Keynote Speakers:

Neurobiology of Hypertension: The Silent killer

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Hypertension affects over 70 million people in the United States and is disturbingly higher in African Americans than in white Americans. About 44% of AA in the age group of 45 - 54 years compared to 29% of whites. Evidence suggests that a genetic predisposition to hypertension may underlie this health disparity. Blood pressure is controlled almost exclusively by the sympathetic nervous (SNS) system and the renin angiotensin system (RAS). Increased central nervous system activity initiates a cascade of signaling events that ultimately leads to increased activity in vasomotor areas in the dorsal medulla. This excessive activity is transmitted through sympathetic ganglia in the peripheral nervous system and culminates in the activation of specific receptors on the surface of blood vessels. This activation initiates arterial vasoconstriction leading to increased peripheral resistance, heart rate, and blood pressure. Current therapeutic agents are effective in normalizing blood pressure in hypertensive individuals; however these treatments do not address the underlying SNS over activity. Our studies investigate the link between the SNS and high blood pressure in two models of hypertension namely the (mRen2)27 transgenic (inherited form) and the Ouabain-induced model (acquired form) of hypertension. We measure the neuronal behavior of autonomic ganglia isolated from hypertensive rodents. Our initial results suggest that increased ganglionic activity is associated with elevated blood pressure in both (mRen2)27 and Ouabain induced models of hypertension. Application of Ang II or ouabain increases ganglionic activity in these animals and represents a potential target for therapeutic intervention. With this new data we are closer to understanding the neural mechanism underlying the initiation and/or maintenance of hypertension. Support provided by National Center for Minority Health and Health Disparities – NIH P20MD000232 and P20MD002303

Behavioral neuroscience: Taste Psychophysics from the Laboratory to the Table

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The development of new psychophysical methods that permit accurate comparisons of taste intensities across subjects led to the discovery of supertasters. Supertasters not only experience the most intense taste sensations, they also experience the most intense oral burn (chilis) and oral touch (fat) sensations as well. This turned out to be caused largely by differences in the anatomy of the tongue. Taste buds are housed in structures called fungiform papillae. These papillae are innervated by nerve fibers that convey oral burn and oral touch information as well as taste. Supertasters have the largest number of these papillae and so have the largest number of nerve fibers mediating taste, burn and touch. Thus supertasters live in a neon food world compared to the pastel food world of those with the fewest fungiform papillae. This sensory variation leads to variation in food preferences and thus affects diet and health. For example, supertasters are at increased risk for colon cancer (they avoid many bitter vegetables) but are at decreased risk for head and neck cancer (supertasters find smoking and drinking relatively unpleasant). Most recently we found that individuals who had serious ear infections as children were more likely to be overweight as adults. One of the taste nerves passes through the middle ear on its way to the brain; ear infections can damage that nerve. This damage releases central inhibition on oral touch (i.e., perception of fat). High fat foods become more palatable leading to weight gain.

Neuroscience Drug Development-Past, Present and Future

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Initial drug discovery efforts for central nervous system (CNS) therapeutics were more serendipitous than intentional. Once the first psychiatry drugs were marketed, a second wave of 'copy-cat' drugs was brought forward. Today, drug discovery in CNS is much more sophisticated and is highly dependent on broad interdisciplinary interactions. However, with the potential for great benefit comes the potential for unwanted side effects that must be mitigated. In addition, the current market environment presents new challenges to the development of CNS drugs: as major pharmaceutical companies face generic competition for some of their blockbuster drugs, they are sharply reducing their drug discovery efforts in neuroscience. There remains a great unmet medical need and this is reflected in the recent NIH initiative to translate basic discoveries at the bench into therapeutics.

Student Travel Award Winners

Effects of Oral 5-HTP Administration on a Computerized Decision-making Task

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Serotonin is a neurotransmitter that is involved in mood, sleep regulation, and cognition; alterations in serotonin activity are associated with several clinical conditions, including major depression. However, little research has been conducted on 5-hydroxytryptophan (5-HTP-the direct metabolic precursor to serotonin), despite its potential value as a therapeutic compound. 5-HTP is a plant product sold over the counter as an herbal medicine-it is not regulated as a drug by the FDA. There are no compelling financial reasons to conduct extensive clinical studies on this compound. As a result, current knowledge is limited in regards to the degree to which oral 5-HTP administration alters serotonin synthesis, vesicular packaging, and release in the brain. The present study examined if oral administration of 5-HTP to young adult volunteers impacted performance on the Iowa Gambling Task (IGT), a measure known to be sensitive to changes in serotonin levels in the orbitofrontal cortex. Participants received either two 50 mg 5-HTP capsules or two placebo tablets, and were tested on the IGT following an absorption period. Results from this study demonstrated that oral 5-HTP impaired performance on the IGT relative to the placebo group, which indicated that 5-HTP may be psychoactive at moderate doses. The deficit seen by the 5-HTP group appears specific to the ambiguous component of the IGT task. Decisions made under ambiguity may be differentially sensitive to increased 5-HT release or associated reductions in frontocortical dopamine activity. Excessive 5-HTP may hijack aromatic acid decarboxylase in dopaminergic neurons resulting in ectopic 5-HT release.

Investigating the Role of the Peripheral α 2A Receptors in Neuropathic Pain Using Recombinant Herpes Simplex Virus Type 1 to Over-express the Adrenergic α 2A Receptor

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Agonists for the α2A–adrenoceptor reduce pain in both animals and humans. Current α2A analgesics produce sedation and cardiovascular depression after systematic or intrathecal injection. The compound ST91, a peripherally active α2A agonist, produces analgesia without hypotension and sedation. The present study used recombinant herpes simplex virus type 1 (HSV-1) containing cDNA sequences for the adrenergic α2A receptor (HSV- α2A) and, as a control, the E. coli lac Z gene marker (HSV-lacZ) to investigate the role of peripheral α2A receptors in neuropathic pain-associated behaviors and α2A –medicated analgesia. Mice were infected on day 7 post-L5 spinal nerve transection, or in the absence of nerve injury, with HSV- α2A or HSV-lacZ. HSV-α2A increased expression of α2A in the DRG and spinal cord, but not the skin at day 15 post-infection as compared to HSV-lacZ control. In uninjured mice, HSV-α2A increased ST-91 analgesia, capsaicin (0.15%) induced de-sensitization and capsaicin (0.015%) induced thermal hyperalgesia as compared to HSV-lacZ. In nerve injured mice, HSV-α2A had no effect on thermal paw withdrawal latency or basal paw withdrawal responses on days 14 and 15 post-infection as compared to HSV-lacZ. In nerve injured mice HSV-α2A had no effect on ST-91 analgesia compared to HSV-lacZ. In summary, HSV-1 mediated expression of α2A in primary afferent neurons, increased capsaicin-induced nociception and ST91 analgesia in uninjured mice but was unable to modify nerve injury-induced allodynia and hyperalgesia. This work was funded by a United States National Institutes of Neurological Disease and Stroke grant (NS26363).

AKAP-mediated Interactions Contribute to the Cell Biology of Cocaine Reinstatement

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Elucidation of the cellular dynamics responsible for drug-seeking behaviors is critical to understanding and treating addiction. Addiction is believed to be associated with cellular changes in structures engaged in the mesolimbic dopaminergic circuitry, including the nucleus accumbens. In the nucleus accumbens, a molecule bound to the synaptic cell membrane, the A-kinase-anchoring-protein (AKAP150), may be related to addictive behavior by coordinating temporal and spatial aspects of cell signaling which underlie drug-seeking behavior. Under normal conditions AKAP150 binds to a number of signaling molecules, particularly the regulatory subunit of protein kinase-A (PKA). This interaction is disrupted by the introduction of a synthetic inhibitory peptide, Ht31. In order to determine if the PKA:AKAP150 interaction contributes to addiction-related behaviors, we microinjected cell permeable Ht31 into the nucleus accumbens and observed the behavioral effects during cocaine-primed reinstatement in a self-administration paradigm. Animal receiving Ht31 microinjections showed decreased drug-seeking behavior compared to those receiving a control peptide; however, overall locomotor behavior in response to cocaine was unchanged between the two groups.. Further, levels of the catalytic subunits of PKA were decreased in a postsynaptic density subfraction from naïve animals following microinjection of Ht31 compared to control peptide, indicating that the Ht31 peptide leads to removal of PKA from this subcellular region. We propose a model in which activation of synaptic PKA within the nucleus accumbens leads to phosphorylation of glutamate receptors and other targets critical for drug-seeking, and that synaptic localization of PKA by AKAP150 is an important component of this signaling pathway. This work was supported by NIH grant DA003906 to P.W.K.

Enhanced Preference for Ethanol Odor and Increased Voluntary Intake Following Exposure to Ethanol During the Early Postnatal Period

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A focus on early development is critical for understanding the etiology of alcoholism. The observation that alcoholism runs in families has strengthened the idea of a genetic basis for the disease. It is also recognized however, that growing up in a household where alcohol cues are omnipresent may provide critical experiences that could increase that risk. Several experiential factors have been identified that contribute to the onset of alcohol drinking behavior by adolescents. Here we further examine that phenomenon using the demonstrator-observer paradigm. Experimental subjects (demonstrators) were exposed to intoxicated siblings (observers) in their home cage on postnatal days 8-12. Controls were exposed to siblings that were administered water. Animals were tested for voluntary ethanol intake (Exp 1) or ethanol odor preference (Exp 2) on postnatal days 30-31. Results revealed that infant experience with ethanol cues in the home environment result in increased ethanol intake and ethanol odor preference by adolescent-age subjects. These responses to alcohol were not only evident in demonstrator subjects, but also in the observer animals. Thus, both direct (intoxication) and indirect (via siblings) exposure to ethanol cues can increase animals' preferences for this drug. These experiments once again demonstrate the influence of early social experiences on later behavioral responses toward ethanol, and suggest that this type of ethanol exposure can have long-lasting consequences. Early learning about alcohol in the context of the home environment may result in increased risk for the later development of alcohol abuse and dependence. [supported by AA015343]

Neonatal Exposure to Alcohol Alters Spinal Cord Innervation

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Alcohol exposure in utero causes physiological and cognitive defects which are termed Fetal Alcohol Spectrum Disorders (FASD) and occurs in 1-10 out of 1000 births. The clinical literature has described somatosensory processing disorders characterized by under-responsiveness to sensory stimulation with increased seeking of sensory stimulation in children with FASD. The purpose of this study is to uncover the neurophysiological changes which underlie this somatosensory processing disorder. A FASD rodent model results in decreased sensitivity to non-noxious mechanical stimuli (Aα- fiber mediated) and increased sensitivity to noxious thermal stimuli (C- fiber mediated). The innervation of the dorsal horn of the spinal cord occurs by activity dependent competition between A- and C- fibers. Examination of A- fiber and C- fiber innervations of the dorsal horn of the spinal cord were assessed by immunohistochemistry. In the spinal cord, a decrease in the area and density of immunoreactivity of Neurofilament 200 (a marker of Aα- fiber terminals) illustrates one part of the dynamics of this competitive relationship. In contrast, an increase in area and density of calcitonin gene related peptide (a marker for C- fiber terminals) immunoreactivity in Lamina II of the spinal cord corresponds with the activity dependent competition for innervation. In conclusion, this study demonstrated that fetal alcohol exposure results in changes in spinal innervation. This understanding gives a physiological context to place the clinical evidence of somatosensory processing defects in the FASD population, as cortical wiring is also activity dependent and consequentially somatosensory messages from the spinal cord can lead to abnormal cortical circuits.

Poster Number 1

Poster Presentations

Dissociating the Behavioral Economic Concepts of Cocaine Consumption and Price Paid Using Self-administration and Pharmacology

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Price and consumption are key concepts in behavioral economics. In the context of cocaine self-administration, consumption refers to rate of drug intake on a simple schedule of reinforcement. Rate of intake under a fixed ratio 1(FR1) schedule, for example, reflects the maintenance of cocaine concentrations in blood/brain within a preferred range. By contrast, the concept of price refers to the motivation of an animal to self-administer cocaine; this is usually measured with procedures such as a progressive ratio (PR) schedule. It remains unclear whether price and consumption are necessarily related during self-administration or whether each can be affected independently of the other. Depending on the schedule used, it is often difficult to determine whether, for example, a particular drug pretreatment is affecting cocaine self-administration through an effect on consumption or price or perhaps both. To investigate the relationship between these variable we developed a technique that provides an independent assessment of both price and consumption within the same experimental session. The technique is an adaptation of a threshold procedure. During the threshold procedure rats are offered a descending series of eleven unit doses (422 – 1.3μg) during consecutive 10 min intervals. An estimate of consumption can be determined from response rates associated with high unit doses; an estimate of price can be determined from response rates surrounding the threshold dose (i.e. the lowest dose at which cocaine consumption is maintained). The effect of pretreatment with compounds from a variety of drug classes on price and consumption was investigated. Haloperidol (a dopamine receptor antagonist) dose dependently increased cocaine consumption and decreased the price paid; conversely, acutely administered amphetamine (a monoamine releaser) dose-dependently decreased cocaine consumption but increased the price paid. Other drugs, however, were found to affect price and consumption independently. For example, baclofen (a GABAB agonist) dose dependently decreased price paid while having no affect on consumption. The present results show that price and consumption are dissociable phenomena. While in some instances price and consumption might both be inversely affected by some drug treatments this is not necessarily the case. These data suggest that the neurobiological substrates of price and consumption might involve separate mechanisms.

Poster Number 2

Older Adult Memory for Crime Information:A Behavioral and Electrophysiological Study

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This study investigated age-related differences in memory for crime-related evidence. Behavioral and event-related potential (ERP) data were collected to examine how older and young adults differ in retrieval of specific types of crime-related details and how memory for such details is influenced by activation of schemas. Fifty-one older and 50 young adults read a paragraph about a criminal suspect that described either a "bad" or "good" childhood. Then, all participants read a separate burglary story that contained equal numbers of incriminating, exonerating, and neutral (with regard to guilt) facts. Finally, participants viewed incriminating, exonerating, and neutral phrases from the burglary story on a computer. Half those phrases were exactly the same as in the story (targets) while half had been modified (lures). Participants were instructed to press "true" if they recognized exact phrases from the story and to press "false" if the phrases were not exactly the same as in the story. Older adults falsely recognized more details than young adults, and this difference was greater for incriminating than for exonerating evidence. Additionally, older adults with lower Mini-Mental scores (MMSE) showed a greater difference in accuracy between incriminating and exonerating details. These data suggest that older adults are more likely to accept incriminating evidence when they are uncertain, and that cognitive decline may focus greater attention on processing incriminating information than exonerating information, perhaps due to dependence on schemas about criminal suspects. Analysis of electrophysiological data also suggested age-related differences in processing of incriminating and exonerating details of a crime.

Poster Number 3

Development of a Novel Delay Discounting Task: Evaluation of Nicotinic Receptor Blockade.

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Delay discounting is a commonly used measure to assess impulsive decision-making. Many of the tasks used involve providing access to a small, immediate reward and a larger, delayed reward, with a choice of the immediate reward thought to reflect greater impulsivity. However, these tasks do not allow assessment of effects of immediate versus delayed access to multiple rewards amounts. Moreover, a direct comparison of the effects of delay before or after reward access is confounded by reward amount. In order to address these issues, additional choices must be made available. In the present experiment, rats were trained in an 8-arm automated radial maze. Three of the arms offered immediate access to low (0.01 ml tap water), medium (0.06 ml), or high (0.10 ml) rewards with a delay imposed after reward access. Three different arms offered delayed access to low, medium or high rewards. In the standard task, the delays were matched before or after reward access for each reward amount (10 sec for low reward, 30 sec for medium reward, 60 sec for high reward). With sufficient training, animals exhibited a significant preference for immediately accessible arms. Additional task manipulations increased the probability of entering an immediately accessible arm and efforts to attenuate this preference, by increasing the delay on immediately accessible arms, had only a minor effect on performance. Finally, administration of the nicotinic receptor antagonist, mecamylamine, did not significantly affect performance. The present experiment represents an initial effort to characterize performance in a novel delay discounting task that allows a direct comparison between immediate and delayed access for matched rewards. Supported by AG030646.

Poster Number 4

Endothelin-1 Induced Nociception and Neuronal Activation Across the Estrous cycle

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Many studies have shown gender differences in response to pain, however the role that the estrous cycle has on nociceptive behavior is not clearly understood. In this study, endothelin 1 (ET-1) was used to study the variations in nociception among females at different stages of the estrous cycle. ET-1 was administered in the left hind paw and paw flinching was counted in 5 minute intervals for 75 minutes. No significant difference in nociception across estrous cycle was found with regards to the total number of flinches and the maximum behavior. In contrast, a temporal variation in nociception was found with diestrous rats showing two peaks in nociceptive behavior as compared to a single peak in proestrous and estrous rats. Neuronal activity was analyzed by looking at the number of c-fos positive neurons found within the L3-L5 spinal cord at 2 hours after ET-1. C-fos analysis did not show any significant difference in ET-1 induced neuronal activation across estrous. Based on the results of this study we concluded that female rats did not show a significant difference in nociception at various stages of the estrous cycle with regards to the total number of flinches and maximum behavior expressed by each group although the temporal nature of nociception varies across estrous cycle. This project was funded by a United States National Institutes of Drugs Abuse Grant DA023593.

Poster Number 5

Caffeine Increases Locomotor Activity in Forager Honey Bees

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Caffeine increases locomotor activity in many animals, including humans and fruit flies. Caffeine is hypothesized to act to promote activity by its direct interactions with adenosine receptors and indirect interactions with dopamine receptors. The object of these studies was to use caffeine as a tool to manipulate dopamine function in honey bees, which are widely used as a model for the study of learning and memory. Earlier studies on young (1 day old) worker honey bees (Apis mellifera) found that ingestion of 2.5 mg/ml increased walking in a laboratory assay. It was hypothesized that a dopamine antagonist would inhibit the effects of caffeine on locomotion. Because of concern that drug treatments would be difficult to interpret in young bees that had just completed metamorphosis, we sought to replicate the effects of caffeine on locomotion in older (foraging) honey bees. We found that the older bees refused to drink the caffeine solution that younger bees freely consumed, a novel finding that implies that older bees are more sensitive than younger bees to the bitter taste of caffeine. We therefore adopted injection as our method of caffeine treatment for forager honey bees. At both 60 and 75 minutes after injection, bees treated with 1 g/ L caffeine (N = 9) showed significantly increased locomotor activity compared with vehicle-injected control bees (N = 10; Mann-Whitney U-test, $p < 0.01$ at both 60 and 75 minutes post-injection). The effect was reversible, as these groups did not differ at later times post-injection. With this finding, the effects of dopamine antagonists on this simple behavior can now be tested.

Poster Number 6

Chronic Ethanol Up-Regulates Synaptic Expression of the Nuclear Translational Regulatory Protein AIDA-1 in Hippocampal Neurons

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Recent studies have identified synaptic proteins that undergo synapse-to-nucleus translocation in response to neuronal activity that modulate protein synthesis. One such translational regulatory protein of the post-synaptic density (PSD) is AIDA-1, which binds to PSD-95 via its C-terminus. Activation of synaptic NMDA receptors induces the cleavage of AIDA-1 and the N-terminus is then shuttled to nuclear Cajal bodies where it plays a possible role in ribosomal RNA splicing. Moreover, prolonged synaptic activity produces an AIDA-1-dependent increase in global protein synthesis. It has been previously identified that, chronic ethanol exposure led to increases in NR2B-containing NMDA receptors, and clustering of PSD-95 at synaptic sites. Consequently, we tested the hypothesis that there would be a corresponding increase in synaptic AIDA-1 following ethanol treatment. As expected, AIDA-1 is highly enriched in dendritic spines co-stained with PSD-95, and acute NMDA treatment increased AIDA-1 co-localization with coilin, a marker of Cajal bodies. Surprisingly, declustering PSD-95 by inhibition of palmitoylation using 10 micro-molar 2-bromo-palmitate had no effect on AIDA-1 clustering. Chronic treatment (4-day) of cultures with 50mM ethanol or the NMDA receptor antagonist APV (100 micro-molar) enhanced the cluster size and density of AIDA-1. These data demonstrate that AIDA-1 expression is sensitive to chronic ethanol exposure and to prolonged inhibition of NMDA receptor activity. The ethanol-induced enhanced localization of AIDA-1 and NMDA receptors at the PSD could promote increased nuclear translocation of the N-terminal fragment to Cajal bodies thus influencing global protein synthesis, mRNA processing and expression of splice variants.

Poster Number 7

The role of BDNF/TrkB Signaling in Acute AMPH-induced Locomotor Activity and Opioid Peptide Gene Expression in the Rat Dorsal Striatum.

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Addiction is a chronic disorder characterized by compulsive drug seeking and abuse. Psychostimulants, such as amphetamines, are a category of abused drugs that have a propensity for abuse potential due to their ability to induce feelings of euphoria. Although amphetamines are the first line of treatment for disorders such as ADHD, an estimated 13 million Americans use amphetamines illegally. Acute amphetamine exposure increases release of dopamine, glutamate, and brain derived neurotrophic factor (BDNF) in the striatum of rats. These three neurochemicals bind to receptors expressed by neurons in the striatum and activate various signaling cascades. These cascades lead to the phosphorylation of nuclear transcription factors such as cAMP response element binding protein. The phophorylation of nuclear transcription factors are thought to contribute to striatal neuropeptide gene expression and behavioral effects induced by acute amphetamine. BDNF is a neurotrophin involved in neuronal cell growth and synaptic plasticity that binds to TrkB receptors. BDNF deficient mice are known to express less opioid peptide mRNA in the mouse striatum and exhibit more prolonged hyperlocomotor activity following acute amphetamine exposure. Therefore, the current study evaluated the role of BDNF/TrkB signaling in amphetamine-induced locomotor activity and opioid peptide mRNA expression in the rat dorsal striatum. Intra-striatal infusion of the Trk antagonist K252a following a systemic injection of amphetamine decreased total distance traveled but increased vertical activity. Assessment of opioid peptide gene expression is in progress. These data suggest that TrkB/BDNF signaling plays an important role in amphetamine-induced locomotor activity. Supported by RO1 DA03982.

Poster Number 8

Age-related changes in Locus Coeruleus T1-TSE signal

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The locus coeruleus (LC) is a brainstem structure consisting of noradrenergic neurons that regulate attentional states through widespread brainstem, sub-cortical, and cortical connections. Atypical LC function is hypothesized to contribute to a variety of behavioral and/or cognitive disorders, including attention-deficit hyperactivity disorder and post-traumatic stress disorder, as well as normal aging. While age-related declines in number of LC neurons have been observed in post-mortem studies, there has been limited research conducted on this important neuroregulatory system because of the small size of the LC, its close proximity to adjacent cortical structures, and the relatively poor resolution of conventional imaging techniques. More recently, high-resolution T1-weighted turbo spin echo (T1-TSE) magnetic resonance (MR) scanning methods (more commonly known as fMRI) have been used to image the LC. The focus of this investigation was to examine the extent to which there was a specific age-related change in LC signal, or whether this change could be attributed to other factors in the T1-TSE scans. T1-TSE MRI brain scans were used to produce a probabilistic map that defined the space of the LC across 44 adult participants ranging in age from 19 to 79 years. The individual raw brainstem images, originally acquired in native space, were normalized into Montreal Neurological Institute (MNI) coordinate space in order to create an accurate study-specific template for future comparison. A non-linear change in LC signal with age was observed and found to be dependent on the pontine tegmentum control region and LC signal decreasing with age, as well as increased noise in the scans of older adults. These two factors explained nearly all the variance in LC signal.

Poster Number 9

Effect of Chronic Alcohol Self-Administration on Nucleus Accumbens Protein Expression

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Alcoholism is a serious and often fatal disease that affects approximately 15 million Americans and 76.3 million people worldwide. The mechanisms by which alcohol affects the brain are not completely understood and this research may serve to elucidate dependence treatments. One area of the brain, the nucleus accumbens (NAcc), has been linked with addiction and reward. The NAcc has been shown to play a role in the abuse of numerous drugs, including alcohol. The medium spiny neurons within the NAcc contain glutamate and related proteins. The goal of this research is to compare the expression of GLT-1, GLAST, xCT and a few other (to be decided) glutamatergic proteins between the NAcc of experimental and control rats. The first aim of this research is to successfully implement sufficient alcohol self-administration in male Sprague-Dawley rats by means of the intermittent drinking paradigm. Rats were housed in separate Plexiglas cages, under reversed light cycle climate-controlled conditions, and given access to food and water ad libitum. The experimental group was given 20% EtOH for a duration of 24 hours, three days a week. Subjects may therefore self-administer the drug without the use of force or confounding variables. Control subjects received the same treatment without the presentation of ethanol. Once animals self-administered sufficient quantities of EtOH on a regular basis (about 6 weeks) they are euthanized without the use of analgesics to prevent any confounding protein alteration. The NAcc core was removed from all subjects and the glutamatergic proteins of interest are compared using Western Blotting, between the alcohol and control groups. At this stage, experimental animals have been successfully self-administering EtOH and their protein levels will soon be analyzed.

Poster Number 10

Synaptic Plasticity in Dysbindin Deficient Mice

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Schizophrenia is a severe brain disorder that affects approximately 1% of the US population. The disorder results in decreased cognition and memory function. Currently, there are no biological tests for schizophrenia and diagnosis is based on the patient's self-reported behavior and observations of friends and family. There have been strong links found between reduced expression of a particular gene, DTNBP1, and the clinical expression of schizophrenia. DTNBP1 encodes for the protein dysbindin. Behavioral studies in dysbindin deficient mice have found decreases in cognitive skills such as spatial working memory. Spatial working memory is dependent upon the hippocampus, a brain region implicated in schizophrenia. Dysbindin is expressed in neural tissue of the hippocampus and is involved in the control and release of glutamate. Dysbindin alleles are thought to regulate synaptic glutamate receptor (NMDA receptors) expression and function. NMDA-dependent synaptic plasticity is thought by many to be the synaptic mechanism for learning and memory. Our hypothesis was that genetic loss of dysbindin expression would decrease NMDA receptor-dependent synaptic plasticity in the hippocampus. Field recordings of the CA1 of the hippocampus, using a long term potentiation stimulation protocol, were conducted using C57 male wild-type and homozygous mutant littermate mice. The results showed a decrease in long term potentiation and NMDA receptor function. These results show a decrease in synaptic plasticity in dysbindin deficient mice and bring to attention an alleged mechanism by which dysbindin may contribute to cognitive deficits in schizophrenia.

Poster Number 11

Sex Differences in the Progression of Alzheimer's

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The percentage of Americans over 71 in the United States living with Alzheimer's disease is 16% of women compared to 11% of men. The factors contributing to this difference in prevalence may also effect the progression of the disease and offer insight into the mechanisms of Alzheimer's. We are conducting a five-year, longitudinal study which we anticipate will reveal that men and women with Alzheimer's differ predictably in their performance on tests of Alzheimer's progression, specifically those of memory (WMS-III Logical Memory I and II,) language (Boston Naming Test,) and global functioning (Trail Making Tests A and B) relative to their clinical dementia rating (CDR) and clinical diagnosis. 219 elderly South Carolinians (average age = 72.6 years) completed a baseline clinical evaluation and diagnosis consisting of a neuropsychological battery of 39 tests, and will return for evaluation annually for the next five years. For those with at least mild cognitive impairment (CDR=>0.5) evident causes of dementia other than Alzheimer's were excluded for during clinical diagnosis. Our initial analysis of 52 participants, who currently have complete data for the first two years, reveals that there is a significantly stronger correlation between WMS-III Logical Memory I (test of immediate auditory memory) and CDR for women versus that of men ($p>0.5$). The difference between men and women and CDR to WMS-III Logical Memory II (delayed auditory memory) is barely insignificant at $p>.1$ ($r=0.162$, critical value for $df=0.164$.) Further analyses on subsequent years and neuropsych tests will be conducted in order to further our hypotheses and provide a better understanding of how the disease presents between the sexes.

Poster Number 12

Aging Speech Systems: Task Demands But Not Perception

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In humans, older adults experience speech recognition problems, particularly in noisy listening conditions, even when they have relatively normal hearing. In attempt to understand age-related changes, we examined the extent to which there were age-related changes in the responsiveness of the auditory cortex with respect to areas sensitive to speech using functional magnetic resonance imaging. In comparison to a study using an aural response in the scanner to a speech recognition task, we used a button press method in which subjects would respond to words in one of four low pass frequency filter conditions. Age-related changes were not observed in the responsiveness of auditory cortex. However, with increasing word intelligibility younger adults exhibited an increased signal in the parietal and posterior cingulate cortex, an area found to be observed when no task demand is placed on the subject. Also, we demonstrated that older adults experienced increased frontal activation in the most intelligible word conditions, suggesting that task demands were greater for the older adults than the younger. These results demonstrate that an understanding of task difficulty on brain activation and performance in aging studies is critical to the interpretation of age-related effects. Funding from The College of Charleston URCA Program.

Poster Number 13

The Effect of Chronic Ethanol Treatment on Cannabinoid-Mediated Behaviors in Mice

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Research has shown cross-tolerance between ethanol (EtOH) and cannabinoids since the 1970's. In addition it has been shown that chronic treatment with EtOH can produce a down-regulation in the primary endocannabinoid receptor in the brain, CB1. Acute treatment with CB1 agonists produces a variety of behavioral alterations including disruptions in performance on cognitive tasks as well as a syndrome of four behaviors known as the mouse tetrad (hypothermia, analgesia, decreased locomotor activity, catalepsy). Currently, there are no published findings on the effects of chronic EtOH exposure on the mouse tetrad. This project is investigating if pre-treatment with repeated doses of EtOH is sufficient to produce an altered response in CB1-mediated behaviors. Male C57BL/6J mice (8 weeks old) were divided into four groups (EtOH-WIN, Sal-WIN, EtOH-Veh, Sal-Veh) based on treatment and the compound they received on the test day. The subjects received either EtOH or saline during the treatment period and either the CB1 agonist, WIN 55,212-2 (WIN), or vehicle (tocrisolve/saline) on the test day. Test compounds were administered via intraparietal injection, and animals were immediately placed in locomotor activity chambers for 25 minutes, followed by the three other mouse tetrad measurements. Three different doses of WIN were used to establish a dose-response curve and EtOH treatment showed a significant decrease in locomotor activity however, repeated EtOH injections failed to alter sensitivity to WIN in all measures of the tetrad. Ongoing work will examine the effects of repeated EtOH administration and EtOH dependence on both the mouse tetrad and cognitive tasks involving water-maze based memory.

Poster Number 14

The Role of Locus Coeruleus and Frontal Cortex in Response Inhibition

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Attention deficit/hyperactivity disorder (ADHD) is the most commonly diagnosed childhood psychiatric disorder, with nearly 9% of children qualifying for diagnosis. ADHD presents as a heterogeneous syndrome classified by three functionally-impairing, diagnostic criteria: inattention, hyperactivity, and impulsivity. Impulsivity also plays a critical role in the maintenance of drug addiction, and it is the focus of this study. Only recently have valid neurobiological explanations of the different criterion of ADHD been offered, including impulsivity. Impulsivity is a poorly-understood, complicated phenomenon and has several subtypes (e.g., response inhibition and delay aversion). Here, we investigated the role of locus coeruleus (LC) and orbitofrontal (OFC) and anterior cingulate cortices (ACC) in response inhibition. In preliminary studies, rats performed a Go/No-Go task to measure the ability to inhibit a prepotent response, a key component of response inhibition. Results of these early studies show: 1) rats do reliably perform the Go/No-Go task, and 2) the activity of neurons in the LC, OFC, and ACC are all modulated during performance of the task. Furthermore, LC activity seems to be modulated in both Go and No-Go conditions, suggesting a role for the LC in both response activation and inhibition. Present studies are underway to elucidate more details of the involvement of LC, OFC, and ACC in impulsivity (i.e., decision-making processes). These initial results, however, support a role of LC, OFC, and ACC in the modulation of both the generation and the inhibition of behaviors. Support contributed by NIH grant P50 MH62196.

Poster Number 15

Facet Joint Injury in the Neck Increases Expression of Metabotropic Glutamate Receptor-5 and Protein Kinase-C Epsilon in Afferent Neurons in a Rat Model of Whiplash Injury.

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Whiplash and other neck injuries are common injuries sustained by both adults and children following automobile collisions and recreational activities. Neck pain can result from damage to a variety of tissues in the cervical spine, among them the facet joint and its ligament. Despite increased information about the biomechanical mechanisms of tissue injury, the exact mechanisms by which pain is produced remain undefined. This project analyzed the cellular and molecular mechanisms associated with one common type of painful neck injury, facet joint distraction. We postulate that during joint injury there is activation of the primary nociceptive afferent neurons that results in mGluR5 mediated activation of PKC-epsilon and pain. A controlled cervical facet joint distraction injury was modeled by applying controlled stretching of the sixth and seventh cervical vertebrae in male Holtzman rats. Injury resulted in the development of sustained mechanical allodynia in the forepaws. Dorsal root ganglia at the level of injury were collected at day 7 for immunohistochemical analysis of mGluR5 and PKC epsilon in the cell bodies of afferents that innervate the injured joint. Injury resulted in a siginificant increase in the number of small diameter afferent neurons expressing mGluR5 and PKE epsilon. In addition, injury increased the amount of PKC epsilon in small and medium diameter afferent neurons. These results support the hypothesis that transient painful mechanical injuries to this joint are sufficient to induceactivation of the mGluR5 and PKC-epsilon channels. Future pharmacological studies will be able to use this information to inhibit the mGluR5 and PKC-epsilon channels in hopes of effectively reducing the pain associated with this and other injuries.

This research was funded by a grant from The Center for Child Injury Prevention Research Studies.

Poster Number 16

Visualizing Zebrafish Retinal Ganglion Cells by Transient Transfection with GFP

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Xenopus laevis tadpoles have long been used to examine early events in vertebrate visual system development, but the tetraploid genome limits the ability to manipulate gene expression in vivo. Zebrafish are an increasingly popular model vertebrate because they provide many of the advantages that Xenopus embryos provide, with the additional benefit of transgenics. In order to transition research questions in our laboratory from tadpoles to zebrafish, we adapted a technique to visualize individual retinal ganglion cells (RGCs). Specifically, we microinjected zebrafish embryos at the 1-2 cell stage with an Isl2b-EGFP DNA construct. Under the control of this islet promoter, enhanced green fluorescent protein (eGFP) is selectively expressed in a subset of RGCs. Three to four days after microinjection, larvae were fixed and retinas were flat mounted. Examination with confocal microscopy revealed GFP labeled neurons that displayed axons exiting at the optic nerve head as well as soma and dendritic morphologies consistent with those RGCs. This transient transfection method allows us to begin to examine RGC dendritic morphology in mutant and transgenic embryos in order to understand the molecular cues that influence RGC dendritic morphology and differentiation.

Poster Number 17

Effects of Long-term Exposure to Distracters on Sustained Attention Task Performance and New Discrimination Learning

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Acute exposure to distracters is known to decrease attention task performance in rats but little is known about the effects of long-term exposure to distracters on attention task performance or on subsequent learning. In the present experiment male Long-Evans rats (N = 15) were trained in a sustained attention task that required discrimination of brief visual signals (500, 100, 25 ms) from trials with no signal presentation. After reaching criterion, the animals continued to train in the same task (n = 7) or in the same task with a houselight in the back of the chamber flashing throughout the session (n = 8). Animals exposed to the houselight showed an initial decrease in accuracy, but their performance improved with subsequent training. All animals were then tested with trials inserted within the sustained attention task that required acquisition of a new visual discrimination. Animals exposed to the flashing houselight showed faster acquisition of the new visual discrimination task compared with animals that had not been exposed to the distracter. The present findings suggest that long-term exposure to a distracter may increase cognitive flexibility to support new learning. Future experiments will examine the neural basis of this increase in cognitive flexibility. Supported by AG 030646.

Poster Number 18

Effect of Insulin-like Growth Factor-I on Phospholipase C Beta4 Translocation in NIH/3T3 Fibroblasts

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The enzyme phospholipase C Beta4 (PLC Beta4), located in clock cells of the brain and in peripheral clock tissues such as the liver, is likely to play a role in metabolic and circadian processes through nuclear and intracellular cell signaling. Previous research has shown the concentration of PLC Beta4 shifts between the cytoplasm and nucleus of cells in the liver of mice in response to light and food; however, agents inducing nuclear translocation of PLC Beta4 have yet to be identified. Using NIH/3T3 fibroblasts in cell culture, the agent insulin-like growth factor I (IGF-I), known to induce nuclear translocation of PLC Beta1, was used to test for nuclear translocation of PLC Beta4. Fibroblasts were exposed to 2.5 ug/100 ul, 5 ug/100 ul, and 10 ug/100 ul doses of IGF-I for 1, 5, 15, 30, and 60 minutes. Immunofluorescent staining was used for visual evidence of protein translocation. There was evidence of nuclear translocation at 1, 5, and 15 minutes and cytoplasmic translocation after 30 and 60 minutes of exposure. The protein levels in the cytoplasm and nucleus of the cell will be quantified using Western blotting techniques. It is expected that the protein quantification results will provide supportive quantitative evidence of the qualitative data gathered. This process will be repeated to test for nuclear translocation of PLC Beta2 and PLC Beta3 for comparison. The expected results will be significant because it will positively identify IGF-I as an agent influencing the translocation of PLC Beta4 into the nucleus where the enzyme can influence nuclear and intracellular cell signaling.

Poster Number 19

Brain Awareness Week 2009 Small Group Based Elementary School Activities.

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For Brain Awareness Week 2009, five days of activities were developed for K-5th grades that promoted an elementary understanding of neuroscience in their daily life. The event took place at The Center for Knowledge, a public magnet elementary school in Columbia, South Carolina. The grades were split for five days of grade specific activities. Each day started with an introductory presentation, followed by small group activities led by undergraduate and graduate students. Each day was concluded with a group demonstration, reading a children's book related to the topic of the day, or by presenting the collected data to explain trends and discoveries. Kindergarteners explored the comparative anatomy of skulls from carnivores, herbivores, and omnivores followed by an egg drop experiment to demonstrate the importance of helmet safety. First graders explored the critical role of the brain in the 5 senses. Second graders explored comparative brain anatomy across different species. Third graders learned about neurons, neurotransmission, and neural circuits and completed a ruler drop experiment to calculate nerve conduction velocity. Fourth and fifth grade students learned about neurotoxins from spiders, puffer fish, and snakes in a hands-on demonstration of neurotransmission and inhibition. This event successfully augmented the elementary science curriculum by giving the students a basic understanding of neuroscience using fun hands-on activities.

Poster Number 20

Face Processing Strategies in Senior and Young Adults

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Previous data support changes in face processing and recognition in the typical aging and dementia populations. The basic cognitive mechanism for these changes is still unknown. The current study investigated the effects of aging on two types of face processing: featural, identification based on individual features such as the eyes or nose, and second-order relational processing, identification based on spacing between features. Based on existing developmental and aging literature, this study posed that older adults rely on relational processing less than young adults. To address this hypothesis, 18 young adults (19-23 years) and 11 senior adults (62-72 years) participated in a perceptual same/different face-matching task in which featural or relational manipulations occurred in different face pairs. Overall, reaction and accuracy rates indicated increased difficulty in face processing with age. Senior adults were slower and were less accurate than younger adults. Contrary to the hypothesis, both young adults and senior adults performed better on featural than relational face processing tasks. Our findings are in opposition to developmental data that indicate young adults of this age group rely more on relational than featural face processing strategies.

Poster Number 21

Modulation of Astrocyte Activation and Neuropathic Pain by Herpes Simplex Virus Driven Preproenkephalin and mu Opioid Receptor Expression in Primary Afferents.

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There is growing recognition of an important role for peripheral opioid analgesia in chronic neuropathic pain, a disabling condition that is often refractory to current analgesic therapies. The present study used recombinant herpes simplex virus type 1 (HSV-1) containing cDNA sequences for mu opioid receptor (HSV-MOR), human preproenkephalin (HSV-PPE), a combination infection with both HSV-MOR and HSV-PPE (HSV-MOR+PPE), or E. coli lacZ gene marker (HSV-LAC) to investigate the role of peripheral opioids in neuropathic pain-associated behaviors and astrocyte activation. Mice were infected on day 7 post-L5 spinal nerve transection with HSV-MOR, HSV-PPE, HSV-MOR+PPE, or HSV-LAC. Expression of mu opioid receptor and enkephalin increased in the skin, dorsal root ganglia, and lumbar spinal cord. SGMOR virus reversed mechanical allodynia, thermal hyperalgesia, and astrocyte activation. KPE virus reversed mechanical allodynia, had no effect on thermal hyperalgesia, and attenuated astrocyte activation. The combination of KPE with SGMOR viruses had no effect on mechanical allodynia or thermal hyperalgesia and increased astroctye activation. These results suggest that increasing primary afferent expression of the mu opioid receptor can reverse nerve injury induced astrocyte activation and allodynia and hyperalgesia.

Poster Number 22

Age-dependent, Ketamine-induced Apoptosis in the Rat Somatosensory Cortex.

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Ketamine is a general anesthesia used in emergency and battlefield medicine. Ketamine functions as an antagonist for the N-methyl-D-aspartate glutamate receptor (NMDAR). Although considered safe for use, recent clinical evidence suggests that, in very young children, general anesthetics such as ketamine may actually induce learning deficits when these children mature. To examine the potential neurotoxicity of ketamine, we injected postnatal day 7 (P7) rats with either vehicle (PBS) or ketamine (20 mg/kg). After eight hours, we examined coronal brain sections for activated caspase-3, a marker of apoptotic injury. We counted AC3 positive cells in somatosensory cortex using a non-biased stereological procedure. We found that ketamine could induce at least a six-fold increase in cells positive for the cell death marker. Recently, NIH has expressed concerns over the toxicity of general anesthesia in young children. Our research not only suggests this concern is valid, but further research is needed to more fully understand both the nature of this injury and how to prevent it from occurring.

Poster Number 23

Description of the Aquatic Brain of Tiger and Spotted Salamanders: Neural and Sensory Structural Comparisons Pre and Post Limb Development

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The amphibian brain can be seen as a prototype for vertebrate nervous systems. Many neuroscience studies have examined central nervous system structures of amphibians, especially the salamander. In this study, the in situ brains of the tiger salamander (Ambystoma tigrinum) and spotted salamander (Ambystoma maculatum) were compared at distinct aquatic stages of development. Specifically, we compared sensory and brain system structure at the early aquatic stage (Harrison stage 40) of ontogeny to the same structures at the late aquatic stage of development. The late aquatic stage occurred at approximately 100 days and was indicated by the retention of gills and aquatic life style. Larvae at the early aquatic stage lack limbs. Other than size, there are no discernable differences between brain structures at early and late aquatic stages. There are however, differences in the visual, vestibular and olfactory sensory structures that involve progressive degrees of maturation. Furthermore, evidence suggests that, although essential nervous system structures between species are similar, there are notable differences in their size. Specifically, the ventricular system achieves greater definition with age as the brain and body grow. Similarly, cells become more differentiated into specific structures between these stages. These results are discussed in terms of common proximal musculoskeletal activation patterns across species and across ontogenetic stages. Simply stated, the locomotor style used by both species in the early tadpole stage is the same as that used in the late aquatic stage after the animal grows limbs.

Poster Number 24

Magnitude Estimation Performance Predicts Right Hemisphere Lesions in Stroke Patients

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The psychophysical function, established by Stevens (1975), quantifies the relationship between perception and an objective measure of a stimulus. Three variables are used to describe the pattern of this function: the exponent, the constant, and the r-squared value. Although it has been observed that psychophysical function parameters are altered in the neurological condition spatial neglect, in this study we examine psychophysical functions in right hemisphere lesion patients (RHL) with and without neglect, left hemisphere lesion patients (LHL) without neglect, and control participants across a wide range of perceptual stimuli spanning 6 sensory domains. A series of one-way ANOVAs with planned contrasts for averaged psychophysical function parameters indicated that patients with RHL (N = 22) overestimated the intensity of lesser stimulus magnitudes and underestimated those of greater magnitudes when compared to controls (N=39), as indicated by decreased exponents and increased constants. RHL with neglect (N = 9) demonstrated decreased exponents compared with those without neglect. LHL patients (N = 12) did not perform significantly different from normal controls. Logistic regression analysis revealed that the three averaged function parameters predicated group membership (RHL or controls) successfully at a rate of 88%. Brain lesions were analyzed using subtraction techniques with the MRIcro and MRIcron software programs. Results of the imagining analysis revealed lesions related to the anterior limb of the internal capsule and superior thalamic radiations occurred more in patients with decreased exponents. Lesions related to the anterior limb and genu of the internal capsule were more likely in patients with increased constants.

Poster Number 25

Profile of phospholipase Cß4 Expression after a Nighttime Light Pulse in the Clock Neurons of the Mouse Brain.

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Circadian rhythms are 24-hour endogenous cycles that can be influenced by external stimuli such as light and food. These rhythms hold wide influence over everything from aspects of organisms' behavior like sleep cycles to biological processes such as patterns of gene expression. In mammals, the internal pacemaker of these daily cycles is the suprachiasmatic nucleus (SCN) located in the hypothalamus. The SCN clock neurons coordinate sensory inputs and their downstream effects throughout the body. In order to further understand the molecular mechanisms underlying SCN function, our lab has been studying the intracellular signaling protein, phospholipase C ß4 (PLCß4). In previous studies, we determined that PLCß4 cycles in the SCN over a 24-hour period with peak levels in the night. The current study aims to further investigate the role of PLCß4 as it relates to light's ability to reset the clock. Mice were kept on a 12 hour L:D cycle for at least two weeks. A 30-minute light pulse was administered at ZT 16 (four hours after their respective night). Mice were sacrificed at either 0, 15, 30, or 60 minutes post-pulse, transcardially perfused, and the brains were removed. To visualize plcbeta4 gene expression, the mouse brains were cut into 30 micrometer sections, processed using an in situ hybridization protocol, and viewed under a light microscope. Preliminary data show a peak in plcbeta4 expression in the SCN 15 minutes following a nighttime light pulse as compared to no-pulse controls. These results suggest PLCß4 may play a role in the light entrainment pathway.

Poster Number 26

Effect of Juvenile Social Isolation on Anxiety-Like Behaviors and Ethanol Drinking in Male Long-Evans Rats

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Previous rodent studies have demonstrated that adolescent social isolation results in numerous behavioral perturbations including increased anxiety-like behaviors, impulsiveness, and deficits in sensory gating. In addition, juvenile social isolation can increase ethanol self-administration, possibly due to the enduring increases in anxiety- and impulsivity-like behaviors. The goal of this study was to determine if juvenile social isolation increases ethanol drinking using an intermittent access procedure that has recently been shown to engender relatively high ethanol intake. Sixteen Long-Evans rats were randomly divided into two housing groups at postnatal day 28. One group was housed individually (socially isolated, SI) and the other in groups of four (group housed, GH). After six weeks, rats were tested in the elevated plus-maze and response to novelty assay and, immediately following these behavioral procedures, all subjects were single housed and ethanol drinking was assessed. SI rats exhibited a significant increase in open arm exploration time on the elevated-plus maze as well as a greater response to a novel object. In addition, SI rats drank significantly more ethanol in the intermittent two-bottle choice drinking procedure (20% ethanol/water available days/week). Surprisingly, when the ethanol intake of GH and SI rats was compared with that of rats maintained under standard housing conditions (singly housed as adults), it appeared that the GH condition had the predominant effect on drinking as ethanol consumption in SI rats was identical to that observed in standard housed subjects. Taken together, these data provide further evidence that juvenile social isolation may represent a good model with which to study the influence of anxiety-like behaviors on ethanol drinking. Interestingly, these data also suggest that the standard housing conditions employed in most rodent home-cage drinking studies may actually recapitulate the ethanol drinking phenotype typically associated with juvenile social isolation.

Poster Number 27

FGF-2's Effects on Retinal Dendrite Morphology do not Influence Xenopus' Phototactic Behavior

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Neurons are compartmentalized so that their dendrites receive synaptic input from other neurons. Dendritic development can be affected by a variety of molecular cues from the environment such as fibroblast growth factor (FGF). Interestingly, previous work in our lab has demonstrated that both exogenous application of FGF-2 as well as inhibition of FGF receptors with DMBI enhance Xenopus Retinal Ganglion Cell dendritic arborization in vivo. Given that RGC axons are the sole output of visual information from the retinal to the brain, this project asked if FGF-induced changes in dendrite morphology affected tadpole visual behavior. In order to address this question, we first developed a simple assay to measure tadpole phototactic behavior. When given a choice, sighted tadpoles preferred bright areas over dark areas, while blind tadpoles showed no preference for light vs. dark areas. Then, to determine if alterations in retinal FGF affected tadpole vision, exogenous FGF-2 or the FGF receptor inhibitor DMBI were microinjected into both retinas. Placed in the phototactic behavioral assay, these FGF- and DMBI-treated tadpoles preferred light areas indistinguishable from controls. Consequently, alterations in retinal FGF levels that enhance RGC dendritic arborization in vivo do not affect the visual ability to discriminate light versus dark.

Poster Number 28

Altered Biogenesis of Brain MicroRNAs in 22q11 Deletion Syndrome-induced Schizophrenia.

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People born with 22q11 deletion syndrome (22q11ds), a genetic disorder where either 1.5 or 3 mB are deleted on chromosome 22, are 25 times more likely to develop schizophrenia than the average person. One gene in the deletion, DGCR8 , codes for a protein integral in the biogenesis of brain-specific microRNAs. We sought to verify predicted targets of microRNAs downregulated in 22q11ds and then determine which of these targets controls aspects of schizophrenia etiology. We cloned predicted targets into GFP-producing expression vectors and microRNAs into non-GFP-producing expression vectors, and subsequently cotransfected human embryonic kidney cells. We are currently in the verification process which will precede knockdowns and knockouts of verified potential schizophrenia-relevant genes.

Poster Number 29

Optimizing Delivery of Lentiviral Vectors in the Rat Hippocampus

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Epilepsy affects approximately three-million Americans. Kainate receptors, a subtype of glutamate receptors, contribute to development of epilepsy. To study the role of kainate receptors in epilepsy, we would like to manipulate the expression of specific kainate receptor subunits. To do this we use a form of gene therapy in which lentiviral vectors are used to introduce kainate receptor subunit genes into neurons in the hippocampus. Our ultimate goal is to study the effects of kainate receptors in epilepsy.The first step towards achieving this goal is to optimize delivery of the lentiviral vector. Twelve male Sprague-Dawley rats, three per group, received hippocampal injections of a lentiviral vector containing either CaMKII, Synapsin, or PGK promoters driving expression of green fluorescent protein (GFP). To compare vector spread the lentiviral vector was injected into either a single or multiple sites in the hippocampus. The animals were allowed to recover for four weeks while the virus transduced hippocampal neurons. Then, the rats were perfused and their hippocampi were examined for GFP expression. We found that lentiviral vectors with the PGK promoter produced the most robust GFP expression, followed by Synapsin and CaMKII. Vectors containing the PGK promoter caused GFP expression in most neuron types in the hippocampus. Comparison of the two injection strategies revealed different patterns of GFP expression. In future studies we will insert kainate receptor subunit genes into this vector to evaluate the role of kainate receptors in epilepsy. Supported by an SCHC Undergraduate Research Fellowship (KSM) and NIH grant NS065869 (DDM).

Poster Number 30

Fimbria-Fornix Transection Impairs Performance on a Delayed Non-matching to Position Task.

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Alzheimer's disease (AD) is a neurodegenerative disorder affecting the hippocampus and other areas related to proper memory function. AD is accompanied by a decline in cognitive abilities associated with memory performance, including short-term memory. The fimbria-fornix is a major projection between the hippocampus and the septum that comprises the septohippocampal pathway. Damage to this pathway has been known to cause impairment in spatial memory function. 18 male Sprague-Dawley rats received either bilateral fimbria-fornix transections or sham operations after reaching criterion in a delayed non-matching to position (DNMTP) task, known to be sensitive to alterations in spatial memory after limbic system injury. After a 6-8 day post-operative recovery period, rats were tested for spatial memory function at a 1 sec, 5 sec, and 15 sec delay for 14 consecutive days and 5 days per week for the following 10 weeks. Rats were found to have impaired performance on the DNMTP task with some behavioral recovery but the rats never returned to pre-operative performance. Performance was impaired in a delay-dependent fashion with the most impairment after a 15-sec delay and the least after a 1-sec delay. There was also a delay-dependent tendency towards perseverative choice behavior towards the left side. Histological analysis with an AChE stain confirmed deafferentation of the hippocampal formation, revealing the complete transection of the fimbria-fornix in surgical cases. The delay-dependent behavioral impairment and accompanying histological data support previous findings that implicate the septohippocampal pathway in spatial memory function. This connection between the hippocampal formation and the septum appears to be crucial for working memory on a spatial task.

Poster Number 31

A Model for Screening Chemesthetic Irritants Activating TRPA1 Channels using Rescued Painless Drosophila

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Chemesthesis is the sense of irritation elicited by chemicals. The transient receptor protein channel TRPA1 of mammals is activated by over 90 irritant compounds and plays an important role in chemesthesis. *Drosophila melanogaster* expresses at least three evolutionary homologs of mammalian TRPA1: dTRPA1, Pyrexia, and Painless. We are studying the ability of the painless gene, expressed in fly taste receptor neurons, to detect irritants. Previously, we demonstrated significant differences in the ability of Painless mutants (lacking the painless gene) and wild-type flies to avoid 10 chemical irritants using a feeding assay. In the present experiment we confirmed this finding in "rescued" flies which had painless re-engineered into their genome. Theoretically, responses of the rescued flies should be similar to responses of the wild-type flies tested previously. This would confirm that the behavioral responses observed in the Painless mutants are due to painless and not some other factor. Fifty flies were starved in 2% agar vials for 24 hours and then placed in a 96-well plate test arena with half the wells containing 1% sucrose + dye (control) and half containing 1% sucrose + 10mM of irritant + different color dye (experimental). After 1 hour of feeding, the flies were frozen and the color of their abdomens examined under a microscope for analysis. Our results indicate that rescued flies behaved significantly different from the Painless knockout flies, demonstrating that painless is responsible for the detection of the irritants tested. The Painless mutant appears to be a good model for determining which irritants activate TRPA1 channels.

Poster Number 32

Enhanced Synaptic Efficacy: Linking Functional and Anatomical Recovery in the Crossed Temporodentate Pathway

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Following a unilateral lesion to the entorhinal cortex, rats lose the ability to perform on a spatial alternating task- a test of spatial working memory. Between eight and 12 days following the lesion, a pathway from the non-lesioned entorhinal cortex to the dentate gyrus, known as the crossed temporodentate pathway (CTD), expands to compensate for the loss of input to the dentate gyrus. The sprouting of the CTD is accelerated when the entorhinal cortex lesion is divided into two surgeries separated by six days. This progressive lesion also causes accelerated recovery of performance on the spatial alternating task to preoperative levels. Long-term potentiation (LTP) is a candidate for the physiological mechanism that links the observed anatomical and functional recovery. This study examined this possibility through electrophysiological analysis of the CTD six days following completion of the entorhinal cortex lesion. LTP was defined as a 115% increase in evoked potential slope or amplitude following high-frequency tetanization of the CTD, relative to pre-tetanization values. Though rats with progressive lesions demonstrate behavioral recovery at six days following lesion completion, the capacity for LTP is not observed at this time point, suggesting that LTP is not the physiological mechanism that accounts for the functional recovery. Rats receiving two surgeries separated by six days demonstrated enhanced synaptic efficacy while rats receiving two surgeries separated by 12 days did not. This suggests that the mechanisms that account for accelerated sprouting in progressively lesioned rats have an upper limit on how long the mechanisms can survive.

Poster Number 33

A Case Study of Using IMPULSE in an Undergraduate Course.

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The online journal IMPULSE has published undergraduate neuroscience articles since 2003, as well as offered students the opportunity to serve as peer reviewers. The journal helps faculty engage and motivate students working in their research labs and integrate advanced scientific writing into a class. A molecular neurobiology course taught at Ursinus College by one of us (RK) required the 20 students to prepare and submit a manuscript to IMPULSE from their semester-long, small-group research projects. The syllabus was planned so that laboratory research coincided with preparing the background and writing the manuscript. Working collaboratively, the students completed their projects using Daphnia magna to examine the impact of drugs on the nervous system in time to submit draft papers by mid-semester (mid-March). IMPULSE reviewed the submissions and returned them by mid-April, and the students resubmitted them before the end of spring semester. Students reported that they spent more time researching and writing the paper with the possibility of publication at stake. They also focused on completing the assays and included a larger data set than they would have for a class-only assignment. The professor found that she was more thorough in critiquing students' papers and, rather than setting expectations for an undergraduate course, she considered what a published author should produce. In responding to reviewers' comments, the students spent more time rewriting sections of the paper and were more thorough in their revisions than in previous courses. Incorporating journal submission into the course promoted a better writing experience for the students and a deeper examination of the neuroscience involved. The exercise of submitting manuscripts to IMPULSE as an integral part of an undergraduate course was a successful learning experience.

Poster Number 34

Attention-Deficit/Hyperactivity Disorder in Childhood Affects Cognitive Test Profiles in the Geriatric Population but is not Associated with the Development of Mild Cognitive Impairment or Alzheimer's Disease.

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Attention deficit hyperactivity disorder (ADHD) is a common childhood learning disorder. The frequency of ADHD in the aging population and its relationship to late-life cognitive decline or the development of dementia has not been studied previously. The Wender-Utah ADHD Rating scale was administered to cognitively normal or impaired geriatric research subjects followed longitudinally at the University of Kentucky Alzheimer's Disease Center. The frequency of ADHD and the association of ADHD diagnosis with cognitive test profiles and diagnosis of mild cognitive impairment (MCI) or dementia were assessed using standard comparative statistical measures. Wender-Utah results were obtained from 320 subjects with normal cognition (n=227), MCI (n=39), cognitive impairment not meeting criteria for dementia or MCI (n=9), and early dementia (n=17). The frequency of ADHD in this sample was 6%. ADHD diagnoses were not related to cognitive diagnoses. Cognitive test profiles demonstrate impairment in tasks requiring sustained attention including Category Fluency ($p=0.003$, Student's t-test) and Trailmaking Test A ($p=0.02$, Student's t-test) for subjects with ADHD. The frequency of ADHD appears stable across generations. The finding of deficits in attentional processing in ADHD subjects in their geriatric years suggests that such traits are stable throughout life and need to be considered when interpreting cognitive test profiles. In addition, ADHD does not appear to be a risk factor for the development of MCI or dementia.

Poster Number 35

Characterization of the Effects of Subthalamic Nucleus Inhibition or mu-opioid Receptor Stimulation on Palatable Feeding and Progressive Ratio Performance in the Rat.

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The subthalamic nucleus (STN) is a brain structure that is part of motor circuitry within the basal ganglia. To alleviate the motor deficits caused by Parkinson's disease, some patients electrically stimulate the STN, a procedure known as deep brain stimulation. However, because the STN processes associative and limbic information, treatments that target it may also cause alterations in motivated behavior. In these experiments, we tested whether pharmacological inhibition of the STN, or stimulation of STN mu-opioid receptors, would affect consummatory or appetitive behavior directed at a palatable food source in the rat. All animals underwent surgical implantation of bilateral guide cannulas above the STN. Two groups of rats were then acclimated to 2-hr daily access of a palatable high fat/sucrose diet. An additional group was trained to lever press for sucrose reinforcement under a progressive ratio schedule. Once rats had completed habituation or training, they received intra-STN injections of muscimol (at 0-5 ng/side) or DAMGO (at 0, 0.025, or .25 µg/side) prior to a feeding test (Experiment 1 and 2) or to a progressive ratio session (Experiment 3). There were no observed effects of STN inhibition with muscimol upon 2-hr food intake. In contrast, stimulation of STN mu-opioid receptors caused a modest increase of feeding on the palatable diet late in the 2-hr session. Neither muscimol nor DAMGO treatment altered progressive ratio performance in a small cohort of rats (N = 6). These data suggest that, unlike other nodes within the basal ganglia circuitry, STN inhibition or mu-opioid receptor stimulation have limited effects on appetitive and consummatory behavior.

Poster Number 36

The Source and Effects of Serotonin on the Abdominal Pump in the Dragonfly nymph, *Libellulids libellula*.

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Dragonfly nymphs, *Libellulids libellula*, have a unique multipurpose abdominal pump that is used for breathing, locomotion and feeding. In breathing, the pump pulls water into the tracheal system where gas exchange takes place. The nymphs rapidly move through the water by using the abdominal pump to propel water out from the anus, while in feeding the pump is used for rapid labial extension. The neurotransmitter, serotonin, initiates and enhances contractile activities of the gut in crayfish, another aquatic arthropod. The serotonin that produces contractions appears to be borrowed from the hemolymph. We asked whether serotonin has a similar effect on the dragonfly nymph's abdominal pump as it does on crayfish hindgut. Both are freshwater arthropods with open circulatory systems and in both the gut is a large organ that may be important in regulating hemolymph levels of serotonin. Given this, we hypothesized that serotonin may be borrowed in *Libellulids libellula* and affect contractile properties of the abdominal pump. We predicted that if serotonin is important in the dragonfly abdominal pump, we should see it increase activity from a quiescent state. Also, if serotonin is borrowed, we should be able to identify cells that take up serotonin. We found that serotonin is normally found on the hindgut and appears not to be borrowed; however serotonin does stimulate contractile activity of the abdominal pump. We also found that the abdominal pump receives central nervous system input. Our studies suggest that innervation of the gut may arise from the first abdominal ganglion.

Poster Number 37

Supplemental Choline Protects the Brain Against Alcohol-induced Amnesia in Adolescent Rats

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Choline is a dietary nutrient that is necessary for proper brain development and function. Several studies have shown that extra dietary choline given during critical periods of brain growth can protect the brain against insult, such as normal aging or fetal alcohol exposure. Our lab has recently shown in adolescent rats that alcohol administration given post-training can lead to severe amnesia for a previously learned hippocampus-dependent task (trace conditioning) but has no effect on a non-hippocampus variation (delay conditioning). In this experiment we asked whether supplemental choline would protect the brain against later alcohol amnesia. Animals were given a daily subcutaneous injection of choline chloride or saline (vehicle) on postnatal days (PD) 15-26. On PD30 animals were trained in a trace fear conditioning procedure. For training, animals were given 5 pairings of a 10 s light and shock; the trace interval was 10 s. For three days after training, animals were administered alcohol (2.5 g/kg/day i.g.) or water. Testing occurred 24 h after the final alcohol dose. For test, freezing (immobility) elicited by the light was scored. The results showed that animals injected with saline on PD 15-26 exhibited ethanol amnesia; freezing was reduced in post-training alcohol groups relative to water controls. Of interest, animals injected with choline did not show this amnesia. Choline-treated subjects given alcohol post-training exhibited just as much freezing as the animals given water. The results indicate that extra dietary choline early in life is protective against alcohol-induced insult to hippocampus-dependent memory.

Poster Number 38

Intracellular Signaling of Lysophospholipid Induced Growth Cone Collapse of Chicken Retinal Neurons

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Developing neurons use growth cones (fanlike structure at the end of developing axons) to sense attractive and repulsive chemical cues to grow towards and form a synapse at the target. We are investigating the effect of two lipids, lysophosphatidic acid (LPA) and Sphingosine-1-phosphate (S1P), on developing chicken retinal ganglion cells (RGCs). We have established that LPA and S1P are inhibitory and cause growth cone collapse. LPA and S1P activate specific G-Protein Coupled Receptors (GPCRs). Therefore we wanted to determine the GPCR intracellular signaling pathways involved in LPA or S1P induced growth cone collapse of chicken RGCs. There are four G-Proteins associated with GPCRs: Gi, Gq, Gs, and G12/13. We used inhibitors to block representative downstream pathways and compared the growth cone collapse rates to controls. LPA induced growth cone collapse was significantly reduced when pathways associated with Gs (Adenyl Cyclase, PKA) and G12/13 (Rho, ROCK) were blocked. Reducing intracellular calcium ion concentration via chelation, associated with Gq, and using pertussis toxin to directly inhibit Gi had no effect on LPA induced growth cone collapse. In conclusion, our preliminary data show that Gs and G12/13 are probably integral to LPA induced growth cone collapse, whereas Gi and G12/13 do not seem to be involved. S1P induced growth cone collapse was not observed for Gs and G12/13 ; however, Gq does not seem to be involved in S1P induced growth cone collapse. Furthermore, our preliminary data with pertussis toxin suggests that Gi may be required for S1P, but not LPA, induced growth cone collapse.

Poster Number 39

Postnatal Supplemental Choline Facilitates Extinction of Fear in Rats Trained as Adolescents

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Supplemental administration of the essential nutrient choline during pre-and/or postnatal development can result in substantial and long-lasting benefits to cognitive performance. Here we evaluated the effects of supplemental choline on long delay and unpaired conditioning in rats trained on PD 30. Subjects were given a single daily sc injection of choline chloride or saline on PD 15-26. On PD 30 subjects were trained in either a long delay or unpaired fear conditioning task in which the conditioned stimulus was a flashing light and the unconditioned stimulus was a mild footshock. Subjects were tested on PD 31 without shocks given during the test. Choline-treated rats, in contrast to the saline-treated group, exhibited facilitated extinction of fear. Choline did not affect initial levels of freezing. In a follow-up to this experiment, we focused primarily on the role choline plays in extinction. The animals were injected with choline or saline on PD 15-26. In this experiment we gave the animals more training trials, and evaluated both short-term and long-term extinction of conditioned fear. On PD 30-31, the animals were classically trained in long delay or unpaired training. They underwent three extinction testing sessions on PD 32-34. Initial results indicate that choline does in fact lead to more rapid extinction, but does not affect initial levels of learning. The findings suggest that supplemental choline may lead to improvements in frontal lobe functioning.

Poster Number 40

Examining Growth Cone Dynamics in Depolarizing Conditions

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Growth cones, the dynamic ends of developing neurons, rely on diffusible and membrane-bound guidance cues to reach their synaptic target locations. Recent research has revealed that attractive guidance cues cause growth cones to turn toward the source and depolarize, while repulsive cues cause growth cones to turn away and hyperpolarize. Additionally, other labs have shown that depolarization leads to growth cone collapse, indicating that membrane potential plays a significant role in cytoskeletal structure. We used time-lapse imaging to analyze how alterations in membrane potential affect growth cone dynamics in vitro. Growth cones were observed in flowing culture media for an initial 30-minute control period, then depolarized with elevated K+ for 30 minutes, and then finally returned to control conditions for an additional 30-minute recovery period. Parameters of growth cone dynamics including extension rate, number of filopodia, lamellopodial area, and branching frequency were all analyzed throughout these three 30-minute observation periods. Studying these parameters provides insight into the role of membrane potential in the guidance of axons to their targets.

Poster Number 41

Choline Acetyltransferase Expression in the Rat Septum During Hippocampal Sprouting

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Many studies have examined the expression of choline acetyltransferase (ChAT) during septodentate axonal sprouting in the dentate gyrus, but none have yet examined it in the cells of the medial septum--the cells that actually do the sprouting. To induce septodentate sprouting, the entorhinal cortices of rats were electrically lesioned on one side, leaving the sham side as a control. The rats' brains were removed 4 and 7 days post-lesion, RNA was isolated and preserved from the medial septa, and the hippocampi were stained for acetycholinesterase to determine if sprouting was occurring. Real-time reverse transcriptase PCR was used to determine the amount of ChAT expression relative to the amount of GAPDH expression, a control gene, in the medial septa at the 4 day and 7 day time points. Though sprouting was observed to be occurring, no significant difference in expression between ChAT and GAPDH was detected both within and between the 4 or 7-day groups. Future studies could determine why no ChAT upregulation was detected.

Poster Number 42

Study of Axon guidance in Retina of LPA1-5 receptors using Reverse Transcription-Polymerase Chain Reaction

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Axon guidance is the mechanism by which axons receive and transmit chemical signals that allow the axon to guide itself to a specific target region. In understanding axon guidance cues, surface receptors are being studied to determine their role in guiding axons to specific target regions. The visual system is being studied in the chick model to understand how retinal ganglion cells (RGC's) are able to guide themselves and make connections into the optic tectum. Retinal Ganglion Cells were chosen because these are the only cells found in the eye that extend their axons out to make connections in the optic tectum to allow processing of visual stimuli being received by the retina. Lysophosphatidic Acid (LPA) is a lysophospholipid that is found throughout the body and is responsible for a multitude of cellular responses such as cell proliferation, growth cone collapse, and much more. My research question was which of the five known LPA receptors are present during different stages of retinal development? Six days of development were selected to determine if LPA is present or not during development: E5, E6, E7, E8, E10, E12. The specific receptors being studied were LPA1-5. Reverse Transcription-Polymerase Chain Reaction (RT-PCR) preliminary results suggest which receptors are being expressed. We found that LPA1-4 is expressed in both brain and retina; however, LPA5 is being expressed only in the brain and not the retina. Expression has been seen in all of the six ages of development. Future works will include in situ hybridization.

Poster Number 43

Determining the Rewarding Effects of Acute and Chronic Toluene Exposure Using the Conditioned Place Preference Paradigm and [F18]FDG/micro-PET Imaging in Mice

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The high abuse liability of the organic solvent toluene has motivated addiction researchers to examine the as-yet uncharacterized neural mechanisms behind toluene's effects on craving and other reward-related behaviors. The current study inspects the effects of acute and chronic exposure to toluene on drug preference behaviors using conditioned place preference (CPP) as well as brain metabolic activity (BMA) using [18F]Fludeoxyglucose ([18F]FDG)/Micro Positron Emission Tomography (micro-PET) imaging to measure reward. Toluene and ethanol (EtOH) yield similar behavioral effects; therefore, it stands to reason that toluene, like EtOH, will manifest biphasic dose effects on reward behavior (i.e., low doses are rewarding and high doses produce aversion). The current research will explore a correlation between the behavioral paradigm and brain imaging, hypothesizing that (a) acute exposure (low dose) will yield greater rates of BMA compared to control BMA, with an inverse effect in the high dose group, and (b) mice in the low dose group will prefer the chamber where they received toluene and display greater rates of BMA, with a reverse effect in the high dose group. Adolescent (5 week-old) C57 mice (n=24) were assigned to control (0 ppm), low (700 ppm) and high (3200 ppm) toluene dosage groups. The mice received their respective doses when they were inside one of two assigned stimuli-distinct CPP chambers as follows: (a) acutely (first conditioning day, following habituation to CPP apparatus) and (b) chronically (daily for 2 weeks, alternating with pure air). Imaging occurred a day after habituation to obtain baseline brain activity, immediately after acute exposure, and 5 days after the last day of chronic exposure. Before micro-PET the mice received an IP injection of [18F] FDG prior to exposure to pure air within their paired-drug chambers. Micro-PET images have been reconstructed but remain to be analyzed, although CPP data shows that mice in the low dose group spent significantly less time in the paired chamber than the nonpaired ($t=5.6245$, $p<.05$). Because this indicates an aversion to a low dose of toluene (which runs counter to the hypothesis that mice in the low dose group would find toluene rewarding) further investigation of this event is necessary. Micro-PET results will help clarify this finding as well as any imprints that toluene leaves on the brain, from the perspectives of both acute and chronic use.