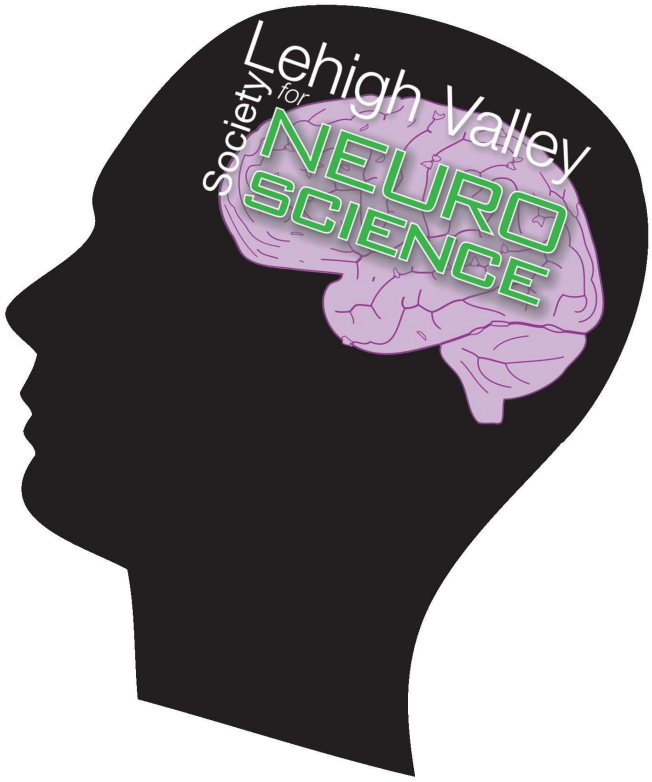
The 11th annual Lehigh Valley Society for Neuroscience Undergraduate Research Conference
Sunday April 25, 2021



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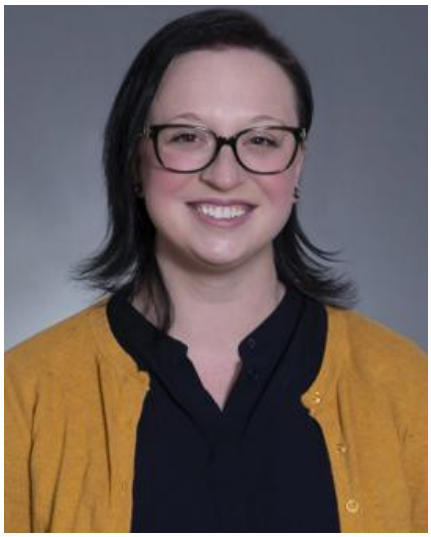
Moravian College
Muhlenberg College

Conference Schedule

10:00 am EDT	Welcome and Keynote Address
	Dr. Jacqui Barker, Drexel University College of Medicine "Innate and alcohol-induced sex differences in behavioral flexibility"
11:00-11:30am	Breakout sessions on addiction and equity
11:30am-12pm	Lunch break and continued discussion
12:00-1:00pm	Research Presentations: Sessions I and II
1:00-2:00pm	Research Presentations: Session III and IV
2:10-3:00pm	Alumni Career Panels & Social

Keynote Address



Dr. Jacqui Barker
Drexel University College of Medicine
"Innate and alcohol-induced sex differences in behavioral flexibility"

Jacqueline Barker, Ph.D., is an Assistant Professor in the Department of Pharmacology and Physiology at the Drexel University College of Medicine. Her graduate training in the Interdepartmental Neuroscience Program at Yale University and postdoctoral training in the Department of Neuroscience at the Medical University of South Carolina have provided a strong background in neurocognitive function and substance use disorders. Her lab investigates the neural circuits and molecular substrates regulating behavioral and cognitive flexibility and their dysregulation in disease states. To accomplish this, her work combines circuit- and cell-type specific approaches with novel pharmacological tools and animal models to assess acquired impairments following HIV infection and chronic drug and alcohol exposure. After the talk, the group will discuss the implications of Dr. Barker's work.

Break-out discussion sections follow Dr. Barker's talk. We encourage you to consider the following questions as starting points for a thoughtful discussion.

- What are some major barriers to access for those in need of SUD treatment? How do socialinequities strengthen or provide additional barriers?
- What factors push historically underrepresented individuals out of science? What strategies can weinclude to retain historically excluded scientists? In what ways might these strategies differ from recruitment?

Research Presentations Schedule

Session I: 12:00-1:00pm
Breakout Room #1

Protective effects of curcumin on the production andmetabolism of dopamine in the6-Hydroxydopamine model of parkinson's disease: aninterdisciplinary study

Raquel Lopez de Boer* and Cecilia M. Fox
Neuroscience Program and Chemistry Department, Moravian College

Postmortem Neuropathological Correlates of Cognitionin Essential Tremor

Marjana Tafader*, Silvia Chapman, Kurt Farrell, Daniella Iglesias Hernandez, Keith Radler, Leigh E. Colvin, Phyllis L. Faust, John F. Crary, Elan D. Louis and Stephanie Cosentino
Cognitive Neuroscience Division, Department of Neurology, Taub Institute and Sergievsky Center, Columbia University Medical Center

The neuroprotective effects of capsaicin in the 6-hydroxydopaminemodel of Parkinson'sDisease

Riley McHugh* and Cecilia M. Fox
Neuroscience Program, Moravian College

Neuroprotective effects of curcumin and vitamin Ein a 6-OHDA rat model of Parkinson's disease

Chloe Mondok* and Cecilia M. Fox
Neuroscience Program, Moravian College

Session II: 12:00-1:00pm
Breakout Room #2

Methods of analyzing taste preference reveal menthol'sability to drive nicotine consumption

Lauren E. Kerr*, Robert J. Wickham, Eric Nunes, Sophia Walton, and Nii A. Addy
Department of Psychology, Elizabethtown College, Elizabethtown PA; Department of Psychiatry, Yale School of Medicine, New Haven CT; Interdepartmental Neuroscience Program, Yale University, New Haven CT

Effects of acute menthol on phasic dopamine releasein rat nucleus accumbens

Kathryn A. Carter*, Robert J. Wickham, Colin W. Bond, and Nii A. Addy
Psychology Department, Elizabethtown College (Carter & Wickham) Department of Psychiatry, Yale School of Medicine (Bond & Addy)

Validating Sholl analysis of Purkinje cell morphology using MetaMorph software

Annabelle Brinkerhoff*, Carli King, and Mary E. Morrison
Lycoming College Biology Department

MAP Kinase Pathway Regulation of Purkinje Neuron Survival

Katie Moon*, Emilie Kramer, and Mary E. Morrison
Lycoming College Biology Department and Neuroscience Program

Session III: 1:00-2:00pm
Breakout Room #1

Identifying dyslexia: Link between maze learning and dyslexia susceptibility gene, DCDC2,in young children

Lisa A. Gabel, Kelsey Voss, Evelyn Johnson, Esther R. Lindstrom, Dongnhu T. Truong, Erin Murray, Karla Carino*, Christiana M. Nielsen, Steven Paniagua, and Jeffrey R. Gruen
Department of Psychology, Lafayette College, Program in Neuroscience, Lafayette College, Department of Special Education, Boise State University, Department of Education and Human Services, Lehigh University, Department of Genetics, Yale School of Medicine, Department of Pediatrics, Yale School of Medicine

Effect of acute seizure on associative memory in*D. melanogaster*

Matthew Wierzbicki* and Elaine R. Reynolds
Department of Neuroscience, Lafayette College

Can self-esteem impact color choice? Using the Manchester Color wheel to determine colorchoice differences

Emily Frantz*, Sarah Holstein, and Tina Norton
Lycoming College, Psychology Department

SIDS, serotonin, and nicotine exposure: Using an animal model to explore underlyingpathology

Muhammad Siddiqui*, Nicole Lester*, and Jeffery Erickson
Biology Department, The College of New Jersey

Session IV: 1:00-2:00pm: Research Blitz
Breakout Room #2

Measuring mitochondria membrane potential in cilia mutants with mito tracker and JC-1

María Padilla* and Tamara Stawicki
Neuroscience Program, Lafayette College

Motor activation facilitates perceptual understanding: a post-hoc exploration

Jacob Lader* and Matthieu de Wit
Department of Neuroscience, Muhlenberg College

The relationship between motor skill and affordance sensitivity in a virtual interception task

Caitlin Strain*, Michelle Rajan, and Matthieu de Wit
Department of Neuroscience, Muhlenberg College

The effect of antidepressants on stem cell neurogenesisin cichlid fish *Rocio octofasciata*

Hannah Kemperman* and Audrey Ettinger
Department of Biological Sciences, Cedar Crest College

Observing mitochondrial number differences in *Drosophila* bang-sensitive mutants

Madi Wahrmund* and Elaine Reynolds
Neuroscience Program, Lafayette College

Sulforaphane's effect on BTBR T + Itpr3tf/J mice

Samantha Riebesell,* Nicole Toumanios, and Lisa Gabel
Neuroscience Program, Lafayette College

Testing the effects of anxiolytic drugs on Jack Dempseycichlid behavior

Christina Scaffidi* and Audrey Ettinger
Department of Biological Sciences, Cedar Crest College

ERK signaling in hair cells

Grace Guerin* and Tamara Stawicki
Neuroscience Department, Lafayette College

Building interactive models of the GABA(A) Receptor

Emily Drake*, Jonathan Henry*, Jessica Orofino*, and Jeremy Alden Teissere
Department of Neuroscience, Muhlenberg College

Abstracts

Session I

Protective effects of curcumin on the production and metabolism of dopamine in the6-Hydroxydopamine model of Parkinson's disease: an interdisciplinary study

Raquel Lopez de Boer* and Cecilia M. Fox
Neuroscience Program and Chemistry Department, Moravian College

Parkinson's disease (PD) is a neurodegenerative disorder that results from a loss of dopamine neurons in the nigrostriatal pathway due to oxidative stress and chronic inflammatory responses. Therefore, to prevent dopamine neurons from cell death, multiple protective agents have been studied as a preventative therapy. Curcumin is a plant-based polyphenol compound that can be found in the spice, turmeric. It has been seen to have anti-inflammatory and antioxidant potential in other neurodegenerative models, thus gaining interest as a neuroprotective agent for the treatment of PD. Recently, our lab has identified that intraperitoneal injections of curcumin is capable of protecting dopamine neurons as well as reducing activation of microglia in a 6-hydroxydopamine rodent model of PD. The purpose of this study was to determine the effects of "dietary" curcumin on the production and metabolism of dopamine in this same animal model. There was significant improvement in motor behavior over time in the foot fault and the rotarod tests when the animals were treated with "dietary" curcumin for 8 weeks post-lesion. In addition, curcumin treated animals had a higher percentage of dopamine cell survival in the substantia nigra of the nigrostriatal pathway. Liquid chromatography mass spectroscopy was hoped to be used in assessing concentrations of dopamine and its metabolites, DOPAC and HVA. However, the need of an internal standard or LC-MS/MS was imperative to further proceed with quantification. foxc@moravian.edu

Postmortem Neuropathological Correlates of Cognitionin Essential Tremor

Marjana Tafader*, Silvia Chapman, Kurt Farrell, Daniella Iglesias Hernandez, Keith Radler, Leigh E. Colvin, Phyllis L. Faust, John F. Crary, Elan D. Louis and Stephanie Cosentino
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Essential Tremor (ET), historically viewed as an exclusively motor disorder, has been linked to increased risk for cognitive decline and dementia. The neuropathological basis of cognitive impairment in ET is unknown and likely complex. Imaging and neuropathologic

studies point to cerebellar dysfunction in ET, raising the possibility that cognitive deficits in ET arise secondary to a cerebellar-cortical syndrome. However, epidemiologic studies suggest that individuals with ET are at increased risk for developing Alzheimer's disease [AD]. At present, there is no way to tell whether a given ET patient, based on his/her observed cognitive features, has an early degenerative disease such as AD, or merely the expected cognitive profile of ET. The current study examined the extent to which cognitive functioning in older adults with ET is associated with cerebellar versus AD pathology. sc2460@cumc.columbia.edu

The neuroprotective effects of capsaicin in the 6-hydroxydopaminemodel of Parkinson'sDisease

Riley McHugh* and Cecilia M. Fox
Neuroscience Program, Moravian College

Parkinson's Disease (PD) is a neurological condition caused by the degeneration of dopaminergic neurons throughout the nigrostriatal pathway. Neuroinflammation, as a result of oxidative stress, has been seen to be one cause of chronic degeneration. Capsaicin, a historically powerful anti-inflammatory with antioxidant properties, decreases both peripheral and central nervous system inflammation in the rat lesion model of PD. This study investigated the neuroprotective potential of capsaicin in rodents challenged by the neurotoxin, 6- hydroxydopamine. The experimental group was injected with 0.25 mg/kg of capsaicin in 0.1 mL vehicle of DMSO three times per week for eight weeks, while control animals received only DMSO vehicular injections. Behavior testing was then administered for 8 weeks, concurrent with the intraperitoneal injections. Data from the behavior testing indicates significant motor protection for the experimental group in the rotarod test. The foot fault test data suggests the control group performed significantly worse over the length of the experiment compared to the capsaicin-treated group which saw no significant differences over the same period. Brain tissue from each group was processed for tyrosine hydroxylase immunocytochemistry. It was determined that there was no significant difference in dopamine cell survival between both groups, signifying the importance of further research to understand the protective mechanisms of capsaicin in the nigrostriatal pathway. foxc@moravian.edu

Neuroprotective effects of curcumin and vitamin Ein a 6-OHDA rat model of Parkinson's disease

Chloe Mondok* and Cecilia M. Fox
Neuroscience Program, Moravian College

Parkinson's disease is a common neurological disorder that is growing in prevalence among older populations. One of the reasons it is thought to develop is due to cellular changes that impair dopamine neurons of the nigrostriatal pathway. Curcumin has been shown to have anti-inflammatory and antioxidative effects which reduce inflammation and free radical levels; additionally, vitamin E is an antioxidant that also reduces lipid peroxidation, thus reducing cellular damage. Both curcumin and vitamin E have demonstrated neuroprotective properties in Parkinson's models. In this study, curcumin and vitamin E were used to determine whether there could be an enhanced neuroprotective effect in a 6-hydroxydopamine rodent model of Parkinson's disease. Experimental animals received dietary curcumin and vitamin E for eight weeks post-lesion. During this time, behavior testing using the rotarod, foot fault, and cylinder tests assessed motor function, endurance, and grip strength. These results demonstrated a trend in neuroprotective effects for experimental subjects. Immunohistochemistry was performed to determine the percent survival of dopamine neurons remaining in the substantia nigra. Though no significant difference was found in the percentage of dopamine cell survival between control (30%) and experimental (52%) subjects, these data reflect a trend in neuroprotective effects of curcumin and vitamin E in a rodent model of Parkinson's disease.
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Session II

Methods of analyzing taste preference reveal menthol'sability to drive nicotine consumption

Lauren E. Kerr*, Robert J. Wickham, Eric Nunes, Sophia Walton, and Nii A. Addy
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Flavored tobacco products are widely popular, suggesting that taste may play a critical role in tobacco use. Evaluating how the addition of flavorants to oral tobacco products influences consumption is necessary to understanding how such flavorants drive nicotine use and addiction. Taste preference, in the context of oral nicotine consumption, has been previously investigated in various experimental paradigms. However, there is variability among existing literature on the best method of analyzing and presenting this measure. In the present investigation, we developed a series of two-bottle choice tests to evaluate taste preference in rats, comparing nicotine alone to nicotine solutions flavored with saccharine or menthol. We also compared several methods of analyses for taste preference (% preference, difference score, and taste index) in order to determine which offered the most valid and sensitive assessment of preference. The results of the analysis revealed that oral consumption of nicotine, on its own, is aversive. It was initially expected that the inherently appetitive flavorant,

saccharine, would serve to increase preference. We did not find significant effects for the saccharine group, which may be attributed to the high concentration of nicotine used. Mentholated solutions, however, were found to be significantly preferred across all measures, suggesting that menthol may be a superior masker of nicotine's aversive taste. Additionally, it was found that % preference and taste index offered identical results and provided the most sensitive measure of preference among those that were tested, indicating the importance of which assessment tool is utilized to evaluate taste preference. wickhamr@etown.edu

Effects of acute menthol on phasic dopamine release in rat nucleus accumbens

Kathryn A. Carter*, Robert J. Wickham, Colin W. Bond, and Nii A. Addy
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Nicotine is the primary psychoactive ingredient in tobacco cigarettes and menthol is well known to exacerbate tobacco addiction. Nicotine is a nicotinic acetylcholine receptor (nAChR) agonist that increases dopamine firing in the ventral tegmental area (VTA) causing increased dopamine levels in the nucleus accumbens (NAc). Menthol is a flavorant commonly used in tobacco products to mask the aversive taste of nicotine and relieve irritation from smoke inhalation. Menthol smokers have poorer cessation outcomes, and higher expression of nAChRs in the VTA and other reward-related areas. Thus, menthol may influence neural responses to nicotine and the poorer cessation outcomes menthol smokers experience. The purpose of this study was to examine if menthol alone, or co-administered with nicotine, alters phasic dopamine release. Dopamine measurements were recorded for 30 minutes post-exposure to each drug condition, nicotine alone and menthol alone, and a subset of the nicotine condition also received a subsequent menthol injection after the initial 30-minute exposure, dopamine measurements were collected for an additional 30 minutes. Results revealed a significant increase of dopamine release from nicotine alone, however, menthol alone did not have a significant effect on dopamine release, and menthol did not have an additive effect on dopamine release when combined with nicotine exposure. These results confirm that nicotine enhances phasic dopamine release in the NAc, however, acute menthol does not impact dopamine release. This indicates that the role of menthol in tobacco addiction is more complex than facilitating a direct increase in dopamine release which warrants future study. wickhamr@etown.edu

Validating Sholl analysis of Purkinje cell morphology using MetaMorph software

Annabelle Brinkerhoff*, Carli King, and Mary E. Morrison
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The cerebellum, responsible for balance and coordination, including fine motor skills, contains highly branching cells known as Purkinje neurons. Because each Purkinje cell has such complex dendrites, Purkinje neurons are ideal models for studying dendritic development. The Morrison lab treats mouse Purkinje cells grown in culture with pharmacologic inhibitors to better understand the cells' signaling pathways. The resultant changes in dendritic growth can be quantified using semi-automated Sholl analysis, a custom designed computerized software algorithm which overlays concentric circles onto images of neurons and plots the neural intersections with each circle. Sholl analysis provides detailed information about changes in dendritic growth. This project developed an enhanced and updated version of a previous students' original algorithm, as well as a lab manual-style user guide to bolster accessibility. Both documents were validated extensively with trial runs of novice users to ensure user-friendliness. Using this new algorithm with the semi-automated software, users spend an average of 3-5 minutes analyzing a cell, compared to 20 minutes with the previous manual tracing method and generate less biased results. morrison@lycoming.edu

MAP Kinase Pathway Regulation of Purkinje Neuron Survival

Katie Moon*, Emilie Kramer, and Mary E. Morrison
Biology Department and Neuroscience Program, Lycoming College

The cerebellum coordinates fine and gross motor movements as well as balance. Purkinje neurons serve as the sole output of the cerebellum, and function as a calculator for motor error. Purkinje neurons degenerate and die in patients with cerebellar ataxia. Knowing the different signaling pathways involved in Purkinje neuron growth and survival is essential for future development of drugs and treatments for these patients. The goal of these experiments was to quantify Purkinje neuron survival after inhibiting the MAP kinase pathway in cerebellar cultures. In one experiment, Purkinje cell survival was statistically significant between a treated 100uM group with PD98059 MAP kinase pathway inhibitor and a 0uM untreated group. In a separate independent experiment, no significant difference was found between treated and untreated groups. morrison@lycoming.edu

Session III

Identifying dyslexia: Link between maze learning and dyslexia susceptibility gene, DCDC2,in young children

Lisa A. Gabel, Kelsey Voss, Evelyn Johnson, Esther R. Lindstrom, Dongnhu T. Truong, Erin Murray, Karla Carino*, Christiana M. Nielsen, Steven Paniagua, and Jeffrey R. Gruen
Department of Psychology, Lafayette College, Program in Neuroscience, Lafayette College, Department of Special Education, Boise State University, Department of Education and Human

Services, Lehigh University, Department of Genetics, Yale School of Medicine, Department of Pediatrics, Yale School of Medicine

Dyslexia is a common learning disability that affects processing of written language despite adequate intelligence and educational background. If learning disabilities remain untreated, a child may experience long term social and emotional problems which influence future success in all aspects of their life. Dyslexia has a 60% heritability rate, and genetic studies have identified multiple dyslexia susceptibility genes (DSGs). DSGs, such as DCDC2*, are consistently associated with the risk and severity of reading disability (RD). Altered neural connectivity within temporo-parietal regions of the brain are associated with specific variants of DSGs in individuals with RD. Genetically altering DSG expression in mice results in visual and auditory processing deficits as well as neurophysiological and neuroanatomical disruptions. Previously, we demonstrated that learning deficits associated with RD can be translated across species using virtual environments. In this two-year longitudinal study, we demonstrate that performance on a virtual Hebb-Williams maze in pre-readers, is able to predict future reading impairment, and the genetic risk strengthens, but is not dependent on, this relationship. Due to the lack of oral reporting and use of letters, this easy-to-use tool may be particularly valuable in a remote working environment, as well as working with vulnerable populations such as English language learners. gabell@lafayette.edu

Effect of acute seizure on associative memory in D.melanogaster

Matthew Wierzbicki* and Elaine R. Reynolds
Department of Neuroscience, Lafayette College

Drosophila has been used effectively as a model for human epilepsy by using mutant flies that have seizure phenotypes, by feeding flies drugs that generate seizures, or more recently, by using optogenetics to generate seizure in specific regions of the brain. The optogenetic seizures are created using the Gal 4 UAS expression system: a light responsive channel rhodopsin behind a UAS promotor is expressed in a pattern that corresponds to the enhancer near the Gal 4 insert. We are using optogenetically-generated seizures to explore the impact of seizure on learning and memory (L&M). Since both L&M are established by increased frequency or associative firing, we expect that seizure in specific brain regions should have an impact on these processes. We are assessing learning initially using fly mating assays. Males learn and remember to suppress their courtship activities when exposed to previously mated females. We propose that seizure will disrupt these processes. Flies with Gal4 inserts in three different loci were studied, two expressing in various lobes of the mushroom bodies (MB) and one expressing in several integrative regions. Expression was confirmed via GFP imaging. Our findings suggest that the gene 117Y expressed in the MB is necessary for normal courtship as Gal4 insertion

disrupted mating. We found that seizure in the ring neurons, large field neurons and ellipsoid body just before the courtship assay is sufficient to disrupt courtship. However, a single instance of seizure delivered right before the assay in the alpha and beta lobes of the MB did not disrupt L&M. Our findings open the door for future studies to further utilize this model to investigate the role seizure has on L&M in different brain areas and perhaps over repeated instances. reynolde@lafayette.edu

Can self-esteem impact color choice? Using the Manchester Color wheel to determine colorchoice differences.

Emily Frantz*, Sarah Holstein, and Tina Norton
Psychology Department, Lycoming College

This study evaluated the relationship between depression, self-esteem and color choice in order to confirm not only that depression predicts variations in color choice, but also whether the relationship between depression and color choice may be mediated, in part, by variations in self-esteem. In this correlational study, participants (N= 53) completed questionnaires on self-esteem and depression before being asked to choose a series of colors, adapted from the Manchester Color Wheel, based on the question "Which of the following four colors best corresponds to your day-to-day mood over the last few months?". Bivariate correlational analyses revealed a strong negative correlation between self-esteem and depression, and a moderate positive correlation between depression and color choice. However, there was no significant correlation between self-esteem and color choice. A moderation analysis was then conducted to evaluate whether the relationship between depression and color choice was mediated by variations in self-esteem. A linear regression confirmed a significant relationship between depression and color choice, and self-esteem and depression. Self-esteem, however, did not predict color choice and when controlling for self-esteem, the relationship between depression and color choice remained significant. The data suggests that self-esteem does not mediate the relationship between color choice and depression, meaning that differences in color choice may be due to depression alone. This research can be applied to our understanding of how depression may alter some aspects of daily functioning and may provide insight into the perceptual changes associated with depression. holstein@lycoming.edu

SIDS, serotonin, and nicotine exposure: Using an animal model to explore underlyingpathology

Muhammad Siddiqui*, Nicole Lester*, and Jeffery Erickson
Biology Department, The College of New Jersey

Serotonin (5-hydroxytryptamine, 5-HT) is a neurotransmitter that modulates a number of brain functions, including homeostatic regulation of respiratory rhythm and heart rate. A deficiency of central 5-HT is correlated with Sudden Infant Death Syndrome (SIDS) in humans. One proposed cause for SIDS is an inadequate autoresuscitation (self-resuscitation) response, where infants fail to reestablish regular breathing and heart rate following prolonged apnea and hypoxia-induced gasping. The incidence of SIDS is increased in infants born to maternal smokers, likely the result of "in utero" exposure to the neuroteratogen nicotine. However, the mechanisms by which 5HT deficiency and developmental nicotine exposure increase the risk for sudden death are unclear. We have used the "Pet-1" knockout (KO) mouse as an animal model for SIDS. This mouse strain is characterized by a severe deficiency of central 5HT, increased incidence of spontaneous apneas, impaired autoresuscitation, and higher neonatal mortality compared to wild-type littermates. All of these characteristics are reminiscent of SIDS. Here, we compared autoresuscitation responses of wild-type and "Pet-1" KO neonates to experimentally induced apnea following developmental exposure to saline (control) or nicotine. We hypothesized that nicotine exposure would exacerbate the breathing deficits found in KO mice and increase neonatal mortality. Unexpectedly, we found that nicotine "normalized" breathing behavior in KO mice without improving survival. This suggests that abnormal cardiac control, rather than breathing deficits, may be responsible for the higher neonatal mortality in the 5HT-deficient mice. We are currently incorporating ECG recordings into our experimental protocols to explore this possibility further. erickson@tcnj.edu

Session IV (Research Blitz)

Measuring mitochondria membrane potential in cilia mutants with mitotracker and JC-1

María Padilla* and Tamara Stawicki
Neuroscience Program, Lafayette College

Side-effects driven by aminoglycoside antibiotics contribute to hair cell death, resulting in hearing and balance disorders. Cilia mutatants, known to be resistant to aminoglycoside-induced hair cell death, have shown influence on mitochondria which is also involved in aminoglycoside-induced hair cell death. Thus, we wished to investigate mitochondria in cilia mutants to further investigate mechanisms by which cilia mutants display resistance to aminoglycosides. To further investigate if mitochondria activity is altered in hair cells of cilia gene mutants we analyzed mitochondrial activity with mitotracker and JC-1 dyes. Mitotracker green shows fluorescent mitotracker dyes stain mitochondria, whereas accumulation of mitotracker red depends on changes in mitochondrial membrane potential. The JC-1 dye is also a useful indicator of mitochondrial membrane potential. We focused on ift88, dync2h1, and cc2d2a mutants. The reason why we placed our focus on these three

mutants was because of their resistance to aminoglycoside-induced hair cell death. We utilized both dyes, mitotracker and JC-1, to record changes in response to mitochondrial potential in both wildtype and mutants. We did not notice any significant changes between both of these groups when using mitotracker red and green dyes. However, when we used JC-1 dye we saw significant differences between our wildtype and intraflagellar transport mutant fish. These findings shape our future directions to investigate whether mutants are resistant to neomycin due to this mitochondria change or if this change is a consequence of reduced hair cell activity in these mutants. " stawickt@lafayette.edu

Motor activation facilitates perceptual understanding: a post-hoc exploration

Jacob Lader* and Matthieu de Wit
Department of Neuroscience, Muhlenberg College

Understanding the functions of brain regions is a foundational question in cognitive neuroscience. One view holds that single brain regions perform unique functions ("functional specificity"). An opposing view holds that a single brain region can be activated during a range of different tasks due to multiple, different cognitive domains drawing on the same neural resource ("neural reuse"). In this study (N=12) we created a dual-task paradigm in which a motor task (periodic arm/hand raising) was paired with one of two randomly interspersed primary visual tasks of equal difficulty (detecting Gabor stimulus orientation or brightness). An adaptive staircase procedure was used to determine stimulus difficulty at ~75% accuracy for each primary task and individual. If two primary tasks of the same difficulty show differing proficiencies after the addition of a secondary task, then the affected task may share neural resources with the secondary task. We found that our secondary task facilitated concurrent performance of the orientation -but not brightness-task. Our dual-task approach potentially allows for specific causal tests of neural reuse hypotheses guided by neuroimaging data, adding to the current supporting evidence which has largely come from correlational neuroimaging meta-analyses. While these initial results are promising, we performed a power analysis to determine the correct sample size for a confirmatory replication study. We also planned a follow-up study designed to probe the facilitatory effect of motor behavior on orientation detection performance, and performed various post-hoc exploratory analyses to better understand the current data set.
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The relationship between motor skill and affordance sensitivity in a virtual interception task

Caitlin Strain*, Michelle Rajan, and Matthieu de Wit
Department of Neuroscience, Muhlenberg College

Action-scaled affordances are action possibilities available to individuals determined by bodily skills. Affordance boundaries demarcate what an individual can and cannot do. To understand participants' (N=22) sensitivity to their action-scaled affordance boundaries we asked them to intercept a virtual ball traveling at a range of speeds (very slow to very fast) superimposed over a response button, in two phases that were repeated over two days. Absolute difference between time of ball arrival and button press was calculated to determine an individual's skill level. In the learning phase, the screen provided interception success feedback after each trial. In the affordance sensitivity phase, participants were instructed to only attempt to intercept balls that they believed they could successfully stop. Participants did not receive feedback during this phase. While there was variability between the participants, there is a suggestive correlation between skill level at particular ball speed and the likelihood of interception attempts for that speed, particularly for more highly skilled individuals. We also observed an increase in performance between day one and day two, indicating continued gains in interception skill level and affordance boundary sensitivity. Our data suggests that more highly skilled individuals are more sensitive to their action-scaled affordances. These findings will be used to inform future neuroimaging (fNIRS) studies in the lab that look into the neural correlates of the connection between motor skill and affordance boundary sensitivity. Finally, this data may be useful in the future development of physical therapy and motor rehabilitation treatments.
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The effect of antidepressants on stem cell neurogenesisin cichlid fish *Rocio octofasciata*

Hannah Kemperman* and Audrey Ettinger
Department of Biological Sciences, Cedar Crest College

This study investigates the effect of the selective serotonin reuptake inhibitor (SSRI), Sertraline, on the behavior and stem cell generation in the brains of Jack Dempsey cichlid fish ("Rocio octofasciata"). SSRIs are a pharmaceutical designed to treat depression in humans. SSRIs have some immediate effect in patients, but significant improvement is usually seen only following several weeks of administering the drug. SSRIs work quickly by increasing serotonin, but part of the mechanism of long-term antidepressant effects occurs via increased hippocampal neurogenesis. The adult brain generates new neurons throughout life. One primary location of adult neurogenesis is in the hippocampus. After long term use, antidepressants increase hippocampal neurogenesis and as a result, decrease depressive symptoms. The molecular mechanism underlying the etiology of depression is not fully understood; however, hippocampal neurogenesis seems to play an integral role in long term antidepressant action. The cichlid fish model is used to further investigate the effect of SSRI antidepressants on neurogenesis by examining the effect antidepressants have on behavior and the

quantity of stem cells produced within cichlid fish brains. An increase in stem cell production in cichlid fish treated with the SSRI Sertraline would support the involvement of hippocampal neurogenesis in the role of antidepressant action to decrease depression in humans.
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Observing mitochondrial number differences in drosophila bang-sensitive mutants

Madi Wahrmund* and Elaine Reynolds
Neuroscience Program, Lafayette College

Drosophila melanogaster, the fruit fly, have long been used as a model organism for human disorders. Drosophila "bang-sensitive" (bs) mutants have been utilized as models for neurological conditions including epilepsy, sensorineural deafness, and age-dependent neurodegeneration. These mutants also have a shortened lifespan. Diet has been shown to modify these phenotypes both in flies and humans. For example, a ketogenic diet has been shown to be an effective treatment for refractory epilepsy in humans. Bang-sensitive flies mutants display a lower percentage of seizures on high protein food, but also reduced viability and lifespan. Recent data has suggested that both the mutants and the diet manipulation might affect mitochondrial function since cytochrome oxidase, a mitochondrial enzyme involved with energy production, is altered under different dietary conditions. For this experiment, bs mutants and a control were fed either a standard low protein food (MYC) or yeast-sugar (YS) food with equal amounts of sugar and protein. The purpose of the experiment is to examine how the protein-carb ratio of food impacts mitochondrial number using citrate synthase (CS), a Kreb cycle enzyme that has been shown to have activity proportional to mitochondrial number. After the flies hatched, mitochondria were isolated using an isolation kit and then assayed for CS (both kits purchased from Sigma-Aldrich). Activity was determined per mg of protein as determined by a BCA protein assay (Pierce). The experiments are in progress, and our initial findings on the standard four with three bs mutants will be presented. reynolde@lafayette.edu

Sulforaphane's effect on BTBR T + Itpr3tf/J mice

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Sulforaphane is an isothiocyanate derived from broccoli sprouts. Its therapeutic potential is based on its function of the upregulation of genes that control mechanisms in aerobic cells. These aerobic cells are more likely to protect themselves against oxidative stress, inflammation, and DNA-damage, which are all characteristics of autism spectrum disorder (ASD). One reason to use sulforaphane to treat the symptoms of ASD is that sulforaphane prevents many biochemical and

molecular abnormalities associated with ASD, like oxidative stress and reduced antioxidant capacity. Sulforaphane is also considered to have low toxicity and the drug is well tolerated by humans. I hypothesize that daily treatment with sulforaphane at levels achieved by oral administration will reduce the severity of ASD behavioral symptoms in BTBR T+Itpr3tf/J (BTBR) mice. This study will also be one of the first studies to administer sulforaphane orally to examine the effects of memory and anxiety as well. The BTBR mouse, originally bred for studies on insulin-resistance, diabetes-induced nephropathy, and phenylketonuria, was identified only a decade ago as displaying strong and consistent autism-relevant behaviors. Oxidative stress is reported to be at elevated levels in both human autistic subjects and BTBR mice. In summary, my study aims to use sulforaphane in asocial BTBR mice and its social counterpart C57/BL6 (C57) mice to assess sulforaphane's therapeutic effect on autism-like symptoms (repetitive behaviors and impaired sociability), and short-term/working memory and anxiety; impaired short-term memory and heightened anxiety are frequent comorbidities among ASD patients. gabell@lafayette.edu

Testing the effects of anxiolytic drugs on Jack Dempseycichlid behavior

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Stress is a part of everyday life for all organisms, and responding to it is vital to survival. Many kinds of stress experienced by animals are transient. However, a long-term exposure to stress can affect both physiology and behavior. This study will measure social behavior in three identical communal tanks of Dempsey cichlid fish ("Rocio octofasciata"). First, we will establish a baseline of aggressive behavior in dominant individuals and fleeing behaviors in non-dominant individuals as a proxy for stress and anxiety. Two tanks will then be treated with an anxiolytic drug at two different concentrations comparable to those used to treat human patients, with a third tank serving as an untreated control. The behavioral baseline will be used as a comparison to identify any behavioral effects of the anxiolytic drug treatment, particularly to determine if there is a decrease in expressed aggression or fleeing behaviors, and to establish an appropriate dose. Next, each tank of fish will be introduced to a relevant, stressful stimulus, such as a fake predator, and behavioral responses will be measured. A reduction of aggression or fleeing behaviors would represent an increased ability to cope with prolonged stress, and would contribute to better understanding how the response to long-term stress may be modulated by drug treatments. In addition, these experiments could provide a potential animal model for screening additional compounds to be used as anxiolytic drugs.
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ERK signaling in hair cells

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The ERK signaling pathway is a member of the mitogen-activated protein kinase (MAPK) signaling family and it is implicated in cell proliferation and survival. It is largely unknown whether ERK promotes or protects hair cells from aminoglycoside-induced death. It has been shown that ERK is activated in the cochlear cells of mice in response to cisplatin, a chemotherapeutic drug that is similar to aminoglycosides in its ototoxic and vestibulotoxic properties, so I expected that ERK would also be activated in response to aminoglycosides. A greater understanding of how ERK functions in hair cells and in response to will allow us to better understand hair cell function and death as well as the ERK signaling pathway. I investigated ERK function in hair cells using the zebrafish lateral line system. We failed to see a noticeable difference in lateral line hair cell death between fish treated with neomycin and control fish. Additionally, we stained for pERK and failed to see noticeable staining in mature lateral line hair cells, but did see staining in possible hair cell precursors and the lateral line nerve. The developmental role of ERK is currently being investigated and future work will look at whether pERK is upregulated following aminoglycoside activity. stawickt@lafayette.edu

Building interactive models of the GABA(A) Receptor

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The gamma-aminobutyric acid receptor ($GABA_AR$) is an ionotropic neurotransmitter receptor and the primary inhibitory receptor in the vertebrate brain. The effects of benzodiazepines, barbiturates, neurosteroids, alcohol, and anaesthetics occur through allosteric molecular interactions with the $GABA_AR$. Our laboratory has previously shown that phytoextracts derived from plants that induce anxiolysis and/or hypnosis, including *Passiflora incarnata* (passionflower) and members of *Citrus,* are also positive allosteric modulators with direct actions on synaptic $GABA_AR$. We have begun to characterize the structural determinants of the receptor that permit modulation by these phytoextractsand have identified key amino acids in the fourth transmembrane domain of the α1 subunit that appear to confer sensitivity. In parallel, we have developed detection strategies using chemical derivitization, HPLC, and GC-MS to identify the specific putative compounds in the phytoextracts responsible for these effects. Through modelling different subtypes of the $GABA_AR$, we can better understand the impact of structure on receptor function, predict the effects of these plant-derived compounds, and provide insight into the future synthesis of novel GABAergic drugs. teissere@muhlenberg.edu

Alumni Career Panels & Social

Breakout Room #1: ***Health Professions***

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Breakout Room #2: ***Graduate School/Research***

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Breakout Room #3: ***Education/Administration***

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Breakout Room #4: ***Faculty Social***

Please feel free to join the breakout room for this special faculty event!