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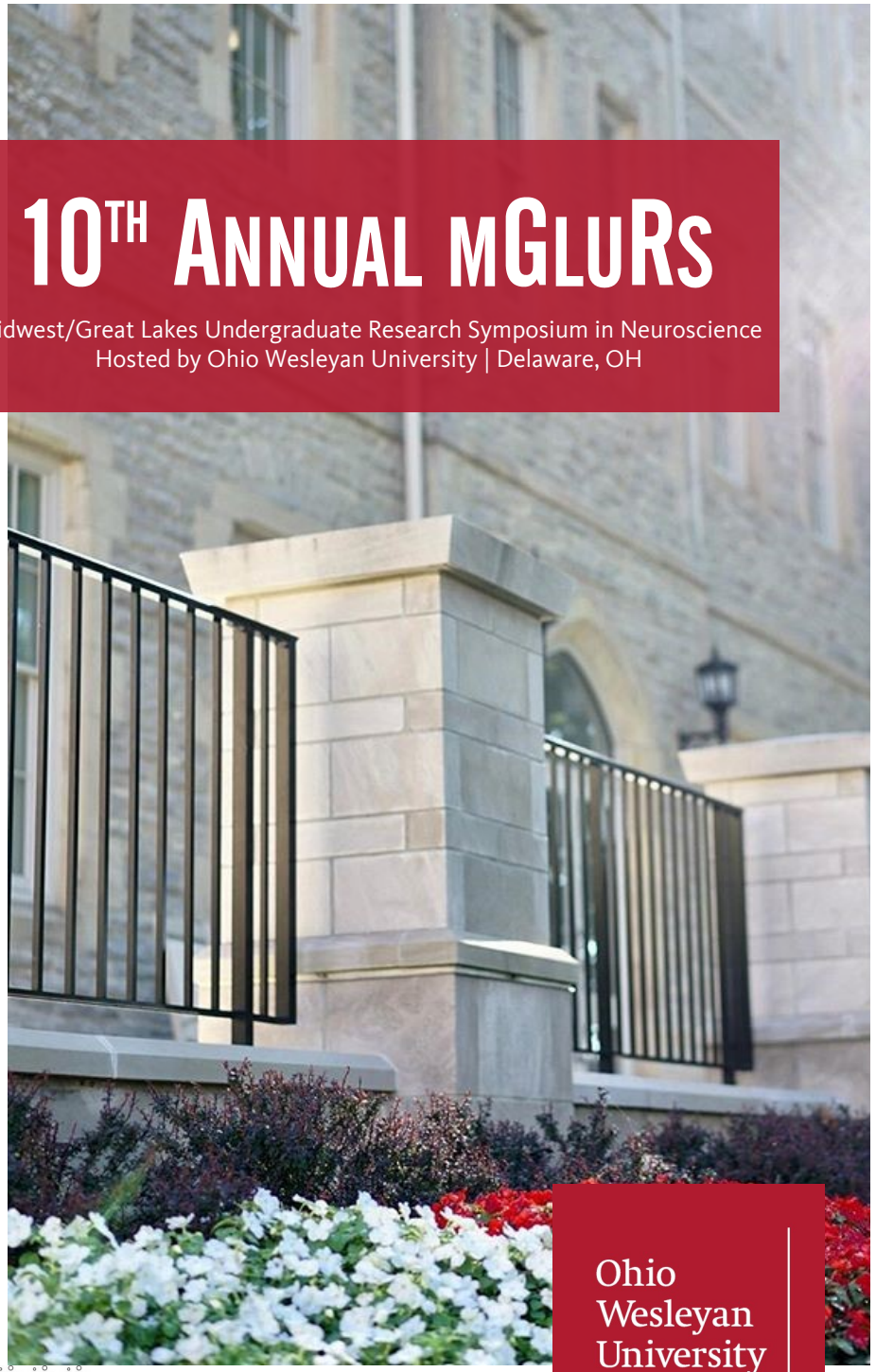
10TH ANNUAL mGLuRs

Midwest/Great Lakes Undergraduate Research Symposium in Neuroscience
Hosted by Ohio Wesleyan University | Delaware, OH



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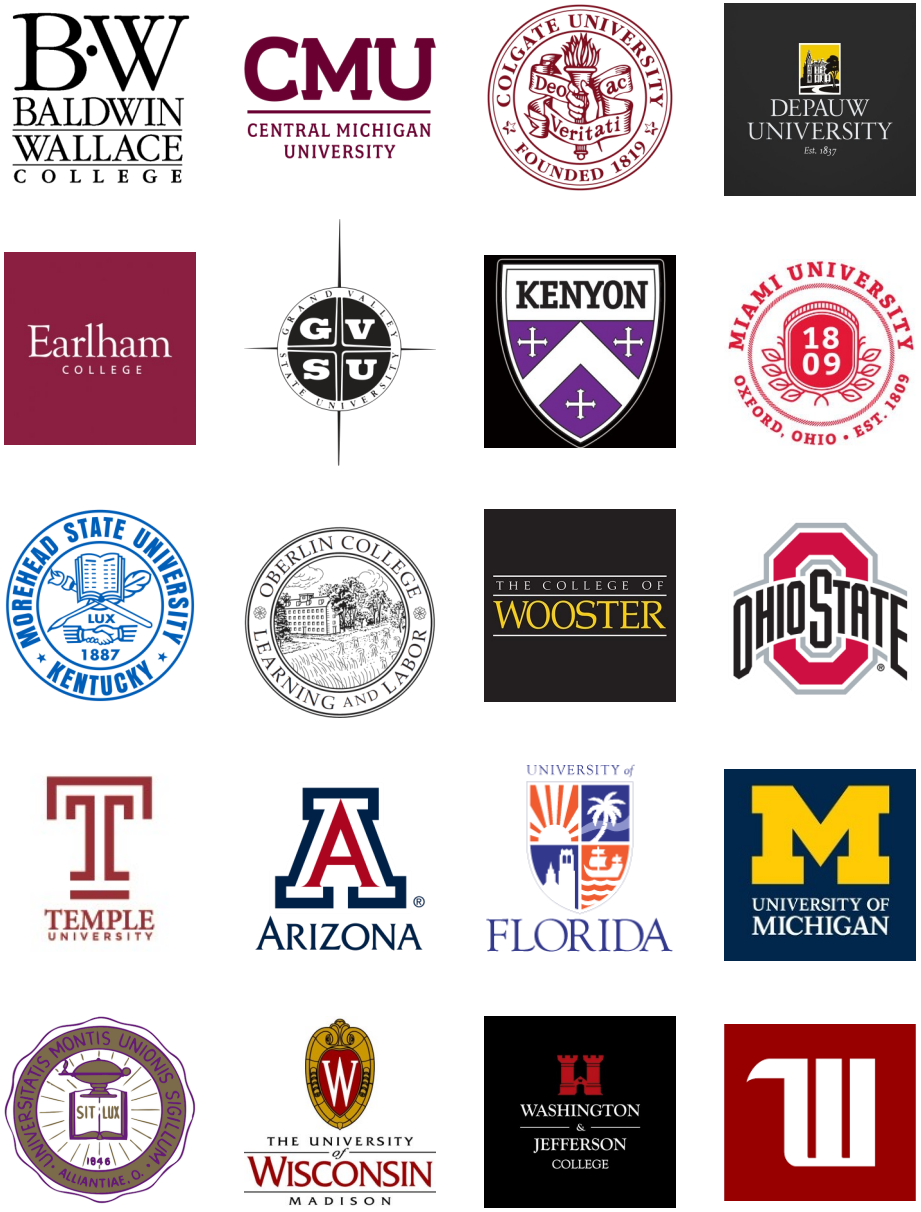


THE 11TH ANNUAL MGLURS 2019 CONFERENCE

WILL BE HOSTED BY:

THE COLLEGE OF
WOOSTER

NOTES

SCHEDULE

9:00AM - 10:00AM — BREAKFAST & REGISTRATION

Breakfast: Merrick Hall, 1st Floor
Registration: Science Center Atrium

10:00AM - 11:00AM — KEYNOTE ADDRESS: *Dr. Janet Best*

Depression: Listening to what the brain can tell us.

Merrick Hall, 3rd Floor

11:00AM - 11:25AM — COFFEE BREAK

Merrick Hall, 1st Floor

11:30AM - 12:15PM — STUDENT ORAL PRESENTATIONS

Merrick Hall, 3rd Floor

12:15PM - 1:15PM — LUNCH

Box lunches available on 1st floor for early registrants. Seating on 1st and 2nd floor or outside.

Merrick Hall

1:15PM - 1:30PM — POSTER SET-UP

Science Center Atrium (1st Floor)

1:30PM - 3:30PM — POSTER SESSION

Even posters from 1:30-2. Odd posters from 2-2:30.

Science Center Atrium (1st Floor)

3:30PM - 4:30PM — BREAKOUT SESSIONS

Student session on 3rd floor: Career Panel.
Faculty Session in room 202: Increasing Diversity in Neuroscience Courses.

Merrick Hall

4:45PM - 5:00PM — CLOSING REMARKS & POSTER AWARDS

Merrick Hall, 3rd Floor

KEYNOTE ADDRESS

Janet Best, Ph.D.
Department of Mathematics, Associate Director of Mathematical Biosciences Institute, Ohio State University.

DEPRESSION: LISTENING TO WHAT THE BRAIN CAN TELL US

Fundamental questions about depression are particularly pressing due to the rising global health burden posed by this illness. Malfunction in the serotonergic system is generally believed to underlie symptoms of depression, but do depressed individuals really have lower levels of serotonin? Does the frontline treatment – Selective Serotonin Reuptake Inhibitors (SSRIs) – actually raise serotonin levels, and if so, by what mechanism? How can we account for the surprising variability in patient responses to currently prescribed SSRIs? What can we say about the outlook for developing new treatments for depression?

In this talk, we explore how combining emerging brain chemistry tools with mathematical modeling can address these questions. We're finding new insights into depression by investigating the relationship between dynamical processes of neurons, brain chemistry, and the role of inflammation. Such interdisciplinary approaches have the capacity to profoundly improve therapeutic strategies.

NOTES

NOTES

CAREER PANELISTS

Denisse Paredes

Ph.D. Graduate Student
Neuroscience Graduate Program
University of Texas Health Science Center at San Antonio

Zach Ford

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

C.J. Tosino

D.O. Medical Student
Ohio University Dublin

Sverre Aune, Ph.D.

Medical Writer
Navicor, Syneous Health Communications

PRESENTATION ABSTRACTS

Author index located on page 61.

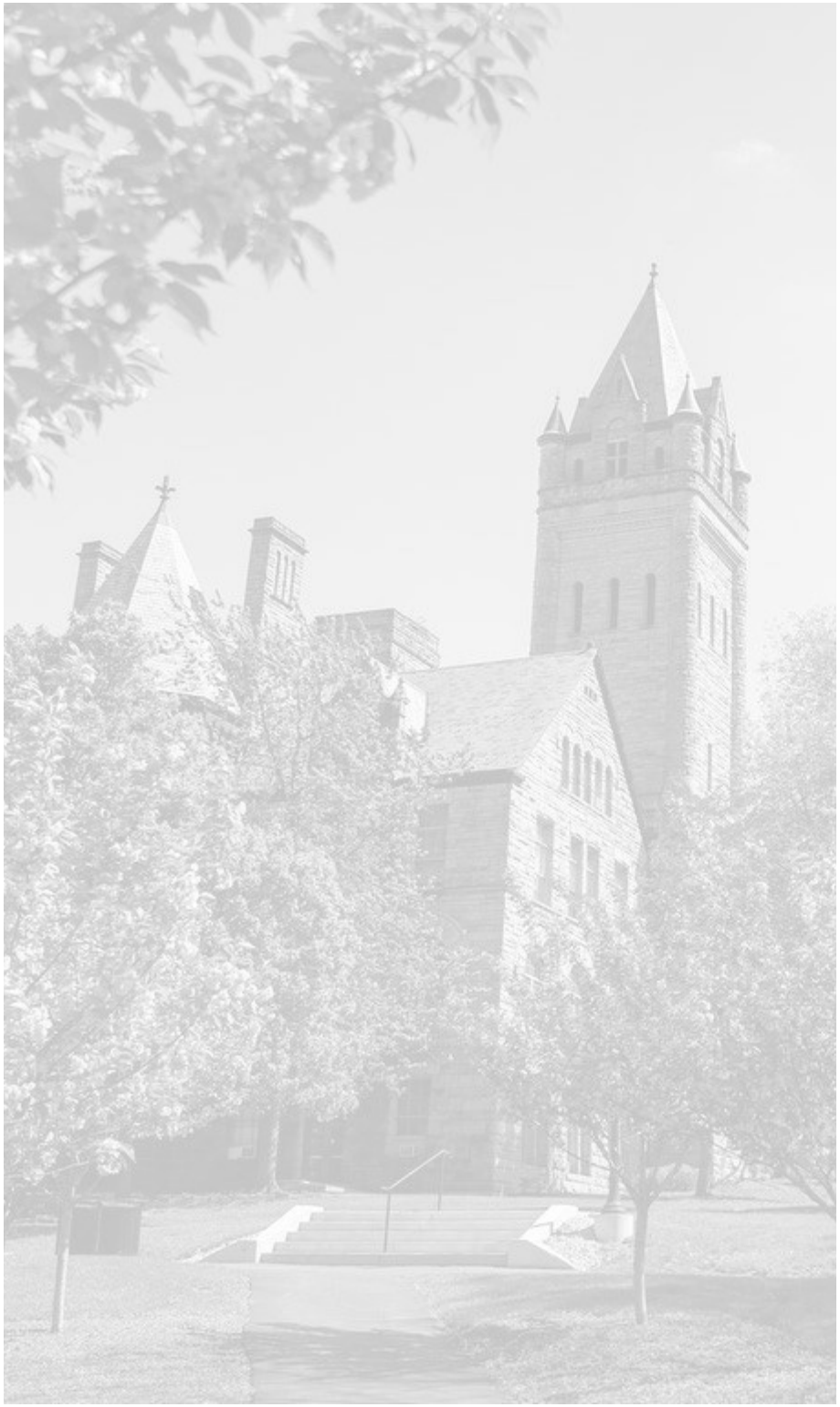
Co-first authors are indicated with an asterisk.

A Drum-playing Modulates the Post-auricular Muscle Response: Intention or Attention?

ZOE SWANN *Oberlin College*
PATRICK SIMEN *Oberlin College*

The post auricular muscle, located behind the ear above the mastoid bone, is known to produce a brief, involuntary reflex roughly 10 milliseconds after high-frequency sounds in many (but not all) human participants. The electrical signature of this vestigial response, the post-auricular muscle response (PAMR), is a muscle action potential observable in electrodes placed above the mastoids. We found that the PAMR was bilaterally modulated by task goal for equivalent sound stimuli (snare drum sounds). We discovered this PAMR effect in the process of investigating the brain basis of inter-beat interval timing in rhythmic behavior. In prior work, we used electroencephalography (EEG) to monitor neural and muscular signals while human participants actively produced sequences of drumbeats, with passive listening and non-rhythmic drumming tasks as control conditions. We developed an attentional task as a new control condition In each condition, participants produced or listened to rhythmic or non-rhythmic patterns of snare drumbeats at 70-100 beats per minute for two minutes. Resulting PAMR amplitudes, surprisingly, ranged from 5-200 microvolts (μV) in active drumming, versus 3-70 μV in passive listening. Within participants, active drumming produced a dramatically larger average PAMR than passive listening, and rhythmic drumming/listening produced larger average PAMRs than corresponding non-rhythmic conditions. We further observed a distinct latency difference relative to stimulus time between active and passive trials, with active responses occurring approximately 16 milliseconds (ms) after the snare sound, and passive responses at approximately 25 ms after the sound. The latency of the PAMR suggests it is mediated by only a few synaptic connections between cochlea and motor neurons. Modulation by task goal suggests a top-down effect in which attention to rhythmic production can functionally enhance this connectivity. However, we observed no correlation between PAMR magnitude and inter-beat interval duration, nor any obvious, beat-by-beat basis for PAMR modulation. We hypothesized that active playing accentuates the PAMR simply because participants attend more to the stimuli in that case. To test this hypothesis, we randomly generated oddball bass drum sounds instead of a snare in a third task condition. Participants responded when they heard this oddball timbre-change. In one-quarter of the attentional conditions, participants showed a PAMR latency that mimicked the passive, not the active condition. In the remaining participants, the PAMR mimicked the active condition's latency. These data suggest that certain participants may ignore sounds during passive listening, while in others the PAMR reflects motor planning/performance monitoring in the auditory system. Thus, the PAMR magnitude and latency may serve as useful tools for investigating the brain basis of timed, rhythmic behavior.

INDEX OF AUTHORS



B Neuronal Populations in Multiparous Versus Virgin Mice

Breann Losiewicz *Wittenberg University*
Andrea Stathopoulos *Wittenberg University*

Neurogenesis, the process by which new nervous system cells are created in the brain, is thought to occur mostly early on in human development, and shows a steady decline as we age. However, there is new evidence to suggest that we, as humans, can increase our rates of neurogenesis after certain events have occurred. One of these events is the onset of maternal care instincts and childbirth - a period of time during which many neurotransmitters, hormones, metabolic processes, stress levels, and learning demands are all elevated. We therefore selected three neuronal markers to investigate in multiparous mice versus virgins: oxytocin, orexin, and doublecortin. It is suspected that these will show an increase in concentration due to the relationship between maternal care and the neurotransmitter's inherent function. Oxytocin is important in fertility, delivery, and lactation processes, orexin functions in metabolism regulation, and doublecortin is simply a marker for new neurons, which might indicate the need for improved learning or demonstrate how neurogenesis responds to demanding stressors. Immunochemistry processes (IHC) were used to stain and attach antibodies to brain slices taken from the hippocampal and hypothalamus regions of virgin and multiparous mice. Once the specimens were stained, they were viewed under a microscope, photographed, and the neurons were counted by hand. As anticipated, differences were observed between the two groups of mice, but not always in the expected direction. Some of the counts for the multiparous mice were actually lower than in virgin mice, showing that maternal care instincts and motherhood experience may have complicated and lasting effects on the brain. Future work should include more testing and higher sample sizes, as well as assess a few more possibly important neuronal markers, such as serotonin.

C Functional Dissection of Nigrostriatal Dopamine Projections Across Striatal Subregions

Jacob A. Nadel *Oberlin College*
Sean S. Pawelko *Oberlin College*
Della Copes-Finke *Oberlin College*
Christopher D. Howard *Oberlin College*

The dorsal striatum, an input nucleus of the basal ganglia, is generally thought to be involved in action selection and control of behavioral strategies. A key modulator of striatal function is the neurotransmitter dopamine (DA), which is released by projections from the substantia nigra pars compacta (SNc). The dorsal striatum can be subdivided into two subregions based on cortical inputs and differing functional roles. Specifically, the dorsomedial striatum (DMS) is thought to be involved in goal-directed actions, whereas the dorsolateral striatum (DLS) is believed to be involved in habitual behaviors. However, the behavioral function of nigrostriatal dopamine projections to the DMS and DLS remain largely unknown. To investigate the roles of nigrostriatal dopamine in these subregions, we selectively expressed the light-activated cation channel channelrhodopsin-2 (ChR2) in the SNc of DAT-cre mice. This manipulation allows us to selectively stimulate dopamine neurons using laser light. Next, we bilaterally implanted mice with fiber optic implants in either the DMS or DLS, allowing for subregion-specific stimulation. Mice first performed a ten-day intracranial self-stimulation (ICSS) task where they were given access to two levers in an operant chamber. In the first five days of this paradigm, pressing one lever resulted in optogenetic stimulation of dopamine terminals and robust responding. After five days, the active lever was switched. DLS mice tended to press the initially active lever at higher rates and, consistent with the canonical roles of DMS and DLS in habit formation, DLS mice were less efficient altering their behavior following changing the active lever, indicating accelerated habit formation. Mice were next placed in an open field assay to explore the effect of stimulating DA on locomotion. Stimulation of DA projections in both subregions led to changes in velocity, such that mice moving quickly pre-stimulation began moving slowly with stimulation, and vice-versa. Finally, mice were placed in a two chamber, real-time conditioned place preference (RTCPP) paradigm, where moving to one chamber led to laser stimulation and being in the other chamber led to no stimulation. Contrary to DA's reported role in place preference, mice did not show a preference for the stimulated chamber, regardless of the stimulated subregion. These results suggest differential roles for nigrostriatal dopamine in action selection and reinforcement, where activating dopamine release across subregions similarly reinforces action and invigorates locomotion, but where DLS stimulation may specifically increase behavioral inflexibility.

48 Adapting Touchscreen Technology for a Balb/c Cancer Survivor Model to Assess Cognitive Disturbances

Lindsay Strehle *Institute for Behavioral Medicine Research, The Ohio State University*
Gabriel Perez-Otero *Institute for Behavioral Medicine Research, The Ohio State University*
Jasskiran Kaur *Institute for Behavioral Medicine Research, The Ohio State University*
Ashley A. Lahoud *Institute for Behavioral Medicine Research, The Ohio State University*
Julie Fitzgerald *The Ohio State University*
Zachary M. Weil *The Ohio State University*
Leah M. Pyter *Institute for Behavioral Medicine Research, The Ohio State University*

Many cancer patients suffer from a wide range of collateral issues from their treatments, such as emotional and cognitive disturbances. There are a wide variety of rodent models and paradigms in practice today to biologically investigate effects of the tumor itself and subsequent treatments, but few detect clinically-relevant, subtle impairments in cognition. Here, we aim to master and adapt a visual discrimination task that utilizes a clinically-relevant touchscreen platform to detect subtle executive function changes in female Balb/c mice for future application to our breast cancer survivor model. In this preliminary study, 8-week-old BALB/c female mice were food restricted, trained to use the touchscreens and reward mechanism to criteria, and then progressed to the visual discrimination task. After learning the "correct stimuli", the cognitive flexibility of the mice was assessed by challenging them to learn the previously incorrect stimuli as the new correct stimuli (i.e., reversal learning). Through adjustments of other visual discrimination protocols tailored to the C57BL/6 strain of mice, a new protocol has been adapted for BALB/c mice, taking into account their anxiety-like phenotype. Future application of this protocol comparing tumor survivor mice to controls aims to advance the mechanistic understanding of subtle cognitive changes that affect cancer patients and survivors alike.

47 Midkine: Mediator of Inflammation and Cell Death in the Retina

MANDY FRITSCH-KELLEHER *The Ohio State University*
ANDREW FISCHER *The Ohio State University*

Midkine (MDK), a small-heparin binding growth factor, has been widely implicated in many different biological functions including neuronal development and pathology. MDK has been implicated in retinal development and may influence the mature retina and the progression of retinal disease. Retinal degeneration involves the activation and reactivity of different types of retinal glial cells, including microglia, astrocytes, and Müller glia. Using single cell RNA-seq data from zebrafish, chick and mouse retinas, we identified damage-induced up-regulation of MDK and candidate receptors. Our analysis indicated the Müller glia transiently up-regulate MDK following excitotoxic damage. Further, we find that candidate MDK-receptors, including Ptpz, Itbg1, Pak1 and Sdc-4 are widely expressed by different types of retinal glia. To investigate MDK's effects on proliferation and cell death, we applied two doses of MDK prior to NMDA-induced damage. We found that MDK-treatment decreased the proliferation of Müller glia-derived progenitors and suppressed the reactivity and accumulation of microglia and non-astrocytic inner retinal glia (NIRG) cells. In addition, we found that cell death was significantly reduced by MDK treatment. These effects could not be replicated with the MDK-related protein, pleotrophin. To further investigate the influence of MDK-signaling in damaged retinas, we applied the Ptpz inhibitor sodium vanadate ($NaVO_3$) prior to NMDA-induced damage. Application of $NaVO_3$ exacerbated cell death, influenced glial reactivity and suppressed the proliferation of Müller glia in damaged retinas. The application of MDK with FGF and insulin, a combination of secreted factors that stimulate the proliferation of Müller glia in the absence of retinal damage, did not decrease the proliferation of Müller glia. Collectively, our findings suggest that MDK acts to influence glia-mediated neuroprotection and also the reprogramming of Müller glia into proliferating progenitor-like cells. Our results implicate MDK as a therapeutic molecule that could be applied to reduce inflammation and promote the survival of retinal neurons.

POSTER ABSTRACTS

Author index located on page 61.

1 Antidepressant-like Effects of the Muscarinic receptor Antagonist Scopolamine in Rats

MAHA RASHID *The College of Wooster*
RACHEL WATSON *University of Wisconsin-Madison*
EMILY JUTKIEWWICZ *University of Michigan Medical School*

Scopolamine is a non-selective, muscarinic acetylcholine receptor antagonist prescribed for motion sickness. Recent literature suggests scopolamine given intravenously could have possible fast-acting antidepressant effects in depressed patients. The present study evaluates the antidepressant-like effects of scopolamine in rodent models, including the forced swim test (FST) and the novelty-induced hypophagia (NIH) test. In the FST, male Sprague Dawley (SD) rats are video-recorded swimming in 25 C water for 15 min and later behaviors such as immobility, swimming, and climbing are scored. In the NIH test, single-housed SD rats are trained for three days to drink a vanilla-flavored nutritional shake and the latency to drink and volume consumed in their home cage and a novel cage is recorded. In both tests, different doses of scopolamine (0.001 – 0.1 mg/kg) or the known tricyclic antidepressant despiramine (DMI, 10 mg/kg) are administered 60 min prior to the tests. In the NIH test, acute treatment with DMI shows no decrease in latency in contrast to effects observed following chronic treatment with DMI. Acute treatment of scopolamine produced dose-dependent decreases in latency to drink with the most robust effects observed at 0.01 mg/kg. In the FST, scopolamine significantly decreases immobility at doses 3 to 10-fold larger than doses that produces significant effects in the NIH assay. In conclusion, these findings suggest that muscarinic acetylcholine receptor antagonists have potential as rapid -acting treatments for depression in comparison with the traditional reuptake inhibitors typically prescribed. This work is supported by R01 MH107499 and University of Michigan Summer Research Opportunity Program.

46 Stress-enhanced Anxiety-like Behavior is Related to GABA Signaling in the Brain and Intestine of Mice Regardless of IL-1R1 Signaling

ELIF KOLANCI *The Ohio State University*
BRETT R LOMAN *Center for Microbial Pathogenesis, Nationwide Children's Hospital*
MICHAEL T. BAILEY *Center for Microbial Pathogenesis, Nationwide Children's Hospital and Institute for Behavioral Medicine Research, The Ohio State University*

Background: The gut microbiota is likely involved in the development of emotional disorders, including anxiety and depression, but the mechanisms linking the two are not completely understood. Signaling through the IL-1 type 1 receptor (IL-1R1) contributes to stressor-induced increases in anxiety-like behavior, and gut microbiota are necessary for stressor-induced increases in IL-1. Thus, this study tests whether signaling through IL-1R1 is necessary for stress-induced increases in anxiety-like behavior in germ-free mice colonized with microbiota from a patient with an anxiety disorder.
Methods: Germ-free, adult male C57BL/6 mice (N=12) received fecal microbial transplant from an adult human with generalized anxiety disorder. Mouse behavior was tested on day 1 (marble burying and open field) and day 2 (light-dark preference and step-down) of the experiment. Following behavioral tests on day 2, half of the mice (n=3 wild type [WT; n=3], whole-body IL-1R1 knockout mice [KO ;n=3]) were sacrificed for baseline analysis. On days 3-9, the remaining mice were restrained in 50 mL conical tubes with ventilation for 2 hours at the beginning of the light cycle. Behavior testing was repeated on days 8-9 after which the remaining mice were sacrificed. Expression of genes related to inflammation and GABA signaling in brain and intestinal regions, and abundance of Lactobacillus spp. and Bifidobacterium spp. in intestinal contents were assessed via qPCR.
Results: Stress and KO independently altered anxiety-like behavior. Stress increased grooming events ($p<0.0001$) and rearing events ($p=0.0055$) regardless of genotype and increased dark preference in WT mice ($p<0.05$). Stress also increased GABAAR 1 expression in hypothalamus ($p=0.055$) and medulla oblongata ($p=0.00035$). However, this stressor-induced increase in behavior was not due to signaling through IL-1R1, since KO mice also showed stressor-induced increases in anxiety-like behavior. KO mice exhibit increased grooming ($p=0.052$), but decreased rearing ($p=0.015$). KO mice also have lower and increased expression of GABAA1 receptor in the hypothalamus ($p=0.068$) and medulla oblongata ($p=0.066$) Lactobacillus spp. in the proximal colon increased with stress in WT but decreased with stress in KO mice ($p=0.028$). Interestingly, Bifidobacterium spp. follow the opposite pattern in the cecum, proximal colon, and distal colon; stress decreased the abundance in WT, but increased abundances in KO mice ($p=0.034$).
Conclusions: Rearing and grooming behaviors exhibit different relationships to GABARA1 expression contrary to the fact that they are both anxiety-like behaviors. Stress increased the incidences of anxiety-like behaviors in both WT and KO mice, indicating that IL-1 signaling is not the sole mediator of these anxiety-like behaviors. Contrary to what we expected, KO mice demonstrated more anxiety-like behavior pre- and post-stress than WT mice as evidenced by light-dark preference as well as grooming behavior. While intestinal bacteria are affected by stress, further studies including healthy microbial transplant and female mice are needed to determine the role of the intestinal microbiota on stress-induced anxiety.

45 An Electroencephalography Investigation of the Effects of Auditory Attention on Crossmodal Temporal Acuity

CHARLOTTE BABARINSA *Oberlin College*
ZOEY KEELEY *Oberlin College*
TAWNI HOSEIN *Oberlin College*
LESLIE D. KWAKYE* *Oberlin College*

Coherent and complete perceptions of the world relies on our brain's ability to integrate multiple crossmodal stimuli from the environment into a single percept. This multisensory integration is dependant on our ability to discern small temporal differences between stimuli (crossmodal temporal acuity). Attention has been shown to affect multisensory integration, however, attentional effects on temporal acuity in particular remain unclear. Past research in our lab aimed to investigate attentional effects on temporal acuity and found that increasing attentional load has a detrimental effect on performance on a crossmodal temporal order judgment (CTOJ) task. The study used a dual task paradigm in which participants were presented with a flash and a beep at various stimulus onset asynchronies (SOAs) and were asked to report which came first, while concurrently completing a visual or auditory distractor task. While a negative effect on performance was found with increasing attentional load, the neural mechanisms underlying this effect are unknown. The current study used the same dual task paradigm with only visual distractors, along with electroencephalography (EEG) recordings from 64 scalp electrodes in order to investigate the underlying neural mechanisms of the observed attentional effect on temporal acuity. EEG data was time locked to the onset of the flash, and was averaged across participants for each perceptual load and SOA combination. Results confirmed that increasing attentional load decreases accuracy. We found differences in event related potential (ERP) amplitude with increasing visual load occurring at parietal electrodes 125 ms to 250 ms post flash. The differences in ERP amplitude across load were localized to visual cortex using a distributed source analysis method (classical LORETA analysis recursively applied, CLARA). These findings suggest that visual distractors largely interrupt early processing in unisensory visual pathways rather than multisensory pathways. Future studies will aim to investigate the effects of auditory distractors to see if similar results are obtained for auditory processing. Gaining a better understanding of the relationship between attention and temporal acuity will lead to a more complete knowledge of how our brains integrate all of the stimuli presented to us to create a cohesive picture of the world.

2 Environmental Enrichment Reduces Vulnerability to Repeated Traumatic Brain Injury

KATARINA SCHNEIDERMAN *The Ohio State University*
JULIE FITZGERALD *The Ohio State University*
SARTHAK SHAH *The Ohio State University*
KATE KARELINA *The Ohio State University*
ZACHARY M. WEIL *The Ohio State University*

Traumatic brain injury (TBI) is a major public health issue, affecting approximately two million people in the United States each year. Insult causes damage to microvasculature and cellular structures, prompting a rapid neuroimmune response characterized by inflammation and metabolic dysfunction. This series of events precipitates further damage to the nervous system and renders it more vulnerable to subsequent TBI. Repeated injury prior to the recovery of normal metabolic physiology generates more severe functional and histological pathology. Since insult has long-lasting consequences for behavior, cognition, and brain health, discerning neuroprotective strategies for recovery is of large societal importance. Housing mice in enriched environments, characterized by larger living quarters, varied toys, space for climbing and hiding, and social stimulation, has been shown to confer motor, cognitive, and histological benefits after injury in animal models of TBI. We hypothesized that housing animals in enriched environments after a mild TBI would render animals less vulnerable to a subsequent brain injury. Young adult mice received a mild, closed-head injury and were then housed in enriched or standard housing environments. One week later, mice underwent a more severe controlled cortical impact injury and were assessed for cognitive deficits, anxiety- and depression-like behaviors, motor deficits, and tissue damage. Our preliminary data suggest that housing brain injured mice in an enriched environment improves motor function, modulates depressive- and anxiety-like behaviors, and improves memory in a fear conditioning task compared to standard housing. These preliminary trends suggest that not only is recovery improved after intense physical and cognitive rehabilitation immediately after injury, but vulnerability to repeated injury may also be ameliorated.

3 Genetic Control of Neural Development: dNab2 is Required for Correct Brain Development in Drosophila Melanogaster

GARGI MISHRA *The College of Wooster*
SETH KELLY *The College of Wooster*

Intellectual disability (ID) affects about 200 million people worldwide, leaving patients with impaired cognitive and adaptive abilities. Characterized by low average IQ of 30-50 (vs. 100 for those unaffected), those with ID suffer problems with learning and memory. Increasing number of genes are being associated with various forms of ID, indicating indicating the apparent nature of genetic influence over brain development. While this correlation has been observed, the molecular mechanism underlying the genetic control of brain development remains elusive. Formation of neural networks relies on axonal pathfinding mediated by chemical signals and cues from the local neural environment. The growth cone at the axonal tip is receptive to these chemical cues, encouraging axonal extension towards or away from the target site. Any defects in axonal pathfinding could hinder correct brain development, inducing problems with learning and memory. The human ZC3H14 gene has been commonly observed to be mutated in patients with ID. The loss of ZC3H14, a poly-adenosine-tail- RNA binding protein, results in increased length of poly-A-tail, suggesting its possible role in poly-A-tail length regulation. Previous research has identified the Drosophila melanogaster Nab2 as a gene ortholog for human ZC3H14. dNab2 is required for correct mushroom body (MB) development, an axon-rich bilateral structure in the fruit fly brain responsible for learning, memory, and sleep. Given its axon-based structure, the MBs deem appropriate as a model to study axon pathfinding, and hence, the genetic control of neural development. Previous studies have shown that loss of Nab2 causes changes in gene expression, resulting in defects in brain development, learning, and memory. Adult dNab2ex3 flies show overgrowth of the past the midline of the brain. However, the timing of the defect formation is not clear (larval/pupal stage?). Immunofluorescence imaging of Nab2 null and WT showed Nab2 does play a role post-transcriptionally in controlling gene expression during development. Nab2 null larva had defected (crossing over) MB lobes. Larva with a functional Nab2 gene showed no MB lobe defects. Identification of life stages when Nab2 is required is important for identifying time points over the neural developmental period that are key to ensuring correct brain development. Knowledge of these life-stages will allow for an RNA-seq experiment in the future. This would facilitate the generation a profile showing the key protein changes (thereby highlighting control of the dynamic genetic activity) that occur over the course of, and are required for, correct brain development.

44 An Electroencephalography Investigation of the Effects of Visual Attention on Crossmodal Temporal Acuity

ZOEY KEELEY *Oberlin College*
KATHRYN HIRABAYASHI *Oberlin College*
ZOII BARNES-SCOTT *Oberlin College*
SAMANTHA PAPADAKIS *Oberlin College*
ALEXY JABBOUR *Oberlin College*
JIACHEN LIU *Oberlin College*
LESLIE D. KWAKYE *Oberlin College*

Coherent and complete perceptions of the world relies on our brain's ability to integrate multiple crossmodal stimuli from the environment into a single percept. This multisensory integration is dependant on our ability to discern small temporal differences between stimuli (crossmodal temporal acuity). Attention has been shown to affect multisensory integration, however, attentional effects on temporal acuity in particular remain unclear. Past research in our lab aimed to investigate attentional effects on temporal acuity and found that increasing attentional load has a detrimental effect on performance on a crossmodal temporal order judgment (CTOJ) task. The study used a dual task paradigm in which participants were presented with a flash and a beep at various stimulus onset asynchronies (SOAs) and were asked to report which came first, while concurrently completing a visual or auditory distractor task. While a negative effect on performance was found with increasing attentional load, the neural mechanisms underlying this effect are unknown. The current study used the same dual task paradigm with only visual distractors, along with electroencephalography (EEG) recordings from 64 scalp electrodes in order to investigate the underlying neural mechanisms of the observed attentional effect on temporal acuity. EEG data was time locked to the onset of the flash, and was averaged across participants for each perceptual load and SOA combination. Results confirmed that increasing attentional load decreases accuracy. We found differences in event related potential (ERP) amplitude with increasing visual load occurring at parietal electrodes 125 ms to 250 ms post flash. The differences in ERP amplitude across load were localized to visual cortex using a distributed source analysis method (classical LORETA analysis recursively applied, CLARA). These findings suggest that visual distractors largely interrupt early processing in unisensory visual pathways rather than multisensory pathways. Future studies will aim to investigate the effects of auditory distractors to see if similar results are obtained for auditory processing. Gaining a better understanding of the relationship between attention and temporal acuity will lead to a more complete knowledge of how our brains integrate all of the stimuli presented to us to create a cohesive picture of the world.

43 Bayesian Causal Inference Modeling of Multisensory Attention

Victoria Fisher *Oberlin College*
Margaret Jackson *Oberlin College*
Leslie Kwakye *Oberlin College*

In order to understand the world around us, we combine information across the different senses; this is referred to as multisensory integration. Integration is imperative in optimal functioning and deficits in integration have been implicated in neurological disorders such as autism and schizophrenia. Further, previous research has demonstrated that the brain integrates information in a Bayes' optimal way, such that individuals rely on previous experience to determine the most likely source or sources of stimuli. Integration of temporally disparate audio-visual stimuli has been modeled using Bayesian statistics. While attention has a well established impact on multisensory integration, leading to an increase in integration of unrelated stimuli, its effect on Bayes' optimality is unknown. Further the influence of distractor modality on integration is also unclear.
The present study investigates the influence of attentional load from different modalities on the integration of simple stimuli through Bayesian modeling. Participants were asked to determine whether temporally offset, 0-500 ms differences, flash-beep stimuli occurred synchronously or asynchronously. Participants were also simultaneously presented with rapid serial audio or visual presentation (RSAP/RSVP) streams of varying difficulty in order to direct attention away from the primary task.
Visual distractors lead to a decrease in the likelihood of assuming synchronicity (prior). Auditory distractors lead to an increase in the size of the window of asynchronies at which participants were likely to report synchronicity and a decrease in prior. Overall, preliminary data indicate that distractors affect judgements of audiovisual synchrony and that distractors from different modalities modulate this effect differently. Further analysis using a Bayesian causal inference model will investigate if these changes in multisensory integration are due to sensory noise.

4 Finding Proactive Control in the Flanker Task

Erika Shultz *Ohio Wesleyan University*
Kira Bailey *Ohio Wesleyan University*

The Dual Mechanisms of Cognitive Control theory posits that individuals adaptively use reactive or proactive cognitive control based on current task demands (Braver, 2012). The frontal slow wave ERP component has been identified as a marker of proactive control. This component is reliably observed during mostly incongruent blocks of the Stroop task (West & Bailey, 2012). In the Flanker task, detection of the slow wave has been unreliable. In two studies, we replicate the modest slow wave sometimes found in a traditional Flanker task, and then demonstrate a more robust slow wave in the mostly incompatible blocks of a proportion compatible Flanker task. The implications of these data are twofold: 1) as posited by the DMC theory, manipulating task demands influences the use of reactive and proactive control; 2) the unreliable detection of the slow wave in the traditional Flanker task may be indicative of individual differences in the use of proactive control regardless of task demands. Future research should examine what factors other than the current task demands may influence individuals to rely more on reactive or proactive control.

5 The Effects of Video Game Exposure on Cognitive Control

MICHAELA RICE *Ohio Wesleyan University*
ALEXIS LEASE* *Ohio Wesleyan University*
MALIA WALKER *Ohio Wesleyan University*
KIRA BAILEY *Ohio Wesleyan University*

This study examines the effects of brief video game exposure on cognitive control using event-related potentials (ERPs). Cognitive control is examined under the context of the Dual Mechanisms of Control theory, which proposes that cognitive control is made of two types of control, reactive and proactive. Individuals alternate between these two modes based on current task demands (Braver, 2012). Based on previous research (West & Bailey, 2012), the frontal slow wave and conflict SP ERP components index proactive and reactive control, respectively. Participants played 20 minutes of either a first-person shooter or strategy video game and then completed the counting Stroop and Flanker tasks while ERPs were recorded. Past research has shown that proactive control is negatively correlated with higher levels of video game experience, but reactive control shows little difference (Bailey, West & Anderson, 2010). We hypothesize that brief exposure to a strategy video game will improve the use of proactive cognitive control as indexed by accuracy, response time, and amplitude of the frontal slow wave. Participants exposed to the strategy game will show higher accuracy in blocks with more incongruent/incompatible trials and display more prominent slow wave activity, indicating a greater use of proactive control. First-person shooter video games, in contrast, may have no effect or may be detrimental to the use of proactive control.

42 Establishing a Neurobiological and Behavioral Model of Alzheimer's Disease in Rats

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Alzheimer's disease (AD) is a chronic neurodegenerative disease that impairs memory and other cognitive functions. Although the exact etiology of Alzheimer's disease is not fully understood, amyloid beta (Aβ) aggregation consistently accompanies AD-related behavior deficits. As humans age, Aβ proteins begin to unfold and stick to each other, forming clumps called plaques, which interfere with normal brain function. In this pilot study, we sought to replicate a model of Alzheimer's disease in rats by inducing plaque aggregation through intracerebroventricular infusion of Aβ. We sought to confirm model success in two studies by 1) histologically viewing Aβ aggregations in the brains of Aβ-infused but not vehicle-infused animals and 2) observing less accurate radial arm maze performance by Aβ-infused animals compared to vehicle-infused animals. Isoflurane-anesthetized male and female Sprague-Dawley rats (n=12) were placed in a stereotaxic apparatus, and 7.5 microliters of Aβ (25 micromolar per microliter) or its vehicle (0.1 M phosphate buffered saline [PBS]) was infused over 5 min into each lateral ventricle; postoperative analgesia (carprofen, 5 mg/kg sc) and antibiotic (gentamicin, 8 mg/kg sc) was provided on the day of surgery and for each of 3 days after. Half of the animals had been trained the week before in once daily 10-min sessions to find a piece of sweet cereal (Froot Loops) in each of the 8 arms of a radial arm maze. These rats were tested in a single radial arm maze session 7-10 days after surgery and, within 24 h after testing and along with the other untested animals, euthanized with sodium pentobarbital (100 mg/kg sc) and perfused intracardially with 4% paraformaldehyde; brains were harvested, postfixed, and sliced at 40 micrometers on a freezing microtome. Slices encompassing the hippocampus and entorhinal cortex were stained using Congo Red and viewed under Texas red florescence at high magnification (400x). Amyloid beta plaques were visible in 3 of the 5 Aβ-infused brains processed and in none of the PBS-infused rats, suggesting relatively proper stereotaxic targeting for sufficient Aβ aggregation and effective histological technique. However, radial arm maze performance was inconclusive because the baseline data obtained proved unstable. Future studies will ensure solid maze training by using food restriction to motivate performance across more training sessions. These studies will provide procedures for use in behavioral neuroscience course labs and projects for future thesis students who wish to seek AD mechanisms and treatments.

41 Exploring the Effects of Alcohol on Mice in Operant Condition

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Introduction: Alcohol use disorder is characterized by compulsive alcohol intake, or drinking despite negative consequences. Previous studies have shown that female rodents have a heightened vulnerability to drug use across different stages of the addictive cycle. In this experiment, we used an operant conditioning paradigm to test sex differences in consumption of alcohol and resistance to punishment in C57BL/6J mice.

Hypothesis: The hypothesis for this experiment was that female mice would consume more alcohol and be more resistant to a foot shock punishment when responding for alcohol.

Methods: 16 mice (8 female and 8 male) were socially housed and food restricted to 85% of their free feeding weight. The operant paradigm began with the mice nose poking for food pellets starting at Fixed Ratio 1 (FR1) and progressing to Fixed Ratio 3 (FR3) schedule. The mice next moved onto a 10% sucrose solution. After that, 10% sucrose was mixed with 10% EtOH. Then, the mice progressed to drinking only 10% EtOH solution. Then, they moved onto increasing doses of EtOH: 15% EtOH, 20% EtOH, and 25 %EtOH. Finally, the mice drank 10% EtOH until their nose pokes were consistent. Once the mice stabilized drinking at 10% EtOH, a foot shock was added on the second nose poke (poke 1 = cue, poke 2 = footshock, poke 3 = reward). This was done to model aversion-resistant intake of alcohol, or responding for reward despite negative consequences.

Conclusions: Female mice consumed more g/kg/30 min EtOH at 15%, 20%, and 25% concentrations than males. Females also responded more for 15% EtOH solution than males. Interestingly, there were no sex differences in the response to a footshock punishment. Based on these data, we can conclude that there are sex differences in alcohol consumption but not in the response to punishment.

6 Treatment of T Cells from Multiple Sclerosis Patients with Novel STAT3 Inhibitor Improves Teff:Treg Balance

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Multiple Sclerosis (MS) is an immune-mediated chronic CNS disorder in which the body's immune system attacks the fatty myelin sheath surrounding neurons, resulting in poor neuronal signaling. Myelin-specific CD4+ T effector (Teff) cells are pathogenic while T regulatory (Treg) cells beneficially suppress Teff cells in MS and its murine model, experimental autoimmune encephalomyelitis (EAE). We examined the Interleukin-6 (IL-6)/STAT3 pathway in MS pathogenesis, as dysregulation of this pathway has been observed in MS patients. IL-6 is a cytokine that initiates the transcription factor STAT3 to activate Th17 cells, thus releasing the proinflammatory cytokine IL-17. Th17 cells are a subset of CD4 Teff cells and are highly encephalitogenic when injected into wild type (WT) recipient mice. Moreover, IL-6 suppresses the generation of inducible Tregs (iTreg), resulting in an overreactive immune response to myelin antigens, or autoimmunity. We developed a small molecule inhibitor, LLL12b, of STAT3. We hypothesized that by inhibiting the IL-6/STAT3 pathway, the Teff:Treg balance of CD4 T cell responses would normalize and the progression of the disease in the EAE model of MS and in humans would be suppressed. We previously tested LLL12b in mice with EAE and found that the Teff:Treg imbalance of myelin-specific CD4 T responses was repaired and EAE was suppressed. In this study, we examined the effects of LLL12b on CD4 T cells from MS patients. Human PBMCs from 22 treatment-naïve MS patients were activated with αhCD3 for 3 days, in the presence of different concentrations of LLL12b or vehicle control (DMSO). IL-17 production in supernatants was determined by ELISA. Our data show that LLL12b significantly suppressed IL-17 production in human CD4 Teff cells from MS patients, suggesting LLL12b has the capacity to inhibit the effector function of effector/memory CD4 T cells from MS patients. Meanwhile, we also determined LLL12b significantly promoted iTreg development of CD4 T cells from the same 22 treatment-naïve MS patients via flow cytometry, indicating LLL12b has the capacity to normalize the Teff:Treg balance in human patients. Together, these data suggest the novel small molecule STAT3 inhibitor LLL12b has great therapeutic potential for MS.

7 The Role of Inflammation and Gender on Dendritic Spines and Neuron Arborization in NL-G-F Mice

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2017, over 5.3 million people in the United States age 65 and over have AD. Two -thirds of the individuals with AD are women (Alzheimer's Foundation of America, 2016). Beta-amyloid plaques and neurofibrillary tangles represent the two most well-known hallmarks of AD. Beta-amyloid is a protein coming from APP that aggregates between neurons to form plaques. The neurofibrillary tangles are aggregates from the intracellular protein Tau. Tau's function is to support microtubules inside neurons, allowing for transportation of nutrients. When Tau fails to maintain support, the microtubules' structure is destabilized and transportation of nutrients throughout the neuron is prevented (BrightFocus Foundation, 2017). This study utilizes the knock-in NL-G-F mouse model of AD to examine chronic inflammation in male and female mice. This mouse model creates the characteristic beta-amyloid production without over-expressing amyloid precursor protein (APP) (Saito, et al. 2014). We chose this model over other transgenic mouse models because it mimics more closely some of the physiological mechanisms observed in AD. We hypothesize that if chronic inflammation is induced at 12 weeks of age for six weeks, then by four months of age the female mice will accumulate overall more microglial cells and amyloid beta plaques compared to the males. Along with the presence of cognitive impairments, it is predicted these mice will have fewer dendritic processes and dendritic spines throughout the hippocampus and cortex. Inflammation will be induced with weekly intraperitoneal injections of lipopolysaccharide (LPS) for six weeks. The dendritic spine densities and morphology of neurons in the hippocampus and cortex of NL-G-F mice crossed with green fluorescent protein (GFP) will be examined via confocal microscopy. This study aims to determine the role of chronic neuroinflammation as a potential risk factor in AD progression regarding gender. The results of this study have the potential to determine whether timely targeting inflammation would be a viable therapeutic option for maintaining synapse functions leading to the prevention or delay of AD.

40 The Effect of Acute Stress on Voluntary Ethanol Consumption in BTBR T+tf/j Mice

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Autism Spectrum Disorders (ASD) are a class of neurodevelopmental disorders characterized by restricted interests/repetitive behaviors, deficits in communication and social interaction, and heightened anxiety levels. Exhibiting many of these cardinal features, the BTBR T+tf/J strain of mice are widely recognized and utilized to model for autism. Previous studies focusing on neuroendocrinological and behavioral stress reactivity in BTBR mice have shown that, depending on the behavioral stressor being used, BTBR mice may exhibit reduced, comparable or heightened stress reactivity relative to C57BL/6J (B6) mice, a standard inbred strain. With mounting evidence suggesting that heightened stress reactivity may promote excessive voluntary ethanol intake in certain strains of mice (Edwards et al. 2013), this study sought to investigate whether exposure to a specific acute stressor - the Tail Suspension Test (TST) - would affect voluntary ethanol intake in male BTBR and B6 mice. Following a preliminary experiment to test the validity of the TST as a means of eliciting an exaggerated stress response from the BTBR mice (Exp.1), the "Drinking in the Dark" drinking paradigm was used to model the effects of exposure to an acute stressor on binge-like ethanol consumption in group-housed (Exp. 2) and individually housed (Exp. 3) mice. I hypothesized that both individually and group-housed BTBR mice would, in response to an acute stressor (TST), increase their voluntary ethanol intake and that this increase would be greater than B6 mice exposed the acute stressor (TST). Experiment one showed that following exposure to the acute stressor, BTBR mice did in fact exhibit significantly heightened stress reactivity compared to B6 mice. Additionally, the results of experiments two and three suggest that although exposure to acute stressor does appear to have a disruptive effect on voluntary ethanol intake, the nature of this effect on both strains varies over time and housing conditions. Possible explanations for these observations and issues with precise measurement of fluid intake are discussed.

39 Contributions of the Nucleus Accumbens and the Medial Prefrontal Cortex to Conditioned Place Preference

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The ventral tegmental area (VTA) projects to different areas of the brain, including the nucleus accumbens (NAc) and the medial prefrontal cortex (mPFC). These distinct pathways are responsible for reward and aversion, playing a role in neuropsychiatric disorders including addiction. The current study seeks to understand how dopamine neurons projecting from the VTA to the NAc shell and from the VTA to the mPFC contribute to reward and aversion. This study was conducted by using retrograde Designer Receptors Exclusively Activated by Designer Drugs (DREADD) injected into the NAc shell and the mPFC to excite these pathways and examine effects on conditioned place preference (CPP). The retrograde viral vector AAVrg-hSyn-hM3Dq-DIO-mCherry (Addgene) was injected into the NAc shell or mPFC of mutant mice expressing Cre recombinase under control of the promoter for the dopamine transporter (DAT-Cre). This resulted in expression of the excitatory DREADD hM3Dq specifically in dopamine neurons projecting to the NAc shell or mPFC. Control animals were wild type littermates of the DAT-Cre experimental mice that received sham surgeries with saline injected into the NAc shell or mPFC. Following recovery, mice were individually placed into a two-chambered conditioned place preference box for 25 minutes with free roam of both sides. The movement of the mice was recorded and the percentage of time spent in each chamber was calculated. Over the following four days, mice were conditioned by receiving the DREADD ligand clozapine before being confined to one side or receiving vehicle before being confined to the other for 10 min. Mice were randomly assigned to which room was paired with the clozapine. On testing day, mice were individually placed in the box again with free roam of both sides and the percent of time spent in each side was calculated. Results showed that neither mice with expression of hM3Dq in the VTA→NAc shell nor mice with expression of hM3Dq in the VTA→mPFC developed a significant preference when compared to controls. It is unclear why results were inconclusive. First, it is possible that the DREADD method did not work. To test this, histology must be examined to verify adequate expression of the virus. Second, hM3Dq may not be the optimal means to activate the pathway since it is a metabotropic receptor. In short, it may not be strong enough in the absence of another rewarding or aversive stimulus. Future research may examine differential activation in the presence of other stimuli.

8 Binge Drinking Has No Significant Effect on Hippocampus Volume in Young Adult Women

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Many adults consume alcohol recreationally, however the consumption of large amounts of alcohol can have negative effects on the brain. In this study, the MRIs of 24 healthy, right handed female participants between 20 and 30 were examined to measure hippocampal volume. Participants were categorized as binge drinkers (>3.5 drinks in one sitting) and non-binge drinkers (<1 drink in one sitting), while controlling for other forms of drug use or abuse. It was hypothesized that the women who were categorized as binge drinkers would have a smaller hippocampus than those who were non-binge drinkers, based on previous studies suggesting cell reduction due to excessive consumption of alcohol. For our demographic variables, univariate ANOVAs revealed no significant difference between groups for age, total abuse as measured by the Childhood Trauma Questionnaire, total sessions of marijuana, and total hits of cocaine ($p>0.05$). By design, there was a significant difference between groups on number of drinks in one sitting ($p< 0.001$). Medical Image Processing, Analysis and Visualization software was used to trace the hippocampus on alternate slides from the first superior slice the hippocampus was visible until the hippocampus disappeared inferiorly. Total volume was calculated by summing the area of each slice and multiplying by two. No significant difference was found between groups on hippocampal volume ($p> 0.05$). These results did not support our hypothesis that binge drinking would reduce hippocampal volume. Future studies should be conducted with a larger participant pool and more drinker per evening in order to further study this relationship between high alcohol consumption and hippocampal volume.

9 Heterozygous Mutant Huntingtin Promotes Cadmium Neurotoxicity Via Impaired Metal Transport, Caspase Mediated Apoptosis, Erk and PKCd Dependent Oxidative Signaling Mechanisms: Relevance to Pathogenesis of Huntington's Disease

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Huntington's disease (HD) is functionally linked to environmental factors, dyshomeostasis in levels of metals and cigarette use. Interestingly, one of the most abundant heavy metals in cigarettes is cadmium (Cd), which also accumulates in the striatum and causes neurotoxicity upon exposure. Thus, we hypothesized that heterozygous mutant huntingtin (heterozygous HTT), found in most HD patients, in combination with Cd exposure would cause neurotoxicity and neurodegeneration via increased intracellular accumulation of Cd and activation of oxidative stress signaling mechanisms in a mouse striatal cell line model of HD. We report that heterozygous HTT striatal cells are significantly more susceptible to Cd-induced cytotoxicity as compared to wild-type HTT cells upon exposure for 48 hr. The heterozygous HTT and Cd-induced cytotoxicity led to a NADPH oxidase (NOX) mediated oxidative stress that was attenuated by the antioxidant, ascorbic acid, and a NOX inhibitor, apocynin. Heterozygous HTT coupled with Cd exposure caused increased expression of protein kinase C d (PKCd) and other key oxidative stress proteins levels, enhanced the activation of caspase-9 and caspase-3 mediated apoptosis, and blocked the overexpression of extracellular signal-regulated kinase (Erk). We observed significantly greater intracellular accumulation of Cd and reduced expression of divalent metal transporter 1 (DMT1) protein in the heterozygous HTT striatal cells upon exposure to Cd. Pre-treatment with zinc, manganese and iron as well as antioxidant agents significantly attenuated the Cd-induced cytotoxicity. Collectively, these results demonstrate that heterozygous HTT exhibits neurotoxic properties upon Cd exposure to cause cell death via caspase mediated apoptosis, altered metal transport, and modulation of Erk and PKCd dependent oxidative signaling mechanisms.

38 Identification of the Molecular Targets of Caffeinated Alcoholic Beverages

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Alcohol is widely abused by adolescent and college age individuals, and when used excessively can be detrimental to health and decision-making. Alcoholic drinks mixed with caffeine have become more common and may amplify the effects of alcohol alone, worsening the health and social problems typically caused by just alcohol. However, the neural mechanisms by which this drug combination causes these effects are unknown. Therefore, the objective of this project is to determine the unique profile of molecular changes in the brain that result from caffeinated alcohol use, compared with alcohol and caffeine alone. Male adolescent Long Evans rats were allowed to voluntarily self-administer alcohol, caffeine, caffeinated alcohol, or control solutions daily from postnatal day 30 to 50. On the day following completion of self-administration (postnatal day 51), animals were euthanized and tissue punches collected from the nucleus accumbens and orbitofrontal cortex, two brain regions associated with addiction and decision-making. To determine how caffeinated-alcoholic beverage (CAB) intake changes molecular targets within these regions, expression levels of RNA associated with neurotransmitter synthesis, re-uptake, degradation, and receptors were measured using a Targeted RNA Expression analysis (T-REx). Neurotransmitter systems explored included systems associated with neural excitation and inhibition (glutamate/GABA), mood regulation (serotonin), reward/addiction (dopamine), and wakefulness (adenosine). We found that expression levels varied by region and drug condition, but indicated that alcohol and caffeine interact via complex molecular targets to induce a different profile of behavioral effects. This pattern may make addiction more likely or complicate treatment. Our results clarify our understanding of the CABs potentially leading to new treatments targeting the pathways identified, and the identification of risk factors for students at risk of addiction.

37 CRF_2 Receptor Regulation of Anxiety-like Responses Following Long-Term Alcohol Withdrawal

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Corticotropin-releasing factor (CRF) is one of the main regulators of anxiety during ethanol withdrawal. The CRF receptor subtypes appear to have a differential role in regulating anxiety, where CRF_1 receptor activation may increase stress-related behaviors and CRF_2 receptor activation alleviates this response. Previous research has demonstrated that urocortin 3 (Ucn 3), which selectively activates the CRF2 receptor, may reduce anxiety -like behavior during acute alcohol withdrawal. The current study sought to investigate the role of CRF_2 receptors in alleviating anxiety-like behaviors following protracted abstinence from ethanol. Female and male Wistar rats were fed an ethanol or control liquid diet for approximately 4 weeks. Upon removal of the diet, rats were assessed for signs of physical dependence and were then left undisturbed for 5 weeks. At the end of this abstinence period, rates were injected with Ucn 3 or vehicle and were then tested for anxiety-like behavior in the elevated plus maze. There was a significant interaction between ethanol diet condition and Ucn 3 dose on the percentage of time spent exploring the open arms. A post hoc Fisher's test showed that ethanol diet-fed rats spent significantly less time in the open arms, an effect that was reversed by injections of Ucn 3. Our findings support the hypothesis that activation of CRF_2 receptors decreases anxiety-like responses following long-term ethanol withdrawal.

10 A Bayesian Reanalysis of Correlations Between Cortical Thickness and Strategic Choices in Economic Games

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Yamagishi et al. 2015 examined correlations between dorsolateral prefrontal cortex (DLPFC) thickness and giving in two economic games (dictator and ultimatum) and strategic behavior in a Machiavellian game (MG). Their Results showed DLPFC thickness was correlated with selfish behavior in the dictator game (DG) and strategic behavior in the MG. If DLPFC thickness is a sign of increased control, it implies that the selfish behavior seen in the DG is a result of such control. This, in turn, suggests prosocial behavior may not require planned control. These findings run counter to the usual interpretation of human behavior in economic games (that prosocial behavior requires control), and the significance level of their findings was higher than prescribed by recent literature (Benjamin et al., 2017). I reexamined the data from a Bayesian perspective and present a number of Bayesian model fits to the data. I fit two linear models to the residual differences for a linear regression of the cortical region data adjusting for age, sex, and intracranial volume. One model accounted for differences resulting from both games and the other model presumed the game had no effect. The Bayes factor for these modelS showed only slight anecdotal evidence for the former model and a 95% posterior credible set of the means included 0. This suggests the thickness of the DLPFC does not correlate or predict performance economic games in either a positive or negative fashion. Thus my models support neither the idea prosocial nor selfish behavior require cognitive control.

11 Mild Traumatic Brain Injury Increases Vulnerability to Stroke in Mice

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Traumatic Brain Injury (TBI) is a leading cause of death and disability in the United States, contributing to 30% of all injury deaths. Characterized as a complex injury resulting from sudden trauma to the brain, TBI is responsible for causing direct damage to the nervous system and renders the brain more vulnerable to subsequent insults. Prior studies show that TBI is also a key risk factor for subsequent strokes; moreover, individuals that experience a TBI could be more likely to have a stroke capable of producing more damage. Although the mechanism by which TBI increases vulnerability to stroke is not fully understood, evidence points towards TBI-induced impairments in metabolism, a well-known risk factor for stroke. In this study, this relationship between TBI, impaired neurometabolism, and vulnerability to subsequent stroke was explored. This was done through an adult male mouse model, where first, a mild closed head TBI was performed, followed by an induced middle cerebral artery occlusion (MCAO) one week later. Mice that had initially experienced a TBI showed larger infarct sizes that had more than tripled as compared to mice that received a MCAO without prior TBI. In addition, mice that had received both TBI and a MCAO exhibited greater functional deficits, along with more prominent neuroinflammation. Administration of a drug focused on reducing neurometabolic impairments (Pioglitazone) immediately after TBI prevented the TBI-induced worsening of stroke outcomes. These findings are crucial to public health showing that not only are TBI patients more likely to experience strokes, but also that these subsequent strokes are shown to be much more severe, causing greater tissue damage and functional impairments. In addition this study gives insight into the powerful potential of manipulating and reducing neurometabolic impairments after TBI as a method of alleviating the risk of more severe subsequent strokes.

36 Drugs Use Influences Recognition of Emotion in College Students

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Various factors influence expression of emotion. In social situations, behaving appropriately requires accurate discrimination of emotional expression. Inaccuracy in recognition of facial expressions is closely associated with psychiatric disorders. In this study, we examined factors that influence emotion recognition among college students and also examined the effects of drug use on discrimination of emotions, focusing primarily on opiates and marijuana. Opiates are known to suppress brain function through mu-receptors. Marijuana is mildly analgesic and produces hallucinogenic effects through cannabinoid receptors. Subject were student volunteers at Morehead State University. DNAVA2 task measured emotion recognition. This task consists of four subsets of emotion-related stimuli, including visual stimuli (48 trials, faces of adults or children) or auditory stimuli (48 trials, voices of adults or children). Drug effects on accuracy were measured. A drug survey measured frequency and duration of drug use. Overall, college students could more readily discriminate adult and child facial expressions conveying positive emotions rather than negative ones, with no overall sex differences. Such pattern depended on the type of emotion and the stimulus subjects: students could discriminate 'sad' faces of children with a high accuracy. Thus, among college students the ability to discriminate emotions varies with the nature of the emotional stimuli--stimulus type (adult or child), sensory modality (face or voice), and emotional category (positive or negative), Compared to controls, frequent drug users (>3 times/week) of either opiates or marijuana made more errors. A pattern of error also depended on emotion category, with a greater accuracy for happy expressions than for angry or fearful ones. The present findings provide evidence that frequent drug use impairs the ability to process emotions, particularly negative emotions, thereby reducing accuracy in discrimination of emotional expressions. Further study on long-term drug use on emotion recognition is warranted.

35 Left Caudate Nucleus Volume Remains Constant in Women Between the Ages of 20 and 40

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While it is known that the brain decreases in total volume as an individual ages, further research is required to establish the precise location of such loss. Thirty-two right handed women with no serious medical illnesses were divided into two age groups: 20-21 years and 30-42 years. We hypothesized that older women would show a decreased caudate nucleus volume compared to their younger counterparts. To investigate this hypothesis, brain MRIs from unrelated research were selected based on participants' intelligence (Wonderlic Personnel Test), childhood abuse (Childhood Trauma Questionnaire), drinks taken per year, and drug use. There was no significant difference between groups in Wonderlic IQ score, total abuse, number of alcoholic drinks per year, or marijuana joints smoked ($p > 0.05$). By design, there was a significant difference in age between groups ($F = 275.616$, $p < 0.001$). There was also a significant difference in the pack years smoked for the older participants ($p = 0.017$), so this variable was covaried when analyzing caudate volume. Using the Medical Image Processing, Analysis and Visualization application, the left caudate nuclei of these individuals were traced in alternate slices. The areas were summed and multiplied by two to calculate total left caudate volume. Caudate volume was not significantly different between groups based on age ($p > 0.05$). Future research could be done to expand the number of participants and to collect data from a wider age range.

12 Past Pregnancy has No Effect on Hypothalamic Volume

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Pregnancy and the hypothalamus have been directly linked, but few studies have demonstrated how the hormone secretion from the hypothalamus during pregnancy affects the volume of that region. Twenty-four right-handed participants, between the ages of 23-34, with no serious medical illness were divided into two groups: women who've had no children, and those who have had at least two children. We hypothesized that women with two or more biological children would have larger hypothalami than those who had never pregnant. Univariate ANOVAs revealed no significant difference between groups for drinks per year, total childhood abuse level (Childhood Trauma Questionnaire), and intelligence (Wonderlic Personnel scores; $p > 0.05$). By design, there was a significant difference between groups for number of pregnancies ($F(1, 24) = 0.013$, $p = 0.05$). We used the Medical Image Processing, Analysis & Visualization software to trace individual slices of the right hypothalamus in all slices in which it appeared. Each slice was traced twice, averaged, and summed to calculate total hypothalamic volume. There was no significant difference in hypothalamic volume between the two groups ($p > 0.5$). It would appear that there is no difference in hypothalamic volume regardless of past pregnancy. Future studies could measure hypothalamic volumes in women when they are pregnant to see if temporary volumetric changes occur.

13 Sodium and Potassium Channel Expression During Neuronal Regeneration Following Spinal Cord Injury in Lamprey

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In humans, spinal cord injury results in lifelong impairment, including chronic pain and paralysis, with no effective treatment. Lampreys are primitive vertebrates that have the beneficial capacity to regenerate and repair their spinal cords following injury. Various signaling pathways play a role in this regenerative process. Understanding the mechanisms of lamprey spinal cord regeneration may lead to more effective human therapies. These mechanisms may include changes in the expression of voltage-gated sodium and potassium channels. We hypothesized that following injury spinal cord axons would show decreased expression of voltage-gated sodium channels and increased expression of voltage-gated potassium channels, reducing the overall excitability of the recovering neurons and reducing their susceptibility to excitotoxicity. To test this hypothesis, we transected the spinal cords of larval lampreys and measured changes in the expression of Nav1.2 and Kv1.2 channels, at locations in the spinal cords above and below the injury, at several time-points after injury, using immunofluorescence microscopy and image analysis of pixel intensities. Above the injury, both Nav1.2 and Kv1.2 expression decreased during the first week of recovery. Below the injury, Nav1.2 and Kv1.2 expression showed complex time-dependent changes, with a significant decrease in Kv1.2 expression after three weeks of recovery. Thus, some results supported our hypothesis but some did not. Current and future studies include testing channel expression in additional spinal cord sections and more sophisticated image analysis.

34 Traumatic Brain Injury-Induced Neuronal Damage in the Somatosensory Cortex Causes Formation of Rod-shaped Microglia that Promote Astrogliosis and Persistent Neuroinflammation

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Traumatic brain injury (TBI) is a leading cause of morbidity and mortality worldwide. There are about 3.8 million reported head injures per year and there are currently 5.3 million Americans living with the chronic effects of head injury. Many of these injuries are classified as diffuse brain injury, which results when impact to the head causes the brain to move back and forth within the skull. While these injuries lack gross tissue cavitation and necrosis, they are characterized by widespread microscopic injury to neurons. Patients experience myriad symptoms in the acute phase, including sensory deficits and seizures. While many of these acute effects of TBI resolve, there is also increased risk of developing neuropsychiatric complications (depression and dementia) and neurodegenerative disease. Nonetheless, it is unclear how or why TBI causes these chronic complications. One possibility is that unresolved inflammation within the brain may underlie neuropsychiatric and neurodegenerative complications that progress after injury. Therefore, we hypothesize that microglia, innate immune cells resident to the brain, become dysregulated after TBI and play a critical role in perpetuating chronic inflammation following injury. For instance, microglia respond immediately to TBI by releasing inflammatory mediators and phagocytizing debris. Moreover, there are unique sub-populations of microglia, including "rod-microglia" that are detected after TBI and in other chronic neuroinflammatory conditions (i.e. Alzheimer's disease and infection). Here, the goal was to evaluate the inflammatory capacity of microglia after TBI and focus our attention on the origin, alignment, and microenvironment of these unique rod-microglia. To complete this objective, midline fluid percussion injury was used in C57BL6 mice. This TBI model recapitulates the widespread axonal injury observed following diffuse TBI in humans. Here we show that rod microglia were derived from resident microglia and developed with only limited proliferation. Moreover, we used Thy1-YFP transgenic mice to visualize neurons and show novel data that rod-microglia aligned with apical dendrites of layer V pyramidal neurons and express CD68, a marker of phagocytosis. This alignment occurred in regions where TBI caused neuronal axotomy (ATF3+) and dense astrogliosis. To assess micro environmental changes, mRNA levels of key inflammatory and immune mediators (nanoString analysis) were determined 8 h and 7 days after TBI on the cortex. There was a clear progression from an acute inflammatory phase to a distinct chronic phase of inflammation. Based on these data, we used a CSF1-receptor antagonist (Plexxikon 5622) to eliminate microglia prior to TBI. Elimination of microglia prior to TBI and throughout the post-injury period did not affect TBI-induced axotomy, but attenuated astrogliosis. Furthermore elimination of microglia ablated injury-related increases in inflammatory and immune genes (complement, interferon-related, chemokines, antigen presentation, toll-like receptor signaling). In summary, we show that microglia are critical mediators of diverse TBI-induced inflammatory and immunomodulatory signaling cascades, including activation of other glia (astrocytes). Furthermore, we show that rod-microglia respond directly to injured neurons, align in a unique pattern, and orchestrate the neuroinflammatory response following TBI that persists well after the initial injury.

33 Cell Phones Have Our Attention: Effects of Cell Phone Distraction on the P300 Response in an Auditory Oddball Paradigm

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The purpose of the present study was to examine how the expectation for communication on a smartphone influences neuroelectrical correlates of attention, primarily the P300 ERP. Thirteen neurotypical participants (age range = 19-24 years) completed a two-tone auditory oddball task while undergoing high-density event related potential (ERP) recording. One group of participants performed the task in the presence of a smartphone and were told they may or may not receive a text message (n=7), while the other group did not encounter a phone during testing (n=6). There were no significant between-groups differences in accuracy of responding (i.e., no response to frequent tones, button press to target tones) or response time to target tones. Participants in the presence of a cell phone did not produce diminished P300 amplitudes compared to the control group. Opposite of the hypothesized relationship, a marginal effect was observed in which P300 peak latencies were faster among participants in the presence of a cell phone than those in the control group. A noteworthy limitation was the small group sizes, limiting statistical power which may have prevented detection of significant differences in P300 amplitude between frequent and target tones as well as between groups. This preliminary analysis confirms that the research design is effective for investigating the effects of cell phone presence on attention in college students.

14 Hormone Receptor Distribution in a Rhythmic Motor System

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Neuromodulators can bind at multiple sites to elicit physiological changes. This includes hormonal neuromodulators acting within the central nervous system as well as the periphery to modulate rhythmic behaviors such as chewing and locomotion. Central modulation occurs when neuromodulators bind to receptors on central pattern generator (CPG) neurons, the circuit neurons generating rhythmic patterns, and/or on motor neurons. This modulation can alter the strength or pattern of activity, triggering a different muscle response (Fort et al., J Neurophysiol, 2007). Peripheral modulation occurs when neuromodulators bind to receptors on muscles, which causes the muscle to interpret motor neuron activity differently. We are interested in how combined central and peripheral modulation alters motor behavior. We used the well-described pyloric (food filtering) and gastric mill (chewing) motor systems in the crab, Cancer borealis to address this inquiry. All CPG neurons, most of which are also motor neurons, and the muscles they innervate are identified for these two systems, allowing us to determine the extent of central and peripheral modulation within complete motor systems. The neuromodulator Crustacean Cardioactive Peptide (CCAP) acts hormonally to modulate both the pyloric and gastric mill motor systems. All CCAP central targets are identified and include a subset of the pyloric and gastric mill CPG/motor neurons (Garcia et al., J Neurosci, 2015; Kirby and Nusbaum, J Neurophysiol, 2007; Weimann et al., J Neurosci, 1997). Some peripheral CCAP targets have been identified (Weimann et al., 1997), but for most of the 34 muscles in the pyloric and gastric mill systems, whether they are peripheral targets remains unknown. We aimed to identify all the peripheral targets to compare them to central targets. We hypothesized that all muscles in the gastric mill and pyloric systems contain CCAP receptors, enabling them to undergo peripheral modulation regardless of whether they are innervated by neurons that undergo central modulation.
To test our hypothesis, we removed pyloric and gastric mill muscles (N=29 muscle types, n=1-26 of each type) from the crab foregut. RNA of each muscle was isolated, reverse transcribed to cDNA, and analyzed using quantitative real time polymerase chain reaction (qRT-PCR). We found that muscles innervated by all the pyloric and gastric mill CPG/motor neurons had some level of CCAP mRNA and therefore could be targets of peripheral CCAP modulation (n=1-26). However, we found that the receptor expression levels varied between muscles from an average mRNA copy number of 237 in muscle p11 (n=5) to 10089 in muscle cv1 (n=13). These findings support our hypothesis that all muscles are capable of peripheral modulation regardless of whether they are innervated by neurons that undergo central modulation. Overall, our data suggest that unlike central hormonal actions which are selective for particular neuronal targets, peripheral hormonal actions may vary in magnitude, but can potentially affect the entire musculature.

15 Role of Perineuronal Nets in Reducing Memory Interference in Mice

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Perineuronal networks are extracellular structures involved in the formation of long term memory and in the closing of critical periods in development. They may also be involved in decreasing the retrieval time for similar memories, thereby, reducing memory interference. In this experiment, we tested whether perineuronal nets (PNNs) are necessary for reducing memory interference. To do this, we trained mice on an olfactory perceptual learning task with three phases: pre-test, enrichment, and post -test. In the pre-test, we tested the mice's native ability to discriminate between two enantiomers of limonene (highly similar odors). Immediately after the pre-test, half of the mice were infused with Chondroitinase ABC, an enzyme that degrades PNNs, and the other half infused with a vehicle. During the enrichment phase that followed, half the mice from either infusion groups were exposed to +/- limonene and the other half were exposed to mineral oil for a period of fourteen days for 1 hour a day. We expected that the mice with degraded PNNs and were exposed to +/- limonene during would not be able to discriminate between the two odors during the post-test. The experiment yielded inconclusive results which could be attributed to the low sample size and added variance caused by different mice handling methods of different experimenters. We are currently collecting more data for this experiment.

32 Cognitive Decline in Streptozotocin and Fructose Induced Diabetic Rats

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Alzheimer's disease is a neurological degenerative disease that appears to be due to accumulation of metabolic waste products that include phosphorylated tangles and amyloid beta plaques. However, the etiology on how the waste products accumulates is not clearly understood. Recently a waste clearance system, called the glymphatic (glia lymphatic) system, is proposed to be disrupted and may lead to progression of Alzheimer's disease (AD). The current hypothesis is that insulin insensitivity may alter cell waste clearance. Aquaporin-4 (AQP4) is a channel present in astrocytes that is critical to move fluid through the brain to remove waste. When stress or injury occur such as in diabetes type II, the AQ4 proteins may redistribute away from the blood brain barrier where it is typically located. By examining the expression of AQP4, the effects of waste accumulation can be processed and applied towards the understanding of AD. To test the effect of diabetes on AQ4 channel distribution, Streptozotocin (STZ) was injected to destroy pancreatic islet cells. Additional animals were fed high fructose diet for 12 weeks to induce diabetes type 2. Following treatment, animals were tested for cognitive function with the Morris water maze and object recognition. Following behavioral analysis, brains were collected, fixed and sectioned. Fluorescent immunocytochemistry detected AQ4 and phosphorylated Tau in control and diabetic brains. The distribution of AQ4 in the hippocampus and the presence of phosphorylated Tau tangles were measured. In addition, the size of the hippocampus was measured through unbiased stereology.

31 Instrumental Learning, Motivational Behavior, and Behavioral Flexibility in Adolescent Rats Following Pediatric Brain Trauma

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Traumatic brain injuries (TBIs) affect 2.5 million individuals in the United States each year, ranging from mild concussions to severe trauma or death. With 500,000 yearly emergency room visits being attributed to childhood-acquired brain trauma (under 14 years of age), patients endure long-lasting cognitive, physical, or behavioral effects of TBI. Long term behavioral alterations in children and adolescents can include aggression, hyperactivity, anxiety, depression, as well as changes in personality. Due to its prevalence and severe behavioral consequences in children, it is vital to consider the effect TBI has on the development of executive processes, such as learning rates, motivation, and behavioral flexibility, as one transitions from childhood to adolescence. We aimed to examine the consequences of a childhood TBI on measures of goal-directed behavior, impulsivity, and executive function during adolescence. We used two tasks that investigate similar brain constructs responsible for attentional processes in preclinical models, namely an instrumental learning task (ILT) to measure motivation and learning ability to obtain a food reward (i.e. sucrose pellet), followed by the attentional set-shifting test (AST), involving perceptual rule dimensions such as odor or medium. We hypothesized that rats subjected to TBI will display task-dependent impairments in motivated behavior and behavioral flexibility. We examined the effects of moderate parietal lobe (2.2 mm tissue deformation depth at 4 m/sec) or sham controlled cortical impact injury to the right hemisphere in pediatric Sprague-Dawley rats [postnatal day (PND) 17] on behavior during adolescence. After ten days of recovery, they were trained on a fixed-ratio schedule of 1 for 12 consecutive days in operant chambers fitted with three nose-poke holes and a food trough, by learning to poke for sucrose pellet reinforcement in the center when illuminated. Each session lasted 99 trials or 30 min, whichever occurred first. Outcome measures included the number of total trials completed, task-irrelevant pokes (left or right), and latency for pellet retrieval following instrumental nose poking. Rats were then trained/tested on AST at PND 42-43, which involves a series of increasingly difficult stages, including simple and compound discriminations, stimulus reversals, and intra/extra-dimensional set-shifts. Dependent measures included the number of trials to reach criterion, as well as total and perseverative errors. Statistical analyses will employ repeated-measures ANOVA followed by Newman-Keuls post hoc for individual test days when appropriate. Preliminary ILT data (n=8/group, test days 1-5) paradoxically suggest reduced exploratory drive but also reduced impulsivity in adolescent male rats after pediatric TBI, albeit AST results unveiled TBI-induced deficits in total trials and total errors on the compound discrimination and intra-dimensional shift stages. This could advocate for the idea that a parietal TBI during childhood may disrupt the brain structures the modulate attention, distractibility, and impulsivity control in a normal adolescent brain.

16 Endocannabinoid Agonism Increases Sucrose Intake without Impacting Sucrose Detection

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Feeding increases after exogenous cannabinoid administration, and our body contains endocannabinoids (ECB) that may act on feeding and taste. ECB receptors are found in taste buds, and ECB activation increases whole taste nerve responsiveness to sweeteners in mice. My study hypothesizes that ECB agonism increases taste-guided intake of sucrose by rats and will allow rats to more easily discriminate between the taste of sucrose and water. Two experiments were conducted, each with 5 male Sprague-Dawley rats: 1) 30-min intake of 0.03M sucrose by nondeprived rats after injection with ECB reuptake inhibitor AM404 (2.5mg/kp ip) or vehicle (30% EtOH in water) and 2) sucrose-water discrimination after AM404 and vehicle across 5 sucrose concentrations by rats trained in a 2-response operant taste detection. As hypothesized, AM404 doubled sucrose intake. This suggests that ECB reuptake inhibition is able to impact taste-guided behavior in a need-free state and that the chosen AM404 dose was effective in changing taste-guided behavior. In contrast to our hypothesis, AM404 did not enable rats to more easily discriminate between sucrose and water. This suggests that ECB reuptake inhibition does not impact behaviorally assessed sucrose detectability and that the effect of AM404 on sucrose intake may be the result of its other motivational or hedonic impacts on behavior. Our data contrast with the increase in whole-nerve sweetener sensitivity after ECB receptor agonism in mice, which may be due to an incongruency between electrophysiological and behavioral results, differences between rats and mice, and/or influence of direct vs. indirect ECB receptor agonism.

17 Insight to How Mitochondrial Regulation and Targeted Transport May Aid in Axonal Regeneration After Injury

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After a spinal cord injury, mitochondria are observed to undergo unusual changes such as the imbalance of fission and fusion, degradation, fragmentation and dysfunctional axonal transport. Importantly, the disfunction of mitochondrial transportation leads to mitochondrial localization, resulting in axonal blebbing and the retrograde transportation of healthy mitochondria for degradation. Evidence has suggested that the transportation of healthy mitochondria to the distal end of the axon positively contributes to neuronal survival and axonal regeneration after an injury. This experiment sought to test this hypothesis by creating a vector, utilizing the Armcx1 gene, that would then be transfected into an AAV virus. This gene has been shown to increase mitochondrial transportation within axons, positively correlating with the inhibition of cell death and enhancing axonal regeneration after a spinal cord injury.

30 The Effects of the Antidepressant Fluoxetine on the Gene Expression of Mice

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Selective Serotonin Reuptake Inhibitors (SSRIs) are a common class of drug used to treat depression and anxiety disorders. Use of SSRIs has been prevalent in many pregnancies, with up to 8% of women in the U.S. using SSRIs in pregnancy. However, recent studies have found the use of SSRIs during pregnancy can lead to learning deficits (such as anxiety disorders, depression, and autism) in exposed children. The SSRI, Fluoxetine (Prozac) was studied in this experiment. It was hypothesized that Fluoxetine, when administered prenatally and perinatally, may alter the expression of genes related to the serotonin pathway in mice pups and lead to autism-like behaviors. This was tested by treating pregnant mice with Fluoxetine, observing their offspring for behaviors consistent with autism and then analyzing the expression of genes associated with serotonin reuptake. The genes chosen were Tryptophan Hydroxylase 2 (TPH_2) gene and the NMDA receptor (NMDA) gene, with beta-actin gene used as a control. The gene expression was analyzed by extracting RNA from the pup brain, creating cDNA via RT-PCR, and then analyzing intensity of the DNA band via Gel Electrophoresis. Preliminary studies found no changes in gene expression between exposed and non-exposed mice. However, study of this topic using a more quantitative method is ongoing.

29 Characterizing the Effects of Melatonin on Striatal Dopamine Neurotransmission

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Dopamine neurotransmission is necessary for both movement and reward -based learning. Additionally, depletion of brain dopamine, which occurs in Parkinson's disease, results in severe motor deficits and circuit dysfunction the striatum, which receives dopamine input. Parkinsonian patients and their caregivers report a 'sundowning' effect characterized by cognitive abnormalities during evening hours, suggesting a potential circadian cycle of brain dopamine levels. Previous studies have confirmed that striatal dopamine concentrations, transporters, and release kinetics oscillate on a 24-hour cycle. In addition, dopamine neurons are known to express receptors for the neurohormone melatonin, which is involved in entrainment of circadian cycles and in the regulation of sleep-wake timing. The interaction between these systems, however, is unknown. We are therefore investigating the effects of melatonin on dopamine signaling using fast-scan cyclic voltammetry (FSCV) to measure evoked dopamine responses in the mouse striatum. Although we and others have found melatonin to foul electrochemical electrodes under a variety of FSCV waveforms, adhering to the electrode surface and drastically impacting sensitivity, we have been able to limit these effects. We have characterized fouling at both a traditional FSCV waveform, a modified waveform recently used to detect melatonin, and amperometry both in vitro and in vivo. Our results suggest melatonin may decrease evoked dopamine responses in the striatum, suggesting a hormone-driven circadian cycle of dopamine levels.

18 Sex Differences in the Regulation of Phosphodiesterase 10A by the Molecular Components of the Stress Response

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Stress can increase alcohol intake over the long term, promoting relapse in otherwise abstinent individuals. Females are more susceptible to stress-related disorders, likely due to greater stress-induced neuroadaptations. Previous studies implicated phosphodiesterase 10A (PDE10A) in the amygdala and prefrontal cortex as a stress- and alcohol-responsive protein that may contribute to long-lasting effects of stress on alcohol intake. PDE10A is a dual-specificity phosphodiesterase that can inhibit both cAMP and cGMP. Regulation of PDE10A by molecular components of the stress response, specifically activation of adrenergic and corticosteroid systems, and subsequent signaling events were unknown, as were sex differences in these effects. We hypothesized that repeated activation of adrenergic or corticosteroid systems would replicate the effects of repeated stress to increase PDE10A in the amygdala and prefrontal cortex, moreso in females than males, and that activation of the mitogen-activated kinase (MAPK) pathway, downstream of cGMP, might be similarly altered. To pharmacologically mimic components of the stress response, male and female Wistar rats (n=8-12/group) were treated with 5 mg/kg corticosterone (CORT), 2.5 mg/kg yohimbine (YOH), or their vehicles once daily for 3 days, and brains collected 24h later. Expression of PDE10A, pERK, and ERK were measured in synaptically enriched protein lysates using Western Blot. In the basolateral amygdala (BLA), where Pde10a mRNA was previously shown to increase following multiple stressors, CORT only reduced synaptic PDE10A in males. Conversely, YOH did not significantly alter BLA synaptic PDE10A, but levels were higher in all females. In the central amygdala (CeA), CORT reduced PDE10A compared to control in both sexes but YOH did not. No significant effects were seen for PDE10A in the ventromedial prefrontal cortex (vmPFC). Unlike PDE10A, CORT or YOH altered ERK phosphorylation (pERK) in all regions studied. In BLA, pERK was reduced by 3d CORT and 3d YOH. In CeA, CORT decreased pERK but YOH did not, with lower pERK in all females in the YOH experiment. CORT and YOH effects also differed in vmPFC, where CORT, but not YOH, reduced pERK in males and tended to increase pERK in females. Together, these data show no evidence to support PDE10A regulation of pERK across the regions, but do support differential regulation of PDE10A and pERK at the synapse by repeated activation of adrenergic vs. corticosteroid systems. The results do not replicate changes in Pde10a mRNA observed previously in several models of past stress, suggesting that factors other than the repeated activation these two stress-responsive systems may contribute to the increase in Pde10a seen in rats with a history of alcohol dependence or repeated footshock stress.

19 Effects of Amphetamine on Impaired Memory

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Disruption of cholinergic transmission in the brain impairs memory in humans and animals. Scopolamine impairs memory by blocking muscarinic receptor, a subtype of cholinergic receptors. Psychostimulants, such as amphetamine, are known to enhance arousal, increase attention, and also possess addictive potentials. Recent reports indicate a sharp increase in use of psychostimulants as cognitive enhancers among healthy individuals. This study examined the effects of amphetamine on simple memory, using an animal model. Our hypothesis was that amphetamine would reverse scopolamine-induced deficits. Wistar rats were shaped to press the lever for a food pellet. Rats were further trained on a simple task, fixed ratio 5 (FR5), which required 5 lever-presses for a food pellet. Upon reaching a criterion, rats received drugs in a counter balanced manner: amphetamine+saline, scopolamine+saline, amphetamine+scopolamine, and saline+saline. Drug effects were measured by the first response latency (time to press the first lever) and the runtime (time to complete 5 lever-presses). Consistent with our previous reports, scopolamine reliably impaired performance on FR5 by increasing the response latencies and runtime. Compared to saline, amphetamine reduced first response latency, but had no effects on runtime. Amphetamine and scopolamine combination failed to reverse scopolamine-induced impairment on the first response latency. Amphetamine and scopolamine combination substantially increased the runtime, compared to other treatment conditions. Our findings suggest that amphetamine may shorten initial response latency, but fails to reverse scopolamine-induced memory deficits. Given a risk of drug abuse potential, using psychostimulants as cognitive enhancers requires caution. A further study is warranted.

28 Mild Water Restriction of Female Rats Does Not Impact Estrous Cycle Stability

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Many behavioral studies in rodent models impose environmental manipulations, like food or water restriction, to motivate the animal to respond. The level to which these manipulations are effective or the consequences they have on physiology and behavior may differ based on sex. For example, water restriction is necessary to motivate rats to respond in operant assessments of taste detection. Since sex differences related to hormone levels change across the estrous cycle have been observed in taste detection (e.g., Matia et al 2016), it is imperative to determine if water restriction itself influences the estrous cycle. The present study examined if mild water restriction akin to that used in operant taste detection studies would interrupt or destabilize the normal estrous cycling of female rats. We used 24 Sprague-Dawley female rats from 12 litters across 2 study phases in an A-B-A between-subjects design. Sister-pairs of rats were divided into two experimental groups, one of which was never water restricted and one of which was restricted to 10 ml of water per day, Monday through Friday, during the B phase of the experiment; all animals received ad libitum access to water during the two 3-week A phases and over the weekends during the 3-week B phase. The estrous cycles of all the rats were tracked every weekday during the 9 weeks, and the stages were labeled by 2 observers blind to the experimental group via microscopic analysis of stained cells collected via vaginal swab. Five-day cycle periods were scored in a binary fashion as either stable or not, where an unstable cycle period was defined as 3 or more consecutive days of a single stage. Differences in the percent of rats presenting with unstable cycles overall and for each 3-week phase was assessed between experimental groups (water-replete vs. water-restricted) using independent samples t-tests. No significant differences between groups in percent instability were found over the course of the study both during and not during the water-restriction phase; however, nearly all of the rats presented with at least 1 week of unstable cycles across the 9 weeks. This suggests that water restriction has no direct effect on estrous stability in rats, and that mild water restriction can be used as motivation in behavioral tests in both sexes of rats without fear of its impact on the estrous cycle. However, it may be advised that estrous cycle stability of individual female rats should be determined prior to testing, so that the impact of estrous cycle instability can be minimized in behavioral studies.

27 Assessing the Efficacy of Cheek Fistula as Opposed to Intraoral Catheters for Infusing Oral Sucrose to Induce cFos Expression in the Rostral Nucleus of the Solitary Tract in Rats

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Oral sucrose exposure induces the expression of cFos (an early active gene) in the rostral nucleus of the solitary tract (rNST), which is the first central relay for taste information (Harrer & Travers, 1996). cFos expression (in the form of Fos protein) can be used as an index of neural activity, so changes in rNST Fos levels could provide insight into the mechanism underlying changes in taste-guided behavior. Oral exposure to induce rNST Fos typically requires surgically implanted intraoral (IO) catheters. We experienced complications from IO catheters (e.g., persistent infections, headcaps dislodging), and the catheters must be cleaned daily to maintain patency. We hypothesized that we could achieve 1) more surgical success and 2) cFos expression similar to Harrer & Travers (1996) by infusing sucrose through cheek fistulae, which require a less invasive surgery and are more easily maintained (Hintiryan et al, 2006). We therefor compared recovery times and extent of full recovery leading to testing of rats given either bilateral IO catheters or cheek fistulae (the former group also equipped with a 4th ventricular cannula for other purposes). Rats in both surgical groups (IO catheters n= 48; cheek fistulae n=20) were anesthetized with ketamine (100 mg/kg ip) and xylazine (10 mg/kg ip) and lengths of heat-flared polyethelene-50 tubing set with a Teflon washer was, using a 19 G needle, bilaterally inserted into the oral mucosa just anteriolateral to the 2nd molar. Intraoral catheters were tunneled subcutaneously, exited adjacent to the scalp, and affixed to the skull with bone screws and dental cement, whereas cheek fistulae exited through the cheek and were held in place with another Teflon washer. All animals received analgesic (carprofen 5 mg/kg sc) and antibiotic (gentamicin 8 mg/kg sc) on the day of surgery and for each of 3 days following, as well as access to softened food until body weight returned to pre-surgical levels and/or stabilized. Rats from 3 study phases for both surgeries are included in analysis. Similar to Hintaryan et al (2006), we found, on average, that rats given cheek fistulae recovered in 15 days compared to 26 days needed by rats given IO catheters ($p<0.001$), 65% of those with cheek fistulae reached their full presurgical body weight compared to 37% of those with IO catheters ($p=0.057$), and 85% of rats with cheek fistulae were able to be tested as opposed to 79% of those with IO catheters ($p>0.05$). After recovery from surgery, all of the rats were acclimated to oral infusions of water and tested with either water or 1.0 M sucrose (7.2 ml across 30 min). The rats were then anesthetized with sodium pentobarbital (100 mg/kg ip), perfused, and their brains sliced at 50 µm on a freezing microtome. We are currently analyzing brainstem slices immunohistochemically processed to visualize cFos expression to assess if sucrose exposure through cheek fistulae results in more rNST Fos than water exposure, similar to that seen when IO catheters are used (as per Harrer & Travers, 1996).

20 ERP Correlates of Ethical Decision Making: Effect of Who Gets Paid and When

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Insider threats represent a significant source of violations of digital information security, accounting for nearly 50% of security breaches. Current deterrence programs are not highly effective in changing attitudes or behaviors related to cybercrime. Therefore, the current study examined the effects of two independent variables (i.e., the recipient of the benefit and timing of the benefit gained from a violation of information security) on the neural correlates of ethical decision-making related to information security using event-related brain potentials (ERPs). In the study, participants responded as if they were "Josh", an IT specialist who was under financial strain and had extensive access to his employer's digital assets. For each trial, individuals first read a scenario that was 1-2 sentences in length and then responded to a prompt about whether or a not Josh should engage in an unethical or neutral activity. In half of the scenarios, Josh benefited from the action, and in the remaining scenarios, a relative or friend benefited; in the scenarios, the benefit was received after either 0-3 months or 12-24 months. The response choice data revealed that individuals were less likely to say yes to the ethical violation than the control trials, revealing a sensitivity to the ethical violation; and were more likely to say yes to long-term than short-term benefits when Josh was the recipient. A comparison of the ERPs for control trials and the ethical violation trials revealed differences in amplitude 200-2000 ms after onset of the prompt over the occipital, central, parietal, and frontal regions of the scalp reflecting a broadly distributed network that is generally sensitive to ethical decision making. The comparison of trials when Josh versus another benefitting revealed sustained frontal-polar ERP activity, consistent with the engagement of anterior frontal cortex in self-referential processing; and the comparison of the short and long delay trials revealed greater negativity for long than short delay trials over the frontal-polar region of the scalp, consistent with the delay discounting literature. The comparison of control trials with ethical violations replicates our previous studies demonstrating the robustness of the information security paradigm as a tool to examine the neural correlates of ethical decision-making in this domain. More interestingly, the effects of the two independent variables reveal that ethical decision-making related to information security is influenced by both the timing of the reward to be obtained, and whether or not one is acting for their own behalf or that of someone else.

21 Neural Correlates of Moral Foundations: Effects of Foundation, and Moral and Emotional Intensity

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Introduction/Overview: Moral Foundations Theory (MFT) describes five foundations (care, fairness, loyalty, authority, and sanctity) that underpin moral reasoning. Within this theory, the strength of a moral response is thought to be partially defined by the level of emotion that is elicited by the violation of a moral norm. The current study used event-related brain potentials (ERPs) to examine the neural correlates of three moral foundations and the relationship between moral judgement and emotion.
Method: In the study 53 undergraduates completed a task based upon the MFT using materials adapted from Clifford et al. (2015) while EEG was recorded from the scalp. For the task individuals first saw a context prompt for 3000 ms followed by a response prompt that was on the screen until a response was made. The response prompt described either a violation of a social norm or a violation of one of three moral foundations (i.e., authority, caring, loyalty). When the response prompt appeared individuals first judged how moral or immoral the action was, then reported their level of emotional reaction. Both of these were on a 4 point scale from low to high. EEG were recorded from 32 electrodes using a Brain Vision actiChamp system, and the data were analyzed using EEGLAB and ERPLAB.
Results: The ERP data comparing the three moral foundations to social norm violations revealed two types of effects. First, there was greater positivity for social norm violations than for the moral foundations over the central-parietal region of the scalp between 1000-2000 ms after stimulus onset that likely reflects a manifestation of the P3b component for the social norm violations. Second, there were differences in slow wave activity over the left and right lateral frontal regions that distinguished the three foundations from one another. This finding is consistent with the neuroimaging literature demonstrating that lateral and medial frontal regions are recruited during moral reasoning and decision making. Sorting the ERP data into intensity based upon moral violation or emotional response, revealed two interesting effects. First, the P3b was not sensitive to ratings of moral violation or emotional response. Second, higher rating for both measures were associated with stronger negativity over the right lateral frontal region; and greater positivity over the left parietal and occipital regions.
Discussion: The ERP data revealed differences in neural activity between violations of social norms and the three moral foundations. Violating a social norm was associated with attentional orienting related to the P3b, while violating moral foundations was associated with activity over the frontal region of the scalp. Consistent the MFT, there was overlap between the neural correlates of the level of emotion and moral response elicited in the task.

26 Impacts of Positive Affect Traits on Heart Rate During a Parasympathetic Task

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Individuals with low life satisfaction and high stress have been shown to have higher heart rates (Vrijkotte et al., 2000). Cheerfulness and a positive outlook on life have been shown to be positively associated with lower heart rate (Geisler et al., 2010). When participants were exposed to hope evocation the participants had significantly lower heart rates, even when exposed to various stressors (Chadwick et al., 2016). This current study explored the positive effects of satisfaction with life, curiosity, subjective happiness and trait hope on heart rate during a parasympathetic task. 125 college students from the University of Mount Union participated in this study. Five participants were unable to complete the study and were not included in the results. The majority of the participants were women (N = 90). The participants had a mean age of 19.06 (SD = 1.07). The first portion of the larger study was a cardiovascular recording, with three different epochs. The epochs were as follows: a vanilla baseline, an orthostatic position task and finally a supine positioning task. Each of the epochs were three minutes in length. This study focused on responses during the parasympathetic supine positioning task. After the completion of the cardiovascular portion, a set of questionnaires were given including: The Satisfaction with Life Scale, Curiosity and Exploration Inventory, Subjective Happiness Scale, and Trait Hope Scale. Linear regression analyses were utilized with satisfaction with life, curiosity, subjective happiness and trait hope as the independent variables and heart rate as the dependent.

Those students who had higher satisfaction with life had a lower heart rate during the heart rate recovery period ($R2 = .058$, $F (1, 119) = 7.28$, $p < .01$). Subjects who scored higher on the Curiosity and Exploration Inventory scale had a lower heart during the heart rate recovery period ($R2 = .054$, $F (1, 119) = 6.69$, $p < .02$). Increases on the Subjective Happiness scale ($R2 = .098$, $F (1, 119) = 12.88$, $p < .001$) and Trait Hope scales ($R2 = .041$, $F (1, 119) = 5.10$, $p < .05$) predicted decreased heart rates during the task. The results from this current study were similar to previous research in the field of positive affect. In particular, the findings in this study reflected the work of Geisler et al. (2010) on cheerfulness and Chadwick et al. (2016) relating to hope. In each case, increases in positive affect traits were associated with decreases in heart rate while in a parasympathetic task setting. Future research in this area could explore other positive traits that were not discussed in this study such as gratitude, confidence, truthfulness, or enjoyment. It can further be examined how positive affect traits may buffer the negative impacts that traits such as anxiety and depression have on heart rate and heart rate variability.

25 Optimization of Differentiation Protocol for SH-SY5Y Cells for Human Neuronal Cultures

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SH-SY5Y cells are human brain-tumor derived but behave like “neuronal progenitor cells,” which grow and divide indefinitely under normal conditions, but have the potential to differentiate into neuron-like post-mitotic cells. However, several different protocols on differentiation have been published, each with varying levels of success. Our goal was to systematically test the various components from published protocols and derive a treatment protocol that resulted in robust differentiation as efficiently as possible. In previous studies, retinoic acid (RA) has been shown to trigger the inception of “neurogenesis” in SH-SY5Y cells, while other supplements are then later added to maintain neuronal growth, including potassium chloride, vitamin B-12, brain-derived neurotrophic factor (BDNF), Ara-C, db-cAMP, and the proprietary supplement, B-27. Our results show that a 3 -day treatment with RA and BDNF followed by a 7-day treatment with potassium chloride, B-27, and db-cAMP were the parameters that yielded the most neuron-like cells, as observed through light microscopy. Current experiments are further determining if fewer days of treatment in RA and BDNF are still as effective, and what is the longevitiy of differentiated cells in culture. Future directions include confirming the expression of neuronal genes via quantitative PCR (qPCR) and immuno-fluorescence microscopy. With an optimized differentiation protocol, we will be able to use SH-SY5Y cells to study human neuronal development and mechanisms of neurodegenerative disorders such as Alzheimer disease in a reproducible in vitro system.

22 Effects of N-acetylcysteine (NAC) on EAAT3 Transporter Protein Concentrations in OCD Induced Mice

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Obsessive-Compulsive Disorder is a mental health disorder that affects more than 3 million people in the United States (Rosenberg, 2018). It is characterized by recurring thoughts and behaviors that are often distressing and beyond the control of the affected individuals (Rosenberg, 2018). Recent studies show a correlation between OCD affected patients and high levels of glutamate, an excitatory neurotransmitter, in the brain (Naaijen et al., 2018). The high levels of glutamate could be due to a change in the amount or activity of excitatory amino acid transporters (EAATs), which regulate the concentration of glutamate from the synapse (Schirmbeck et al., 2012). Studies also show that N-acetylcysteine (NAC) reduces the amount of glutamate that is released from the synapse (Grant et al., 2009). The goal of this experiment was to understand if NAC treatment would change the abundance of EAAT3 transporters in NAC-treated mice. The Western Blot technique was used to investigate the amount of EAAT3 protein in WT and NAC-treated mouse brains. Results show that there was no difference in the EAAT3 concentration in these mice; thus, NAC does not directly affect the EAAT3 transporter protein abundance.

23 Lowering Luteinizing Hormone (LH) Increased Neuron Number and Microglial Presence in a Rat Alzheimer's Disease Model

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Alzheimer's disease (AD) is a neurodegenerative disorder affecting 5.7 million Americans. Progressive memory loss and elevated β-amyloid levels characterize AD. One of the main structures damaged during AD is the hippocampus, a brain area important for memory. Although age is the primary risk factor for AD, women have a 2.5-fold higher incidence of AD than men, even after controlling for age. It has been hypothesized that menopause, marked by a loss of estrogen, may be involved in the sex differences in AD. Previous evidence suggested that estrogen reduction can increase risk of AD. When estrogen is decreased, luteinizing hormone (LH) is increased. There is evidence that low levels of LH are associated with better memory outcomes. In this study, ovariectomized rats were treated with Antide, a luteinizing hormone releasing hormone (LHRH) antagonist, to investigate the effects of lowered LH in an Alzheimer's Disease model. Disease damage was achieved via neurotoxin infusion and Antide was administered both before and after neurotoxin infusion to explore the preventative and restorative effects of Antide. Behavioral testing and immunohistochemical analysis were utilized to investigate both behavioral and pathological results of the Antide treatment. Our results demonstrated that both preventative and restorative administration of Antide improved spatial memory compared to the AD rats with no treatment. Representative hippocampal tissue from each experimental group was stained with NeuN, Iba-1, and GFAP and analyzed with Image J (NeuN) and Image Pro Premier (Iba-1 and GFAP). Our data showed that while both preventative (Antide Early) and restorative (Antide Late) administration of Antide had higher median levels of NeuN+ cells than the AD group, this data was not statistically significant when compared to the control rats. Antide Early and Antide Late significantly increased microglia in the damaged hippocampus, but did not have the same effect on the astrocytes. This is of interest as previous data has indicated that increased microglia is associated with worsened spatial memory, yet our results presented an opposing relationship. Further experimentation could reveal the microglial products, indicating whether the neuroimmune response is inflammatory, damaging, or protective. Taken together, our data indicated Antide treatment was neuroprotective, and had microglia-directed effects. Discovering more about LH and its actions on microglia could help reveal the phenotype of microglia in post-menopausal women with AD. Overall, lowering LH is a possible neuroprotective therapeutic that works through diverse mechanisms and could be effective before or after AD pathology.

24 "Got Sleep?": An Investigation of Trio, a Guanine Nucleotide Exchange Factor (GEF), as a Possible Contributor to Sleep Regulation in Drosophila melanogaster

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Sleep is highly conserved and regulated in animal species studied so far. However, with the rise of new technologies in modern society and increasing stress from work, chronic sleep loss has become a health epidemic, posing links with cancer and heart disease. Prior studies that focus on decrypting the innerworkings of sleep regulation face the challenge of lack of information on possible molecular-level participants, but the use of model organisms like Drosophila melanogaster, the common fruit fly, in these studies has uncovered certain proteins that may have important roles. Previous data from our lab has demonstrated a large number of proteins that may be altered from sleep deprivation. This study aims to expand the current list of these sleep-regulating proteins by examining Trio, a Rho-family GTPase and several proteasome subunits that have previously been found to change in concentration following sleep deprivation. Through western blot analysis, we found significant changes in the concentrations of Trio and proteasome subunits following sleep deprivation suggesting that these proteins may regulate sleep. Remarkably, we have also demonstrated that Trio mutants have reduced total sleep, bout (sleeping episode) duration, and total night sleep. These preliminary findings suggest that Trio might be regulating sleep in some capacity. Further work must be done in order to solidify these findings in these Trio mutants, determine if there are similar effects occur in proteasome and Rho-family mutants, and if there are changes in the localization of these proteins in the fly brain following sleep deprivation.