experiments with mathematical and statistical models to fill important knowledge gaps that can aid in their recovery. The novelty of our approach is using a series of linked courses that provide authentic research experience for both undergraduate and graduate students. Our presentation will use a case example of the federally endangered James Spinymussel and a team of students doing course-embedded research in Population Ecology, Mathematical Models in Biology, and independent research credits. Field studies are utilizing state-of-the art mark-recapture methods to quantify detection probabilities, population size and mortality rates. Artificial stream channel experiments are exploring environmental conditions that effect surface expression, and therefore the probability of detecting the species when it is present. Both approaches are integrating with mathematical and statistical models which are united into a conservation action plan.

Posters

MODELING HABITAT USE FOR THE ENDANGERED JAMES SPINYMUSSEL (*PLEUROBEMA COLLINA*): AN APPROACH FOR SETECTING RARE CRYPTIC ORGANISMS. Dorottya K. Boisen, Dakota M. Kobler, Katie M. Sipes, Alaina C. Esposito, Patrice M. Ludwig & Christine L. May, Department of Biology, James Madison University, Harrisonburg VA 22807. Freshwater mussels are keystone species in their ecosystems, and their filter feeding ameliorates water quality in downstream areas. Over 70% of freshwater mussel species worldwide are listed as vulnerable or more greatly threatened. The James Spinymussel (*Pleurobema collina*) is a species of top priority for conservation in Virginia. Due to limited research, cryptic appearance and behavior, and small population sizes, freshwater mussel conservation efforts have been hindered. A mark and recapture study has tracked approximately 20 James Spinymussels and 60 Notched Rainbow (*Villosa constricta*) mussels marked with Passive Integrated Transponder (PIT) tags at Swift Run in the summer of 2014. Multiple mussel recapture histories provide data about habitat use, stream-bed surface expression, and inform source sink models. We will present our work on this integrated approach to understanding rare cryptic organisms. This research is funded by the Jeffress Memorial Trust.

Medical Sciences

DESIGN AND SAR STUDY OF SMALL MOLECULE CTBP INHIBITORS. S. Korwar[1], B. L. Morris[2], S. R. Grossman[2] & K. C. Ellis[1,2], [1]Department of Medicinal Chemistry & [2] Massey Cancer Center, Virginia Commonwealth University. This project involves developing small molecule inhibitors of C-terminal Binding Protein (CtBP) which act as anti-cancer agents. CtBP is a transcriptional co-repressor of several tumor suppressor genes. It is over expressed in many colon, breast, and ovarian cancer tumors. CtBP has a catalytic site in which the co-factor NADH and substrate 4-

methylthio-2-oxobutyric acid (MTOB) bind. At higher concentrations, MTOB has been found to inhibit CtBP (IC_{50} = 300 μM). Based on the pi-interactions observed in CtBP-MTOB co-crystal structure, the methylthio- group of MTOB was replaced by a phenyl ring to give phenylpyruvic acid (PPA), which was found to be active (IC_{50} = 116 μM).This further led to the synthesis of non-reducible ketone isosteres of PPA, of which the hydroxy imine analog (HIPP) was the most potent compound (IC_{50} = 0.24 μM). Compounds containing various substituents such as hydroxy-, fluoro-, chloro-, methoxy- and methyl- at the 2, 3 and 4 positions of HIPP were then synthesized. The synthesis of these compounds was achieved in two steps. The first step involved condensing the corresponding aldehyde with hydantoin or 1,4-diacetylpiperazine-2,5-dione, followed by hydrolysis under basic or acidic conditions respectively to give the corresponding α-ketoacid. These α-ketoacids were converted to oximes in a one-step reaction. The α-ketoacids and oximes were tested against recombinant CtBP. The oximes of 4-chlorophenylpyruvic acid (IC_{50} = 0.18 μM) and 3-chlorophenylpyruvic acid (IC_{50} = 0.17 μM) were the most potent compounds in this series. All the compounds were further tested in HCT-116 cells using MTT assay.

ROLE OF TOLL-LIKE RECEPTOR 4 IN ENTERIC GLIA. S. Bhave, P. Brun, M. Kang, W.L. Dewey & H. I. Akbarali, Department of Pharmacology & Toxicology, Virginia Commonwealth University, Richmond, VA 23298. Toll-like receptors (TLRs) are a class of pattern recognition receptors that play an important role in mediating inflammatory responses to pathogens. TLR4 recognizes lipopolysaccharide (LPS), a membrane component of gram negative bacteria. In the gastrointestinal tract, basal activation of TLR4 by LPS has a protective role. However, a compromised gut epithelial barrier leads to enhanced entry of bacterial proteins such as LPS into the lamina propria, leading to neuronal toxicity. Enteric glia are important for the maintenance of neuronal integrity. In the present study we determined the activation of enteric glia in response to low and high concentrations of LPS. LPS dose-dependently increased mRNA expression of IL-6 and TNF-α with 0.1 μg/ml inducing a 25-fold and 10-fold IL-6 and TNF-α mRNA expression, respectively. A 100-fold higher concentration of LPS (1-100 μg/ml) lead to expression of connexin-43 hemichannels and induced ATP release indicative of cell damage. Whole cell voltage clamp studies showed that high concentration of LPS also increased ATP-induced inward currents with a corresponding increase in P2X1, 2, 3, 4, and 7 receptor mRNA expression. Thus, low concentrations of LPS induce cytokine release whereas high concentrations of LPS induce ATP release and P2X receptor activity and further enhance the release of pro-inflammatory cytokines. Understanding the role of P2X in LPS induced enteric glia activation and associated inflammation will point to new therapeutic targets to control gut inflammation while still maintaining the beneficial effects of basal TLR4 signaling. (Supported by DK046367, DA024009)

COMPUTATIONAL DESIGN OF DIRECT ALLOSTERIC THROMBIN INHIBITORS. D. K. Afosah[1,2], S. Verespy[2,3], R. Karuturi[1,2], & U. R. Desai[1,2],

[1]Department of Medicinal Chemistry & [2]Institute for Structural Biology & Drug Discovery, Virginia Commonwealth University, Richmond VA 23219 & [3]Department of Chemistry, Virginia Commonwealth University, Richmond VA 23284. Heparin mimetics present a valuable way of preventing the side effects associated with the use of heparin in thrombotic diseases. Sulfated benzofuran dimers (SBDs), which directly inhibit thrombin by an allosteric mechanism have previously been developed by our group. Considering that the crystal structure of the thrombin–SBD complex remains unsolved to date, we employed molecular modeling to help understand the SAR of these compounds and advance the design of these inhibitors. Using GOLD, a genetic algorithm-based docking and scoring program, a diverse virtual library of SBD analogs were screened to identify potential analogs as thrombin inhibitors. This was followed by chemical synthesis and biological evaluation of the most promising agents. Molecular modeling predicted that benzylated 5-sulfate and phenethyl 5-sulfate containing benzofurans were likely to exhibit higher potency due to pi–pi ligand–protein interaction. Yet, the putative designed inhibitors did not exhibit inhibition potency as predicted. Yet, interestingly majority of these analogs displayed maximal efficacy of only 50-60%, which is different from those reported in the literature. Partial inhibition of thrombin is likely to reduce the risk of bleeding, which typically accompanies inhibitors that completely inhibit this key coagulation factor. Further work is underway to identify the structural basis for the submaximal inhibition of thrombin by these benzofuran analogs.

ESTIMATES OF METABOLIC EXPENDITURE CAPACITY IN CHRONIC LIVER DISEASE (CLD) PATIENTS CAN DIFFER WIDELY BETWEEN CARDIOPULMONARY EXERCISE TESTS (CPET) OF PERFORMANCE AND SELF-REPORTS OF ACTIVITY. Jillian K. Price[1,2,3], Carey Escheik[3], Patrick Austin[3], Lynn Gerber[2,3] & Zobair M. Younossi[3], [1]Department of Rehabilitation Science, George Mason University, [2]Center for Chronic Illness & Disease, George Mason University, [3]Beatty Liver & Obesity Research Program, Inova Health System. The potential impact of chronic liver disease (CLD) on cardiopulmonary function has not been well characterized. Cardiopulmonary exercise testing (CPET) is a strongly validated means of assessing cardiopulmonary function and performance. The Human Activity Profile (HAP) is a questionnaire developed to detect subtle changes in functional level whose scores can be converted to metabolic equivalent (METs) estimates of energy expenditure and has been well-validated in a variety of clinical populations. A convenience sample of 10 subjects seeking treatment at a tertiary liver disease center were recruited to completed the HAP and a modified Bruce CPET [70% male, age 50.4 ± 9.3 years, 3 NAFLD, 7 HCV (4 naïve, 2, relapse, 1 sustained virologic response-SVR), body mass index (BMI) 30.2 ± 6.8]. VO2 Max-derived calculations of metabolic equivalents (METs) differed significantly from self-report-based METs. Maximum METs estimates ranged from 94 to 176% of performance VO2Max. All but one subject overestimated their METs capacity during self-report. All subject self-reports of average daily activity METs expenditure were higher than their anaerobic threshold

(24-213% higher). The general population-derived METs estimate for the HAP requires modification for a CLD population.

CB1 HETEROZYGOUS MICE REVEAL IN VIVO EFFICACY DIFFERENCES OF PHYTO AND SYNTHETIC CANNABINOIDS. T. W. Grim[1], B. F. Thomas[2], J. L. Wiley[2], S. S. Negus[1], & L. H. Lichtman[1], [1]Department of Pharmacology & Toxicology, Virginia Commonwealth University, Richmond, VA 23298 & [2]Research Triangle Institute, Raleigh-Durham, NC 27709. Within the last decade, myriad synthetic cannabinoids (SC) have been detected in abused herbal preparations. Most SCs activate the CB1 receptor to a much greater degree than the phytocannabinoid r^9-tetrahydrocannabinol (THC), rendering them considerably more dangerous. Here, we utilize CB1 (+/+), (+/-), and (-/-) mice to assess these differences in efficacy to produce CB1-mediated catalepsy, hypothermia, and antinociception, and we hypothesize that the potency to produce these effects in CB1 (+/-) will decrease relative to wild-type mice as efficacy decreases with little or no effect in CB1 (-/-) mice. When potency ratios were calculated between CB1 (+/+) and (+/-) mice across each endpoint, these experiments produced a rank order of A-834,735D > WIN55,212-2 > CP55,940 > JWH-073 > CP47,497 > THC. This result aligns with in vitro assays investigating functional activity of these ligands at CB1.

MARBLE BURYING: NOVEL MEASURE OF MURINE NEUROPATHIC PAIN DEPRESSED BEHAVIOR. Jenny L. Wilkerson[1], Ken Hsu[2], Micah Niphakis[2], Mario van der Stelt[3], Benjamin Cravatt[2] & Aron H. Lichtman[1], [1]Dept. Pharmacology & Toxicology, VCU, Richmond, VA 23298, USA [2]The Skaggs Institute, TSRI, La Jolla CA 92037, USA & [3]Leiden Instit. of Chem & NL Proteomics Centre, Leiden, NL. Pathological pain states represent one of the most common reasons to seek medical attention. Most preclinical assays measure behaviors that are evoked by a nociceptive stimulus. Limitations of these endpoints include difficulties in distinguishing between motor impairment and analgesia, as well as the conceptual issue of whether they adequately model clinical symptoms. In the present study, we modified the marble burying test to explore pain-depressed digging behavior in the chronic constriction injury of the sciatic nerve (CCI) neuropathic pain model. We compared the effectiveness of a panel of analgesic drugs (i.e., morphine, valdecoxib, gabapentin), and cannabinergic drugs and a non-analgesic (i.e, diazepam) in reversing CCI-induced decreases in marble burying versus reversing CCI-induced increases in mechanical allodynia and thermal hyperalgesia. Each of the analgesics dose-responsively reversed CCI-induced depression of marble burying as well as CCI-induced allodynia and thermal hyperalgesia. In contrast, diazepam further reduced marble burying, but did not affect pain-stimulated measures. The present results indicate that marble burying represents a simple assay to assess pain-depressed behavior.

α4 nAChRs AND SEPTUM ERK SIGNALING REGULATE AGE-ASSOCIATED CHANGES IN ANXIETY-LIKE BEHAVIOR. Claire I. Dixon[1], Shawn M. Anderson[1],

Alexandra M. Stafford[1], Petra Scholze[2] & Darlene H. Brunzell[1], [1]Dept. of Pharmacology & Toxicology, Virginia Commonwealth University, Richmond VA 23298, United States & [2]Medizinische Universität Wien, Zentrum für Hirnforschung, A-1090 Wien, Austria. Pharmacological inhibition of α4β2 subunit containing nicotinic acetylcholine receptors (α4β2*nAChRs) reduces anxiety-like behavior in rodents. As expression of α4β2*nAChRs declines with age, the present study assessed how reduced expression of these nAChRs during adulthood (ADULT; 6-8 mo.) and aging (AGED; 22-24 mo.) affects anxiety behavior in wildtype (WT) and α4 subunit null mutation heterozygous (α4HET) mice. AGED and α4HET ADULT mice showed reduced anxiety-like behavior, whereas AGED α4HET mice showed increased anxiety-like behavior. Antagonism of α4β2*nAChRs with dihydro-beta-erythroidine (DHβE) increased anxiety-like behavior in ADULT α4HETs. Western blot analysis revealed that AGED WT and ADULT α4HET mice showed elevated levels of septal pERK that were inversely correlated with anxiety behavior. These findings suggest that reduced expression of α4β2*nAChRs that may occur with normal aging supports anxiolysis-like phenotype but that inhibition of this pool of receptors may increase anxiety-like behavior in individuals that have a poverty of α4β2*nAChR expression.

ACTIVATION OF a6*NACHRS IS SUFFICIENT FOR NICOTINE REWARD IN MICE: PUTATIVE INVOLVEMENT OF THE NUCLEUS ACCUMBENS SHELL. Alexandra M. Stafford & Darlene H. Brunzell, Department of Pharmacology & Toxicology, Virginia Commonwealth University, Richmond, VA 23298. Nicotine, a primary addictive component in tobacco, binds to nicotinic acetylcholine receptors (nAChRs), producing its rewarding effects. Rodent studies have shown that activation of β2*nAChRs (*denotes assembly with other subunits) promotes nicotine reward. However, less is known regarding the subunit make-up of β2*nAChRs that regulate reward. β2 primarily assembles with the a4 and L97\f"Symbol"\s106 subunits. a4β2*nAChRs are ubiquitously expressed throughout the brain, while a6β2*nAChRs are selectively expressed in catecholaminergic nuclei such as the ventral tegmental area and projection terminals in the nucleus accumbens (NAc), which are implicated in nicotine's rewarding properties. The goal of this study was to investigate the contribution of α6β2*nAChRs to nicotine reward. We showed that a6β2*nAChR gain-of-function (a6L9S) mice, whose α6β2*nAChRs are hypersensitive to nicotine, exhibit enhanced nicotine conditioned place preference (CPP) compared to wild-type (WT) mice, suggesting that activation of α6β2*nAChRs is sufficient to promote nicotine reward. Further, we showed that antagonism of α6β2*nAChRs in the NAc shell with α-conotoxin MII blocked nicotine CPP in WT mice. Overall, these studies suggest that activation of α6β2*nAChRs on terminals in the NAc shell promotes nicotine reward.

Posters

LEPTIN EXPRESSION IN ADIPOSE AND SKELETAL MUSCLE OF MORBIDLY OBESE PCOS AND NON-PCOS WOMEN. M-A Orciga[1], K. Yao[1], Z. Younoszai[1],

A. Baranova[1,2], Z. Younassi[1,3] & A. Birerdinc[2,3], [1]Betty & Guy Beatty Center for Integrated Research, Falls Church, VA 22042, [2]Center for the Study of Chronic Metabolic Diseases, Falls Church, VA 22042 & [3]GMU-Inova Translational Research Institute, Fairfax, VA 22030. Polycystic ovarian syndrome (PCOS) is one of the most common reproductive disorders of the modern world afflicting an estimated 10% of women of reproductive age. An estimated 30-40% of women with PCOS are obese, and 70% of all PCOS cases are found to have some level of insulin resistance (IR) regardless of weight. Understanding IR and the role it plays in pathogenesis of PCOS is inherent in improving treatment and diagnosis of this disorder. To evaluate IR, we examined the role of leptin, a key insulin modulator mainly produced by adipocytes, in morbidly obese, BMI-matched patients with PCOS (n=16) and those without PCOS (n=17). There was a non-statistical trend of higher leptin expression in non-PCOS (NP) patients than in PCOS (P) patients ($p>0.05$). However, in the non-PCOS, non-diabetic (NPND) group a statistically significant 26-fold increase ($p<0.05$) in gene expression of leptin is seen as compared to the non-PCOS diabetics (NPD), with this trend disappearing in the PCOS group. These results suggest that leptin expression is affected by PCOS state when comparing diabetics versus non-diabetics, which should be further evaluated in future studies.

TOLERANCE OF PROXIMAL THYMINE GLYCOL IN DNA DOUBLE-STRAND BREAK REPAIR BY NONHOMOLOGOUS END JOINING. D. Bafail, S. L. Chalasani, M. Almohaini, L.F. Povirk, & K. Akopiants, Department of Pharmacology & Toxicology & Massey Cancer Center, Virginia Commonwealth University, Richmond VA 23298. DNA double strand breaks (DSBs) are extremely toxic to cells because they can lead to genomic rearrangements and even cell death. Two main pathways can repair DSBs: the homologous recombination repair (HRR) pathway and the non-homologous end-joining (NHEJ) pathway. NHEJ is the primary pathway overall and predominant pathway in G1. In the present study NHEJ was assessed using substrates with complex DSB, specifically DSB accompanied by thymine glycol (Tg) at the end (Tg1), 2nd (Tg2) 3rd (Tg3) and 5th (Tg5) positions from the broken end. Experiments were done using XRCC4-like factor (XLF)-deficient cell extracts, with or without the addition of XLF and/or Artemis, EndoIII and ddTTP, or using purified NHEJ protein Ku and XRCC4/Ligase IV with or without XLF. Our results indicated that cell extract could ligate the plasmid with Tg located at third base and fifth the DSB with high efficiency but ligation was severely inhibited when Tg was located at first or the second position from DSB ends. The joining of the Tg5 plasmid was lower than Tg3 with higher efficiency of base excision repair. On the other hand, end joining by the purified proteins was proportional to the thymine glycol position from the DSB end. While XLF was required for ligation of Tg1 and Tg2 by the purified proteins, there was some repair in Tg3 and Tg5 substrates even in its absence. The extracts did not show ligation of the substrates in the absence of XLF.

CAFETERIA STYLE DIET IMPAIRS MEMORY AND INCREASES RISK OF METABOLIC SYNDROME. M. L. Knabe, C. Curtiss, K. S. Sarfert & S. N. Blythe, Dept. of Biology, Washington & Lee University, Lexington VA 24450. Increasing evidence suggests that excess energy intake and obesity are associated with cognitive dysfunction. Although many studies utilize a Western-style diet to examine the effects of diet-induced obesity on cognitive impairment, we believe the Cafeteria-style diet (CSD) more accurately represents the varied and energy-dense diet that contributes to hyperphagia and obesity in humans. This study aimed to investigate the physiological and behavioral effects of CSD consumption on juvenile rats. Twenty-two male Sprague-Dawley rats were divided into control (n=11) and CSD (n=11) groups from weaning. Diet exposure continued for 10 weeks, during which all animals were given ad libitum access to standard rat chow and water. The CSD animals were offered two alternating energy-dense diets, which included various cookies, chips, processed meats, and sweet drinks. During diet exposure, the rats were subjected to Novel Object Recognition, Novel Place, Novel Context, and Morris Water Maze tasks to assess hippocampal-dependent memory performance, as well as an open field task to assess hyperactivity. Following sacrifice, fat pads, livers, aortas, and brains were excised for post-mortem studies. Cognitive tests revealed that CSD rats had compromised spatial and episodic memory. Tissue analysis demonstrated that CSD rats were composed of more fat than control rats, despite comparable total body weight. These data suggest that exposure to energy-dense CSDs leads to memory impairment and the replacement of lean muscle with fatty visceral tissue, potentially contributing to the development of metabolic syndrome and neurological disorders.

AUGMENTATION OF T CELL FUNCTION AS A SELECTION CRITERIA FOR CHEMOTHERAPEUTIC STRATEGIES IN TREATMENT OF MELANOMA. Se W. Jeong, Dan Gioeli, Michael Weber, Alex McClanahan, Devin Roller & Timothy N.J. Bullock, Department of Pathology, University of Virginia, Charlottesville, VA 22908. Metastatic melanoma has a very poor prognosis. PLX4032 (Vemurafenib) is a specific inhibitor that targets a B-Raf gene mutation that is present in more than half of the melanoma patients. Unfortunately, the tumors escaped the therapy by activating other signaling pathways. To address this adaptive resistance, synthetic lethality screens were used to identify small molecule inhibitors (SMI) that synergized with PLX4720, in a panel of melanoma cell lines. As recent studies have indicated that the effectiveness of numerous chemotherapies is dependent upon the recruitment of an adaptive immune response, we probed how these combinations of chemotherapies impacted T cell function and cytokine expression. Two of six inhibitors were found to be overtly detrimental to T cell responses. In contrast, three inhibitors were found to augment varying aspects of T cell responses (frequency and function) and promoted function of T cells from human tumor samples. The activity of the SMI correlates with the expression of checkpoint molecules such as TIM3 and LAG3, but not PD1. Our data indicate that the immunomodulatory capacity of SMIs should be screened as a selection

criterion for single agent therapy and as a component of combination immunotherapies, and suggest opportunities for synergistic chemo-immunotherapeutic clinical trials.

OBESITY AND ADIPOKINE SIGNALING IN CHILDREN AND ADOLESCENTS: THE EFFECTS OF CHRONIC INFLAMMATORY SIGNALING IN OVERALL HEALTH AND GROWTH. Tooba Khan[1], Ancha Baranova[2] & Aybike Birerdinc[2], [1]Dept. of Biology, George Mason Univ., & [2]School of Systems Biology, College of Science, George Mason Univ., Fairfax VA. 22030. NAFLD is one of the most widely increasing diseases in the world today and it is increasing rapidly in children as young as 3 years old. There are many different symptoms of NAFLD but obesity appears to be the most common amongst all. The purpose of this research was to study the prevalence of obesity in children. PubMed was used for this research and the terms used were: 'pediatric NAFLD,' 'Obesity', 'NASH', 'metabolic syndrome' 'and 'adipokine signaling', 'obesity and pregnancy', 'obesity and depression', and 'obesity and mental disorders'. The search was limited to the past 10 years and excluded animal studies. The search resulted in 447 articles out of which 59 were used. In 2007, the International Diabetes Federation defined NAFLD in children aged 6-10 years who were obese and had family histories of cardio metabolic diseases and children aged 10-16 years who were obese and met the criteria for triglycerides being >150 mg/dL. Obese children with symptoms of NAFLD who turn into obese adults tend to have more chances of developing T2DM, hypertension, and atherosclerotic cardiovascular disease. In the US, 17% of the children have type I NASH while 51% have type II NASH and 16% have simple steatosis.8% of the children have advanced fibrosis while 3% have liver cirrhosis. Generally, 85% of the obese children in the US tend to turn into obese adults.

PROFILING FIBROTIC GENES IN VISCERAL ADIPOSE TISSUE (VAT) AMONG PATIENTS WITHIN THE SPECTRUM OF NAFLD. Kameron Tavakolian, Ancha Baranova, Zobair Younossi & Aybike Birerdinc, School of Systems Biology, College of Science, George Mason University and Fairfax VA 22030 & the Beatty Liver & Obesity Research Program, Falls Church VA 22042. Non-alcoholic fatty liver disease (NAFLD) occurs when fat is deposited in the liver, not as a result of alcohol consumption. NAFLD is a spectrum of diseases ranging from relatively benign fatty liver, to non-alcoholic steatohepatitis (NASH). While studies have suggested a role for proinflammatory and profibrotic in the development of NAFLD and NASH, very few of the aforementioned studies have analyzed the expression of genes encoding these profibrotic with varying degrees of hepatic fibrosis (under the NAFLD spectrum) in the visceral adipose tissue (VAT). The aim of this study is to select and validate a panel of biomarkers of inflammation and fibrosis and correlate their gene expression in VAT with the degree of severity of NASH. Primer design and validation is often an underappreciated step in projects involving qPCR, particularly in studies when it is unclear if said genes are indeed expressed. The first major result of this study was validation of the designed primers (n=8) for genes related to both the fibrotic signaling

process and inflammation. IL13RA2 and COL1A2 were found to amplify the intended target products (gel electrophoresis confirmed). CCL11, AGT, and CSF1 show perfect melt curves, however subsequent gel electrophoresis shows amplification of multiple products indicating non-specific binding or the presence of different versions of the genes. IL5, CSF2, and CSF3 have shown abnormal melt curves, again suggesting either differential expression of gene isotypes.

Natural History and Biodiversity

RESPONSES OF THE CATALPA SPHINX AND ITS PRIMARY PARASITOID TO HIGH AND LOW LEVELS OF IRIDOID GLYCOSIDES. Jessica L. Bray[1], M. Deane Bowers[2] & Karen Kester[1], [1]Dept. of Biology, Virginia Commonwealth University, Richmond VA 23298 & [2]Dept. of Ecology & Evolutionary Biology, University of Colorado, Boulder CO. Caterpillars of the catalpa sphinx, Ceratomia catalpae, feed exclusively on Catalpa, which contains the iridoid glycosides catalpol and catalposide. Many catalpa trees are heavily infested and defoliated by catalpa caterpillars each year whereas others are untouched. Most populations of catalpa sphinx caterpillars are heavily parasitized by a parasitic wasp, Cotesia congregata, but some populations remain unparasitized. We hypothesized that iridoid glycoside levels would vary among trees and that insect responses to relatively high or low levels of these chemicals could explain these patterns of herbivory and parasitism. Iridoid glycoside levels varied among trees and trees with relatively high levels of iridoid glycosides were more heavily defoliated. Catalpa moths preferred to oviposit on trees with high iridoid glycoside levels in choice oviposition assays. Caterpillars displayed no preference for high or low iridoid glycoside leaf discs in choice feeding assays. Searching times of C. congregata on high and low iridoid glycoside leaf discs did not differ in no choice searching assays. Results indicate that observed variability in herbivory among trees can be explained by moth oviposition preferences for trees with relatively high levels of iridoid glycosides.

BEHAVIORAL RESPONSES OF MALE PARASITIC WASPS TO PLANT CUES WITH RESPECT TO HOST-PLANT COMPLEX ORIGIN. Megan Ayers & Karen Kester, Dept. of Biolology, Virginia Commonwealth University, Richmond VA, 23284. The role of plant cues in host location by female parasitic wasps has been well studied; however, very little is known about the use of plant cues by males in mate location. Female *Cotesia congregata* display inherent responses to plant cues that can be modified by post-emergence experience. We hypothesized that males would exhibit similar inherent and modifiable responses to plant cues. Further, we hypothesized that males originating from one of two host-plant complexes, *Manduca sexta* on tobacco or *Ceratomia catalpae* on catalpa, would display different responses to tobacco or catalpa. In no-choice assays, searching responses of both males and females to a non-host plant increased sharply at Day 2 and remained stable through Day 4. In no-choice assays