Medical Sciences

AUTOLOGOUS PLATELET GEL: A NOVEL TREATMENT FOR MYOCARDIAL INFARCTIONS (HEART ATTACK). C. W. Gurnee[1,2], B.Y. Hargrave[1,2], S.J. Beebe.[1] & X. Shu[1], [1]Frank Reidy Research Center for Bioelectrics, Norfolk VA 23510 and [2]Old Dominion University, Norfolk VA 23529. Autologous Platelet Rich Leukocyte Plasma (PRLP) or "platelet gel" as it is sometimes called is a platelet/leukocyte rich concentrate made from the whole blood of a patient. PRLP, when applied to soft tissue wounds, enhances healing by placing a high concentration of growth factors (released from platelets activated by a known platelet activator) at the site of damage. We examined PRLP in the rabbit heart after Acute Myocardial Infarction (AMI) and reperfusion and tested its ability to support and/or improve mechanical left ventricular function. *In Vitro* study: In the rabbit Langendorff heart treated with PRLP (injected into the myocardium of the left ventricle) and exposed to global ischemia there was a shift in systolic and diastolic pressure curves suggesting less systolic and diastolic dysfunction compared to the saline treated controls. The PRLP treated but not the saline treated hearts were capable of increasing left ventricular work function to a level above baseline after 40 min of reperfusion. *In Vivo* study: Fourteen days after an AMI, the rabbit heart treated with PRLP (injected into the myocardium of the left ventricle) at the time of the infarct was stressed with dobutamine and was capable of increasing left ventricular positive dP/d*t* and decreasing negative dP/dt compared to the saline treated heart. PRLP supports mechanical left ventricular function in the rabbit heart following AMI. These preliminary data suggest that PRLP, injected into the myocardium may function to regulate left ventricular pressures and improve function following AMI.

GENE EXPRESSION OF *SACCHAROMYCES CEREVISIAE* EXPOSED TO COMMERCIAL WOOD PRESERVATIVES BY DNA MICROARRAY ANALYSIS AND RT-PCR. Madison M. Stevens & Consuelo J. Alvarez, Dept. of Biol. and Environ. Sciences, Longwood Univ., Farmville VA 23909. Creosote and pentachlorophenol (PtCP) are commercial wood preservatives regulated by the EPA because of their toxicity to wildlife and their possible role as human carcinogens. This baker's yeast was used as a model system to observe changes in gene expression after exposure to these compounds. Cells were exposed to a creosote concentration of 50ng/ml and to a PtCP concentration of 50μM. A total of 20 DNA microarray chips were tested (7 creosote chips, 7 PtCP chips, and 6 solvent chips (used as controls)). 27 genes from creosote and 180 genes from PtCP were found to have significant changes in expression and among them, 15 genes' RNAs were chosen for reverse transcription and RT-PCR to validate their change in expression. In both experimental treatments, genes with roles in cell cycle regulation, drug transport, and response to stress had significant changes in expression. Clustering analysis revealed highly correlated gene expression in genes associated with mitotic controls. Because creosote and PtCP have been indirectly linked to causing cancer in humans, BLASTn and BLASTp analysis on the National Center for Biotechnology Information (NCBI) website was used and confirmed that some genes with significant changes in expression had homology to human genes and protein sequences. Overall, the results of this study are a sign of the necessity for more studies to be done by the EPA and workers' health associations in

order to establish job/health regulations and it could be a starting point for R-1 institutions that concentrate their research in cancer studies.

MBD2 REGULATED CANDIDATE GENES FOR MODULATION OF HUMAN GAMMA GLOBIN GENE EXPRESSION IN ADULT ERYTHROID CELLS. Merlin N. Gnanapragasam[1], Jeremy W. Rupon, Shou Zhen Wang[4], Latasha C. Redmond[1], Omar Y. Mian[3], Catherine I. Dumur, C.I[5], Kelly J. Archer[5], Joyce A. Lloyd[1] & Gordon D. Ginder[1,2,4] Departments of [1]Human and Molecular Genetics, [2]Internal Medicine, [3]Microbiology and Immunology, [5]Pathology, [5]Biostatistics, [4]Massey Cancer Center, Virginia Commonwealth University, Richmond, VA 23298. Reexpression of the silenced fetal γ -globin gene in adult erythrocytes of individuals with β-globin disorders such as sickle cell anemia and β-thalassemia, is of therapeutic interest due to its ameliorating effects[1-5] . We have previously shown that knock out of methyl CpG binding domain protein 2 (MBD2) in transgenic mice carrying the human beta globin gene cluster (β-YAC mice), results in de-repression of γ globin gene expression in adult erythrocytes. However, MBD2 does not directly bind to the γ-globin gene to mediate its silencing. We hypothesize that MBD2 may suppress human γ globin gene transcription in adult erythrocytes by an indirect mechanism ie., by binding to and repressing transcription of intermediary gene/s which may be involved in γ globin gene regulation. Microarray assays were performed on Affymetrix GeneChip® 430A 2.0 array for protein coding genes using RNA from four MBD2-/- and wild type mice adult erythroid cells. Growth factor receptor bound protein 2-associated protein1 (GAB1) and Zinc finger and BTB domain containing 32 (ZBTB32) were identified as priority candidate genes. Functional studies indicate that overexpression of these validated candidate genes can cause reexpression of γ-globin gene in Chemical Inducer of Dimerization dependent β-YAC mouse adult bone marrow cells.

THE PREVENTION OF EPILEPTOGENESIS THROUGH CALCIUM MODULATION IN A HIPPOCAMPAL NEURONAL CULTURE MODEL OF STATUS EPILEPTICUS-INDUCED ACQUIRED EPILEPSY. Nisha Nagarkatti, Laxmikant S. Deshpande & Robert J. DeLorenzo, Dept. of Neurology, VA Commonwealth Univ, Richmond VA 23298. Currently, no treatment exists to prevent the development of acquired epilepsy (AE) following injury such as status epilepticus (SE). In this study, the ability of a new drug, carisbamate, to prevent the development of spontaneous recurrent epileptiform discharges (SREDs) and alter intra-neuronal Ca^{2+} levels ($[Ca^{2+}]_i$) following in vitro SE was evaluated. After treatment in low-Mg^{2+} containing solution to mimic SE, neuronal cultures developed SREDs (in vitro AE). Cultures were treated, post-SE, with carisbamate (200 μM) for 12 h. The drug was then washed out of the system and neurons were evaluated for the expression of SREDs 24 h post-washout. Drug-treated neurons failed to display SREDs, in contrast to the controls. The ability of carisbamate to block elevations in $[Ca^{2+}]_i$ after SE was also tested because alterations in Ca^{2+} dynamics and homeostatic mechanisms have been associated with plasticity changes and ultimately, the development of epilepsy. Following SE, sustained elevations in $[Ca^{2+}]_i$ were observed and carisbamate was able to lower $[Ca^{2+}]_i$ when administered post-SE. When evaluated for the ability to restore Ca^{2+} homeostasis following glutamate challenge, the drug-treated neurons showed

enhanced recovery. This study suggested that carisbamate is able to prevent the development of SREDs in vitro; furthermore, the ability of carisbamate to alter Ca^{2+} dynamics may contribute to its anti-epileptogenic properties. Supported by: NIH grants RO1NS051505, RO1NS052529, UO1NS058213 and AHA Pre-doctoral fellowship.

MODELING PHYSICAL ACTIVITY IN WORKING ADULTS: HOW SUITABLE IS THE EXPANDED PARALLEL PROCESS MODEL?. A. B-H-Sam [1], M. L. Walker [2], S. Plichta [1], and G. Maihafer [2]; [1]School of Community and Environmental Health, College of Health Sciences, Old Dominion University and [2]School of Physical Therapy, College of Health Sciences, Old Dominion University. The usefulness of the Expanded Parallel Process Model in predicting health enhancing physical activity is assessed in the context of risk for coronary heart disease. The study involves secondary analyses of a dataset from a group of working adults who elected first to participate in a health plan 'Quality Improvement Study' and were then randomly selected to receive an intervention program designed to increase activity. Data on self-reported demographics, physical activity levels, health status characteristics and perceptions measured on a Likert-type scale known as the Risk Behavior Diagnosis Scale are analyzed. The Risk Behavior Diagnosis Scale measures represent the model hypothesized mediating variables which are perceived severity, perceived susceptibility, perceived response efficacy and perceived self-efficacy. Results of data analyses offer limited and weak support for use of the Expanded Parallel Process Model to explain differences in health enhancing physical activity behavior of working adults. The magnitude of the hypothesized Expanded Parallel Process Model mediator variables observed in the study, though small, may suffice as a call for further research using a different research approach (longitudinal). Health behavior is complex, and the most important determining factors of physical activity may also not have been included in the analysis. A different theoretical model, in this case, may help to explain physical activity behavior.

PATTERNS OF ETHANOL-RESPONSIVE GENE EXPRESSION IN FYN KINASE KNOCKOUT MICE. Sean P. Farris & Michael F. Miles, Dept. of Pharmacology & Toxicology, Virginia Commonwealth University, Richmond, VA 23298. The molecular mechanisms underlying alcoholism are largely unknown, however, changes in gene expression are proposed as critical molecular adaptations leading to the development lasting ethanol related phenotypes. Two inbred mouse strains with divergent ethanol related behaviors, DBA/2J (D2) and C57BL/6J (B6), also exhibit differing basal and acute ethanol-responsive gene expression patterns among discrete brain regions including prefrontal cortex (PFC). Bioinformatic analysis of D2 and B6 microarray studies implicated Fyn kinase as a potential mechanism regulating ethanol-responsive myelin gene expression. *Fyn* knockout (KO) mice have abnormal CNS myelination and are more sensitive to ethanol related behaviors. Using DNA microarrays we assayed *Fyn* KO mouse PFC to identify downstream basal and acute ethanol-responsive gene expression patterns. Characterizing the associated gene networks will test the hypothesis that Fyn is required for acute ethanol regulation of myelin gene networks, and identify novel *Fyn* related signaling mechanisms. Microarray analysis revealed 565

genes altered by genotype, and 746 genes altered by ethanol ($P<0.001$). Several genes with a functional relationship to myelin including progesterone receptor (*Pgr*) were regulated by ethanol in Fyn KO PFC, suggesting a novel relationship may exist between Fyn and Pgr in regulating ethanol-responsive myelin gene expression. Continued investigation of this functional relationship and associated gene networks may aid in the future development of more successful pharmacotherapies for alcoholism and related CNS myelin toxicity.

NANOSECOND PULSED ELECTRIC FIELDS INDUCE APOPTOTIC-LIKE CELL DEATH IN MURINE E4 SQUAMOUS CARCINOMA CELLS BY MULTIPLE MECHANISMS. Wei Ren & Stephen J. Beebe, Frank Reidy Research Center for Bioelectrics, Old Dominion University, Norfolk VA 23510. Nanosecond pulsed electric fields (nsPEFs) are pulses with ultra-short duration (ns), high power (mega watts), but low energy density (mJ/cc), which are distinctly different from conventional-electroporation pulses. NsPEFs have emerged as a novel method to modulate intracellular structures and functions. To determine the signaling pathways in nsPEF-induced cell death, murine E4 squamous carcinoma cells were exposed to multiple pulses from 0 to 60kV/cm with 300ns duration. Cell death occurred with decreases in the mitochondria membrane potential, the appearance of active caspases, and the release of cytochrome *c* from the mitochondria into the cytoplasm. Using a cell permeable, irreversible inhibitor, the appearance of active caspases was observed within1 hour post pulse. At lower electric fields, active caspases appeared in the apparent absence of cytochrome *c* release. However, at higher electric fields cytochrome *c* release was observed. Using irreversible inhibitors of active caspases, active caspase-8, caspase-9 as well as caspase 3/7 were seen within 2 hours post pulse in an electric-field dependent manner. In addition, inhibition of caspase activity using z-VAD-fmk partially attenuated cytochrome *c* release. These results suggest that nsPEFs induce an apoptotic-like cell death as indicated by using both mitochondria-dependent and –independent mechanisms in E4 cells.

METHYL BINDING DOMAIN PROTIEN 2 (MBD2) MAINTAINS TUMOR SUPPRESSOR SILENCING AND PROMOTES EPITHELIAL DEDIFFERENTIATION IN BREAST CANCER. O.Y. Mian, M. N. Gnanapragasam, S. Z. Wang & G. D. Ginder, Massey Cancer Center, Richmond, VA 23298. Methyl-CpG Binding Proteins (MCBPs) function as interpreters of epigenetic signals encoded in the genome. We study the function of Methyl-Binding Domain Proteins (MBDs) in human mammary epithelial cancers, where repatterning of CpG methylation is common. We find Methyl Binding Domain Protein 2 (MBD2) promotes abnormal multi-cellular morphology of tumor cells grown in extracellular matrix extracts. Stable MBD2 knockdown in MCF7 cells leads to an increased proportion of differentiated epithelial structures (e.g. acinii, 70%, [CI=0.55-0.83]) when compared with untransfected (46%, [CI=0.39-0.53], $p \leq 0.038$) and scrambled shRNA transfected (37%, [CI=0.29-0.45], $p \leq 0.012$) control cells. To identify the genes underlying this MBD2 dependant phenotype, high throughput quantitative PCR data were probed using self organizing map (SOM) analysis. We found a small subset of the breast cancer specific

tumor suppressors known to be silenced by promoter hypermethylation were regulated by MBD2 (n=7, 15%). The MBD2 dependant genes were rapidly re-suppressed upon rescue with a shRNA binding site variant MBD2 and ChIP studies confirmed binding of MBD2 at genes within the MBD2 dependant cluster. We demonstrate MBD2 maintains aberrant dedifferentiation in breast cancer through a network of epigenetically inactivated tumor suppressor genes. Based on these findings, we intimate a pathologic role for MBD2 in the initiation and progression of human mammary epithelial neoplasia. This work was supported by NIH-2R01DK029902-26A2

NEUTROPHIL INFILTRATION AND RELEASE OF REACTIVE OXYGEN SPECIES ENHANCE VASCULAR REACTIVITY TO ANGIOTENSIN II VIA THE RhoA KINASE PATHWAY. Nikita Mishra, MD, Scott Walsh, PhD, OB/GYN/Physiology, VCU, Richmond,VA 23298. Women with preeclampsia have enhanced vascular reactivity to Angiotensin II (Ang II). We hypothesized that neutrophil release of ROS enhances vessel reactivity to Ang II by activating the RhoA kinase pathway. Resistance arteries from omental fat biopsies of normal pregnant patients undergoing C-sections (n=20) were studied. Ang II dose response (10^{-9}M to 10^{-5}M) was done. The Ang II dose response was repeated with neutrophils ($2000/mm^{3)}$ in the vessel lumen. Ang II + neutrophils was repeated with addition of superoxide dismutase (150 U/ml)/catalase (5000 U/ml) to quench ROS or with addition of Y-27632 (3μM), a specific Rho A kinase inhibitor. Ang II dose response was also tested with ROS generating solution (hypoxanthine, 0.36mM + xanthine oxidase, 0.004 IU/ml), Ang II + ROS + SOD/catalase or Ang II + ROS + Y-27632 (3μM). Ang II caused a dose response contraction with maximum response at 10^{-6}M (Change in diameter of -18.6 ± 2.0m, mean ± S.E). With activated neutrophils, vasoconstrictive response to Ang II was significantly greater at 10^{-9}M, 10^{-7}M, 10^{-6}M and 10^{-5}M (-44.5 ± 5.9m, $p<0.01$). SOD/catalase and Y-27632 significantly blocked the enhanced response to Ang II by activated neutrophils. Similar results were observed with ROS. These results suggest that neutrophil infiltration in systemic vasculature of preeclamptic women explain the enhanced reactivity to Ang II in preeclampsia. HL069851.

POSITIVE ALLOSTERIC MODULATION OF α4β2 NEURONAL nAChRs BY DESFORMYLFLUSTRA-BROMINE AND ITS ANALOGS. N. A. German[1], J.-S. Kim[1], A. Pandya[2], M. Schulte[2] & R. A. Glennon[1], [1]Department of Medicinal Chemistry, Virginia Commonwealth University, Richmond VA 23298 and [2]Department of Chemistry and Biochemistry, University of Alaska, Fairbanks AK 99775. Neuronal nicotinic acetylcholine receptors (nAChRs) appear to play an important role in cognitive and attentive processes. An alteration in expression or function of α4β2 nAChRs also has been associated with neurodegenerative disorders and in certain aspects of drug abuse. Positive allosteric modulators (PAMs) have been envisioned as a potential therapeutic treatment; however there is a very limited number of compounds, if any, that have shown selective allosteric modulation of α4β2 nAChRs. A novel indole alkaloid desformylflustrabromine (dFBr), recently isolated from a marine species, has been shown to selectively modulate α4β2 nAChRs through an

allosteric mechanism. The present investigation was designed to determine structural features important for the actions of dFBr. Proposed compounds were prepared using several synthetic schemes. Biological activities of synthesized compounds were evaluated using two-electrode voltage clamp techniques employing *Xenopus laevis* oocytes injected with cDNAs of the human α4β2 receptor. As a result of this study dFBr and its synthetic analogs were shown to be positive allosteric modulators of α4β2 nAChRs and key structural features were identified for this action. [Supported, in part, by a Virginia Center on Aging grant and a VCU Department of Pharmacology and Toxicology Training Grant (T32 DA007027-34).]

THE EFFECT OF SUCRALOSE ON OBESITY AND DIABETES PROGRESSION IN A TYPE II DIABETES MODEL, THE TALLYHO/JNGJ MOUSE. Matthew C. Johnson & Dianne M. Baker, Dept. of Bio. Sci., Univ. Mary Washington, Fredericksburg, VA 22401. The high incidence of type II diabetes is one of the most pressing medical concerns in the United States. Treatment of type II diabetes commonly includes management of blood glucose and body weight through diet and exercise. To regulate blood glucose, type II diabetics often replace dietary sugars with artificial sweeteners such as sucralose (Splenda®). While sucralose is typically considered to be a safe replacement for sugar, some recent studies have found adverse effects on glucose balance and food consumption. In this study, we tested the effects of sucralose on the progression of type II diabetes in mice. We hypothesized that the presence of sucralose in the diet of type II diabetic mice would accelerate the progression of the disease, resulting in increased glucose levels compared to diabetic control animals. Secondly, we hypothesized that sucralose in the diet would increase food consumption and therefore increase body mass in type II diabetic mice compared to control-fed type II diabetic animals. To test these hypotheses, we measured food intake, body mass, and blood glucose levels (both fasting and glucose tolerance test levels) in sucralose-fed and control-fed animals over a 10 week treatment period. Plasma samples were also collected over this same period for measurement of insulin and triglyceride levels. Contrary to expectations, sucralose-fed mice had lower blood glucose and triglyceride levels than control-fed mice. Additionally, we found no significant effect of sucralose on insulin concentration, food intake or body mass. These results suggest that sucralose may slow the progression of type II diabetes.

THE EFFECTS OF Δ^9-TETRAHYDROCANNIBINOL, THE MAJOR PSYCHOACTIVE COMPONENT OF MARIJUANA, ON FOOD AND BRAIN REWARD. M. A. Rolfes[1,2], A. J. Kwilasz[2], L. S. Harris[2], R. E. Vann[1,2], Departments of [1]Psychology, & [2]Pharmacology/Toxicology, VCU, Richmond, VA, 23298. Previous studies in rats using progressive ratio (PR) schedules of reinforcement have suggested a role for cannabinoid receptor 1 (CB1) agonists in both food consumption and feeding motivation. PR procedures that assess motivation commonly use food reinforcement, however these procedures are unable to delineate whether motivation to respond is an enhancement of feeding or reward mechanisms. Intracranial self-stimulation (ICSS), a procedure in which animals are trained to respond for stimulation of the medial forebrain bundle, is well suited to investigate motivation to respond for reward. Accordingly, the CB1 agonist, Δ^9-tetrahydrocannabinol (THC), was assessed for its

ability to alter motivation to respond for food or brain stimulation reward (BSR). ICR mice were trained to respond for food or BSR (158 Hz, 150 μA) on a PR2 schedule of reinforcement, in which the response requirement increased by 2 lever presses after every 4 reinforcers earned. Breakpoints were assessed daily to measure motivation to respond for reinforcement and tests with vehicle and THC (1, 3, 10, and 17.1 mg/kg) were conducted. THC administration significantly increased breakpoints for food and BSR; however, breakpoint increases for BSR were observed at a higher dose than for food. Operant response rates were unaffected by THC. These results add to a growing body of literature that suggests an enhancing role for CB1 agonists in feeding and reward motivated behavior in mice.

THE DOMESTIC FOWL (GALLUS) AS A MODEL OF OBESITY AND SEX SPECIFICITY IN THE METABOLIC SYNDROME. R.P. Wyeth[1], A. Santo[1], K.E. Harris[2], T.V. Palacios[1], R.M. Lewis[3], C.F. Honaker[3] & P.B. Siegel[3], [1]Edward Via, Virginia College of Osteopathic Medicine, [2]Dept. of Human Nutrition, Foods and Exercise, [3]Dept. of Animal and Poultry Sciences, Virginia Polytechnic Institute and State University, Blacksburg. High body fat content is associated with increased morbidity and mortality. The prevalence of overweight or obese humans grows with alarming rapidity. Globally 1.7 billion people are overweight or obese producing a pandemic of cardiovascular disease (CVD). Principal risks for CVD include abdominal obesity, hyperlipidemia, dyslipidemia, hyperglycemia, insulin resistance, and hypertension, known collectively as the metabolic syndrome. The ability to define mechanisms that interact to produce the metabolic syndrome in humans is limited by lack of a single mammalian model that fulfills all criteria for this syndrome. We propose a suitable alternative. A line of chickens developed at Virginia Tech was selected for high juvenile body weight that not only exhibits rapid juvenile growth but also becomes morbidly obese unless feed restricted. Thus, these high weight chickens provide an attractive model for studying CVD associated with the metabolic syndrome. Based on the metabolic characteristics and preliminary data on these high weight chickens, by inducing obesity, we produced a sexually differentiating effect in the α_1 adrenergic response of this proposed non-mammalian model of the metabolic syndrome. This study was funded by a grant from the Harvey Peters Foundation.

THE FLAVONOID LUTEOLIN INCREASES VASODILATATION THROUGH NON-GENOMIC NITRIC OXIDE RELEASE. Hongwei Si[1], Dongmin Liu[1] & Richard P. Wyeth[2], [1]Laboratory of Molecular Nutrition, Department of Human Nutrition, Foods and Exercise, College of Agriculture and Life Sciences, Virginia Polytechnic Institute and State University, Blacksburg, Virginia, [2]Divisions of Anatomy and Physiology, Edward Via Virginia College of Osteopathic Medicine, Blacksburg, Virginia. Luteolin is a plant flavonoid and vasodilator. We tested if luteolin will stimulate endothelial NO synthase (eNOS) phosphorylation and decrease alpha$_1$ adrenergic (α_1AR) contraction, in cultured human aortic endothelial cells (HAECs) and isolated rat aorta. When intact rat aortic rings were constricted with the specific α_1AR agonist phenylephrine, followed by cumulative addition of luteolin, a dose dependent relaxation was produced. In rings pretreated with the eNOS inhibitor, N-nitro-L-arginine methyl ester, luteolin–induced vasorelaxation was partially blocked. When HAECs were incubated

with luteolin, luteolin phosphorylated eNOS and stimulated NO release. These data indicate that, within arterial vasculature, luteolin: 1) induces vasorelaxation through an endothelium dependent mechanism; 2) produces a dose dependent and immediate decrease in α_1AR induced contraction; 3) increases the phosphorylation of eNOS and subsequently improves NO production. Taken together, we suggest that luteolin induces vasorelaxation not by increased eNOS translation but rather by receptor-mediated stimulation of NO production by extant eNOS.

Natural History & Biodiversity

SMALL MAMMALS FROM THE CLOUD FOREST AT CERRO BOBI, SIERRA DE LOS CUCHUMATANES,GUATEMALA. Nicte Ordonez[1], Walter Bulmer[2], Ralph P. Eckerlin[2], & John O. Matson[3], [1]Dept. Biol. Sci., Texas Tech Univ., Lubbock, TX 79409, [2]Div. of Nat. Sci., Northern VA Community Coll., Annandale, VA 22003, [3]Dept. Biol. Sci., San Jose State Univ., San Jose, CA 95192. Very little is known about small mammal ecology and distribution in the highlands of Guatemala. Small mammals were removal trapped from the mixed hardwood/coniferous cloud forest at Cerro Bobi in the Sierra de los Cuchumatanes, Huehuetenango, Guatemala during July 2005 and December 2005/January 2006.The coniferous cloud forest is located at 5km SW San Mateo Ixtatan, NW side of Cerro Bobi (3110m).The habitat can be characterized as follows: overstory of fir (*Abies*), pines (*Pinus*), oaks (*Quercus*), and other broad-leaved trees. A heavy litter of moss, lycopsids, ferns, and fallen logs was on forest floor. A total of 131 individuals representing 10 species of small mammals (8 rodent and 2 shrew) was collected. The site was sampled during two distinct seasons (wet and dry). While there were small differences in the small mammal species composition and abundance between the 2 samples, this was attributed to small sample size and sampling error. *Peromyscus guatemalensis* was the most abundant species in both seasons. Especially important is the collection of the Maya mouse (*Peromyscus mayensis*), not formally reported since its original description in 1975.

IDENTIFICATION OF CRYPTIC CHLOROPHYTES THROUGH MOLECULAR SEQUENCE DATA. Matthew R. Semcheski, Todd A. Egerton & Harold G. Marshall, Department of Biological Sciences, Old Dominion University, Norfolk VA 23529. The phenomenon of phenotypic plasticity is evident in many organisms throughout the natural world and is a byproduct of the biotic and abiotic factors of the environment in which an individual or population inhabits. Plasticity is especially prominent among microscopic photosynthetic taxa, which produce a variety of ambiguous forms. We identified a number of morphologically variable specimens, all originating from a single monoculture of the chlorophyte *Scenedesmus sp*., which included single-cell spinous and non-spinous forms, along with mulitcelluar spinous and non-spinous forms. In order to discern plasticity vs. genetic variation in a number of ecomorphs of *Scenedesmus sp*., the complete ITS-1, 5.8S, and ITS-2 region was sequenced. Phylogenetic analyses confirmed that all samples analyzed, while being morphologically distinct, do indeed belong to the *Scenedesmus* genus. Upon further investigation, it was determined that at the outset, with nutrients non-limiting and an absence of predators, *Scenedesmus sp*. grew rapidly in the single-cell non-spinous form.