the experimental rats (P= 0.792). Data suggests that learning for both groups was variable. Early exposure to ketamine did not alter ability of the animals to learn using spatial memory in the Morris Water Maze. Therefore a loss of neurons early on may be compensated by new neurogenesis. Additionally other forms

of learning might be compromised and will be studied with other learning tasks.

26. NON-INVASIVE NEUROMODULATION TO IMPROVE MEMORY IN ALZHEIMER'S DISEASE

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The default mode network (DMN) is complex brain system that involves anatomically and functionally connected brain areas, which functions when the mind is conscious but not engaged in an active task. One important connection of the DMN is to the hippocampus, which is the center for memory consolidation. This connection helps the DMN carry out functions such as remembering past experiences. This also led to the discovery that the functioning of the DMN was crucial in many brain disorders such as Alzheimer's Disease. The current project targeted two particular DMN regions (medial prefrontal cortex and medial parietal cortex) and hypothesized that stimulating them simultaneously would indirectly activate the hippocampus, and could help improve memory in Alzheimer's Disease patients. This study was a pilot study, carried out with 7 healthy controls. In order to see whether the dual TMS caused increased hippocampal activation, we took fMRI scans of the subjects before and after stimulation. Comparison between the two scans showed that there was greater parahippocampal activation post-stimulation as compared to before stimulation. It lends support to our hypothesis, and a larger subject pool would help further verify our claim.

27. THE EFFECTS OF INTERMITTENT QUETIAPINE ADMINISTRATION ON BEHAVIORAL OUTCOMES AFTER EXPERIMENTAL TBI

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Agitation and aggression displayed by patients after traumatic brain injury (TBI) pose major obstacles to clinicians in the acute hospital and rehabilitation settings. These symptoms are commonly managed with sedatives and antipsychotic drugs, such as haloperidol and risperidone. However, previous preclinical studies have indicated that chronic administration of these drugs can exacerbate cognitive and motor deficits resulting from TBI. Research employing quetiapine, an atypical antipsychotic, in the management of psychosis in schizophrenic and Parkinson's patients has suggested that the drug has no deleterious effects on motor performance and can enhance cognition and spatial learning. Hence, this experiment aimed to test the hypothesis that chronic administration of quetiapine would not hinder recovery of cognitive and motor function after TBI. Forty-five male rats were assigned to one of six

groups combining a TBI (via controlled cortical impact) or sham injury and treatment with haloperidol (0.5 mg/kg, i.p.), quetiapine (10 mg/kg, i.p.), or vehicle (DMSO + saline; 1.0 mg/kg, i.p.). Treatments were administered every other day immediately following behavioral testing. Motor function was assessed by beam balance/walk tests on post-operative days 1-5 and cognitive function was assessed using the Morris water maze task on days 14-19. In all tasks, the SHAM group was significantly better than all TBI groups, regardless of treatment. Furthermore, there were no significant differences between the TBI + vehicle and TBI + quetiapine groups. This indicates that quetiapine does not exacerbate TBI-induced cognitive and motor deficits, and may thus prove useful as an alternative antipsychotic treatment for management of agitation and aggression following TBI.

28. PRELIMINARY EVIDENCE THAT THE OLFACTORY TUBERCLE IS SUFFICIENT TO DRIVE NEURAL ACTIVITY IN THE TASTE CORTEX, BUT NOT NECESSARY FOR FLAVOR-GUIDED BEHAVIOR

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The olfactory tubercle (OT) was first described in 1896. There surprisingly has not been much research on the functional properties of the OT. Early research has demonstrated the OT is anatomically connected with numerous brain structures. Through our research, we found a novel link between the OT and the primary taste cortex, the agranular insular cortex (AI). To explore whether this connection was monosynaptic versus polysynaptic, we used virally mediated tracing techniques. No direct monosynaptic input was observed following AAV-mcherry injections into the AI.

In addition, we examined if OT-AI connectivity had any influence on flavor perception. After inactivating neurons in the OT using virally-mediated expression of inhibitory channels, we established a two-bottle taste preference test, based on previous literature, to determine if the connection between the AI and OT is indeed vital for flavor perception. In a subpopulation of mice with confirmed infusions of DREADDs into the OT, no effect of OT inactivation on flavor preferences was observed.

29. KAPPA OPIOID REGULATION OF DEPRESSIVE-LIKE BEHAVIOR DURING ACUTE AND PROTRACTED WITHDRAWAL FROM ETHANOL

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Alcohol withdrawal can create short- and long-term changes in the brain's physiology, which may lead to mood disorders such as depression. Though the short-term impact of withdrawal has been well characterized, much less is known about the long-term changes in brain function and behavior resulting

from prolonged abstinence from alcohol. Recent studies involving animal models of alcoholism have found that changes in the dynorphin (DYN)/kappa opioid receptor (KOR) system may play a role in regulating the symptoms of depression and other mood disorders. The aim of the current study was to examine the role of the DYN/KOR system on depressive-like behavior in the forced swim test during acute and protracted withdrawal from alcohol. Ethanol dependence was induced in male Wistar rats via liquid diet administration. During acute withdrawal, ethanol dependent animals showed increased immobility compared to controls, which has been proposed to indicate a depressive-like state. This effect was reversed following injection of the KOR antagonist nor-binaltophimine (nor-BNI). However, during protracted withdrawal, no significant differences were observed between ethanol dependent and non-dependent animals, although this result was likely due to a supply shortage of nor-BNI at the time of testing and small sample size. The results observed during the acute withdrawal suggest that the DYN/KOR system may play a role in mediating depressive-like behavior during short-term withdrawal, and experiments are currently in progress to address the shortcomings of the protracted withdrawal condition to more fully understand the role of the KOR/DYN system following long-term abstinence from alcohol.

30. Treatment of Compression Spinal Cord Injury in Guinea Pigs Using Methylprednisolone and 6-Chloro-Tryptophan

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Spinal cord injury is a debilitating condition which can cause severe loss of motor or sensory function in humans. This loss of function results from the primary deficits caused by the initial injury and the secondary deficits due to the pathological processes mediated by the inflammatory immune response. We studied the treatment efficacy of two drugs in combination to attenuate the secondary pathology of spinal cord injury in guinea pigs. Methylprednisolone (MP) is the only drug currently approved for acute spinal cord injury in humans. The administration of MP has been found in past research to lead to long-term motor and sensory improvements in spinal cord injured guinea pigs. The other treatment is 6-Chloro-Tryptophan (6-Cl-Tryp), which is not yet approved for human use. 6-Cl-Tryp is a precursor to 4-Chloro-3-hydroxyanthranilate, which has been found to reduce quinolinic acid production. This decrease in quinolinic acid accumulation reduces the secondary deficits of spinal cord injury in guinea pigs. To test the effects of the combination of these two drugs, we performed a lateral compression injury at thoracic level 12. The animals were divided into treatment groups using four combinations of MP and 6-Cl-Tryp or their vehicles. MP or its vehicle was administered at 60 mg/kg at 0.5 hours post-injury and at 30 mg/kg at 2, 4, and

6 hours post-injury. 33 mg 6-Cl-Tryp or its vehicle was administered at 5 hours post-injury, and then every 12 hours for 12 days. Functional deficits were quantified by simple motor and sensory tests such as proprioceptive placing, toe spread, and *cutaneus trunci* muscle response, and complex tests, such as incline plane, contact righting, and air righting. Our preliminary results indicate stronger trends in attenuation of moderate spinal cord injury in guinea pigs using 6-Cl-Tryp and MP.

31. CITRULLINATION INCREASES WITH AGE IN A UNIQUE ALZHEIMER'S MODEL

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Citrullinated proteins of are the product of a post-translational modification of arginine amino acid residues, catalyzed by calcium-activated peptidylarginine deiminase (PAD). During neuro-degeneration increased PAD enzyme leads to citrullination of myelin basic protein, GFAP and vimentin. For instance GFAP is citrullinated in the hippocampus of AD mouse models that overexpress Tau or APP. In addition to the hippocampus, the entorhinal cortex may be one of the earliest brain regions affected in AD. In order to determine if proteins are citrullinated in the entorhinal cortex, we measured citrulline in a unique AD mouse model which overexpresses a truncated APP or AICD (amyloid precursor protein intracellular domain) in the entorhinal cortex using a mouse model. These mice begin to express AD pathology around 8-9 months of age. Brains were collected for immunohistochemistry with peptidyl-citrulline antibodies at ages 3, 8, 9 and 18 months. Citrullination is increased in AICD mice compared to controls at 9 and 18 months. Thus citrullination increased with age in the entorhinal cortex in AICD overexpressing mice.

32. INHIBITION OF PAD 2 AND ITS IMPLICATIONS IN NEURONAL DEVELOPMENT

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Peptidylarginine deiminase 2 (PAD 2) converts arginine residues in myelin basic protein (MBP) into citrulline. An increase in citrullinated MBP is present in multiple sclerosis patient nervous tissue and may have a role in the disease. In addition to an increase in disease, PAD 2 is a protein that is present early in development but the importance of this enzyme during development is unknown. Since PAD 2 is present early in development and then re-emerges during disease states, the role of PAD 2 during this critical stage might provide insight into this increased expression. In order to determine the role of PAD 2 in early development, we inhibited PAD 2 in zebrafish embryos (2-24 hpf) with 2-chloroacetominophen (0.5 mM, 1.0 mM, 1.5 mM). The embryos (24 hpf)

were fixed and stained for acetylated-tubulin to mark the nervous system. It was determined that zebrafish treated with 2-chloroacetominophen had fewer Rohon Beard cells, early sensory neurons, compared to controls. Rohon Beard (RB) cells are a transient population and generally undergo apoptosis at 5 days post fertilization (dpf). After RB cell death, dorsal root ganglia develop, therefore, the PAD 2 enzyme or citrullination may be involved in neuronal differentiation of sensory neurons.

33. CRH Expression is Increased in an Animal Model of Visceral Hypersensitivity

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Irritable bowel syndrome (IBS) is a common functional gastrointestinal disorder characterized by visceral hypersensitivity and altered bowel habits. IBS is the second most commonly encountered diagnosis in clinical practice and is a $21 billion economic burden with 2.2 million prescriptions written annually. Impairment of the brain-gut axis is commonly associated with IBS, as fifty percent of IBS patients also suffer from behavioral disorders including depression and anxiety.

Serotonin, corticotropin releasing hormone (CRH) signaling and gender may contribute to IBS pathology. Many IBS patients display genetically determined deficits in serotonin transporter (SERT) function which increases the availability of serotonin in the gut. Additionally, CRH signaling is associated with both behavioral disorders and IBS. IBS is twice as prevalent in females as in males. SERT knockout (KO) rats serve as an animal model for studying the relationship between gender, serotonin and visceral sensitivity. Female SERT KO rats display sex-specific increases in visceral hypersensitivity and colonic serotonin associated with colon-projecting sensory neurons. Coincidentally, dorsal root ganglia (DRG) from SERT KO animals exhibit a 117-fold upregulation in CRH mRNA.

We compared CRH protein expression in the DRG of SERT KO and wildtype (WT) rats using immunohistochemistry. We measured the size of CRH-containing neurons and colon projecting neurons using image analysis software. Our results indicate that female SERT KO rats display significantly greater CRH expression ($p < 0.0001$) than female WT. CRH-expressing neurons are significantly smaller ($p < 0.05$) in cell body diameter than non-CRH expressing neurons.

34. The Role of Indirect PAR1 Activation in Tissue Repair after SCI

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PAR1 (Protease-Activated Receptor 1) is a G protein-coupled receptor present on many different CNS cell types. When cleaved by serine proteases, PAR1

initiates several signaling cascades associated with tissue repair. After spinal cord injury (SCI), PAR1 is upregulated within the damaged tissue. We hypothesized that PAR1 activation would promote the proliferation of oligodendrocyte progenitor cells (OPC's), which are known to play a role in repair after SCI. Indeed, activation of PAR1 within the spinal cord via microinjection of a PAR1 agonist promoted an increase in OPC proliferation, accompanied by an acculumation of microglia surrounding the microinjection site. However, when pure OPC cultures were treated with the same agonist, PAR1 activation had no effect. This suggests that other cell types in the *in vivo* microenvironment may indirectly promote the OPC response. Our goal is to determine if the activation of PAR1 on microglia or astrocytes will promote the proliferation of OPC's *in vitro*. OPC's will be co-cultured with microglia and astrocytes, followed by activation of glial cells by PAR1 agonist. Through quantificaiton of these mixed glial cultures, we hope to determine if there is indeed a relationship between PAR1 activated microglia/astrocytes and the proliferation of these progenitor cells. This data may help elucidate the role of PAR1 in the regeneration of damaged spinal tissue and therefore repair after SCI.

35. INVESTIGATING THE NEUROMODULATORY ROLE OF ALPHA-SYNUCLEIN IN HEAVY METAL TRANSPORT AND HOMEOSTASIS

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Neuroscience Department, Oberlin College

Alpha-synuclein(a-syn) is a small soluble protein expressed primarily in the central nervous system. Mutations in a-syn are known to cause Parkinson's disease (PD), which is characterized by motor, cognitive and psychiatric abnormalities. Despite the paucity of information on the function of normal/wild-type a-syn protein, there has been evidence to support the accumulation and aggregation of both wild-type and mutant as a pathological hallmark in PD. In fact, heavy metals are implicated in the pathophysiology of PD. However, the neuromodulatory role of wild-type or mutant a-syn in metal transport and homeostasis dynamics are poorly understood. Here, we utilized an established mesencephalic dopaminergic cell line expressing human wild-type a-syn (N27-syn) or empty vector (N27-vec) to conduct a gene-metal screen aimed at uncovering the neuromodulatory functions of wild-type a-syn in the attenuation and potentiation of mitochondrial dependent cell viability. Our preliminary data suggest that expression of human wild-type a-syn enhances selenium (Se) induced dopaminergic toxicity in N27-syn compared to N27-vec cells. However, human wild-type a-syn protects dopaminergic cells from copper (cu^{2+}), manganese (Mn^{2+}) and cobalt (Co^{2+}) neurotoxicity in a dose-dependent manner. On going experiments are to understand if the aforementioned differences in cell viability are directly related to a-syn neuromodulation of

metal transport and homeostasis. In addition, we seek to investigate the subcellular localization and induction of a-syn aggregation and degradation pathways following heavy metal exposure.

36. Investigating the neurotoxic effect of agrochemicals (dieldrin and lindane) in Huntington's disease neuropathology

Anne Chege & Gunnar Kwakye
Neuroscience Department, Oberlin College

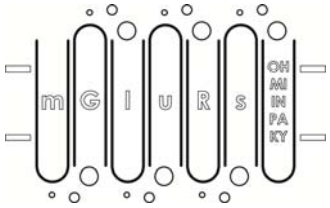
Huntington's disease (HD) is a genetic neurodegenerative disease that results in movement, cognition, personality and mood impairments. The striatum, necessary for normal motor function, is mostly affected in HD. In this study, we investigated the effects of two potentially exposed pesticides, dieldrin and lindane in HD neuropathology. Dieldrin and lindane are organochloride pesticides reported to accumulate in Parkinson's disease (PD) postmortem brain tissues and cause dopaminergic cell loss. Recognizing the similarities in pathophysiological mechanisms between PD, HD and pesticide neurotoxicity, we hypothesized that exposure to lindane and dieldrin may potentiate the neurotoxic properties of mutant HD protein, causing enhanced striatal neurodegeneration. We examined the effects of lindane and dieldrin independently and cooperatively in an established mouse striatal cell model of HD expressing 7 (wild-type) or 111 (mutant) polyglutamine repeats. Following exposures of wild type and mutant HD striatal cells to varying concentrations of dieldrin or lindane at different time points, we observed that mutant HD striatal cells exhibited a time-dependent toxic gain of function and decreased mitochondrial-dependent cell viability compared to wild type. However, we report no genotypic and time-dependent differences in survival upon lindane exposure. Interestingly, preliminary results suggest that dieldrin and lindane cooperatively potentiate striatal HD neurotoxicity. In summary, we have uncovered a novel disease-toxicant interaction between mutant HD and dieldrin. Ongoing experiments will examine the effects of dieldrin induced oxidative stress and protein kinase C delta mediated cell death pathway. This research hopes to uncover and further the understanding of gene-environment interaction that may modify HD neuropathology.

37. A Disease-Toxicant Interaction Reveals Cadmium Exposure as a Potential Modulator in Huntington's Disease Neuropathology

Edmund Korley & Gunnar Kwakye
Neuroscience Department, Oberlin College

Huntington's disease (HD) is an congenital neurological disorder characterized by selective neuronal loss in the striatum of the brain. A genetic mutation in the HTT gene results in overproduction of glutamine in the HTT protein, motor, psychiatric, and cognitive impairments. The HTT protein is ubiquitously

expressed in the nervous system with critical functions in neurodevelopment and metal transport. In spite of the genetic cause of HD, there is emerging evidence that suggests that environmental factors may contribute to the variability in age of onset, disease progression and symptomology. Interestingly, heavy metal neurotoxicity and HD cellular pathology share similarities in cellular targets including dysregulated metal homeostasis, protein aggregation and oxidative stress. Thus, our overarching research goal is to understand the basis of selective neuropathology of HD by leveraging cadmium neurotoxicity sharing common modes of neuropathology. Here we report that expression of mutant HD protein enhances cadmium neurotoxicity in a dose-dependent manner compared to healthy striatal HD cells. Recognizing the substantial number of heterozygous HD patient cases, we investigated the effect of Cd exposure on metabolic capabilities and mitochondrial function in heterozygous striatal HD cells. Interestingly, expression of one mutant and wild-type HD alleles changes the neuroprotective functions of wild-type HD protein and significantly potentiates striatal neurotoxicity when compared to wild-type or mutant HD striatal cells. Furthermore, we examined the production of free radicals in the HD striatal cells and report a genotypic-dependent potentiation of oxidative stress in HD striatal cells following Cd exposure. We hypothesize that expression of mutant HD protein causes a toxic gain of function that alters Cd transport dynamics and homeostasis via increased influx/uptake and cellular storage or decreased efflux/export mechanisms compared to healthy cells. Ongoing experiments are aimed to better understand cadmium transport kinetics in striatal HD cells. Understanding the disease-toxicant interaction (HD-Cd) will hopefully elucidate the prime environmental modulator in HD neuropathology and contribute to future therapeutic targets.



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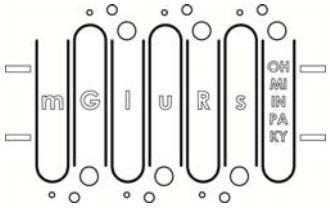

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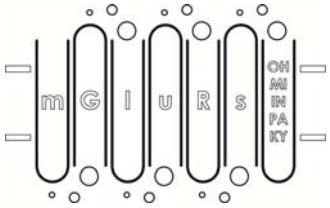

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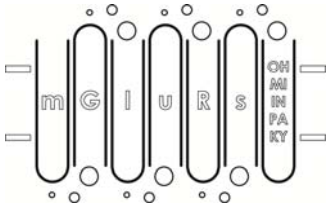

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RESTAURANT SUGGESTIONS

In town:

- Little Mexico, 211 E. Main St. – take Wabash east to Water, left, left on Main (Main is one-way towards campus)
- Creekside Lodge, 613 Lafayette Ave. – steaks and pizza, on Sugar Creek – head north on Grant, cross Market, Lafayette Rd. is in a little valley, go left. If you cross the creek and get to UPS, you've just overshot.
- Yamato, 1885 US Hwy 231 South (by Culver's and Walmart) – sushi, finally, in Crawfordsville!

In Indianapolis, on 38th St. (~45 min from campus):

(Take I-74 towards Indy, then north on 465 one exit, right/east on 38th St.)

- Abyssinia Ethiopian restaurant, 5352 W 38th St, north side of 38th (behind Ginza)
- Ginza sushi, 5380 W 38th St, north side of 38th

Just past Indianapolis (~1 hour from campus):

- Exit 109 from I-70 has lots of restaurants – Bob Evans, Applebee's, and more

Not recommended: the Indian restaurant at Exit 123 from I-70 – the food is quite watered down and disappointing

Huber Heights, OH, Exit 36 from I-70 (~2.5 hours from campus):

- Osaka sushi, north side of I-70, right side of street, right next to the Speedway gas station

We hope to see you again at The Ohio State University
on Saturday, October 3, 2015!