Education Posters

**E-2000**

The Effect of High Fat Diet on Skeletal Muscle Inflammation in Obesity. I. ADITYA, M. Khan, and M. C. Samaan. Division of Pediatric Endocrinology, Department of Pediatrics, McMaster Children's Hospital, McMaster University, 1200 Main Street West, Hamilton, ON, CANADA. Email: ishanaditya5@gmail.com

While response of adipose tissue to obesity is well characterized, virtually nothing is known about the interaction between skeletal muscle and macrophages. This is important because muscle is the largest metabolic organ in the body that takes up about 75% of meal related carbohydrate load and is a major determinant of blood sugar levels after a meal. The aim of this study was to investigate whether macrophages infiltrate skeletal muscle in obesity in mice. The hypothesis is that high fat diet in mice leads to macrophage infiltration of skeletal muscle when compared to mice fed normal diet. RNA isolated from muscle was used to generate cDNA that was used in Quantitative Real-Time Polymerase Chain Reaction (qRT-PCR). This showed an increase of macrophage content in muscle of obese mice compared to lean mice using f4/80 macrophage specific marker. This was associated with increased expression of a known macrophage attractant called Monocyte Chemoattractant Protein-1 (MCP-1). The findings of this study demonstrate that high fat diet results in production of factors by muscle that attract macrophages. As skeletal muscle tissue becomes inflamed, in obesity, this may be a contributing factor to muscle insulin resistance seen in obesity. This may allow the design of therapeutic interventions that may help combat obesity-related complications including type-2 diabetes mellitus.

**E-2001**

A Physiological Response of Human Airway Epithelial Cells to Arsenic Exposure. P. ANGELI1, C. Sherwood2, and S. Boitano2. 1Catalina Foothills High School, 4300 E Sunrise Dr., Catalina Foothills, AZ 85718 and 2Bio5 Institute, 1657 E. Helen St., Tucson, AZ 85721. Email: pangeli95@gmail.com

Arsenic's potency and the difficulty long associated with its detection have made it famous as a poison. However, the real danger of arsenic to human health is through consumption of contaminated drinking water, as arsenic is ubiquitous in the environment. Arsenic exposure has been associated with both malignant and non-malignant diseases in multiple organ systems, and has been identified as a health risk around the world. Despite its long history as a human toxicant, little is known about arsenic's mode of action in human disease. In this study we investigated the impact of arsenic-induced reactive oxygen species on cytotoxicity in human bronchial epithelial cells (16HBE14o-). We used a high throughput assay capable of monitoring cell growth over an extended period of time (Roche xCelligence Real Time Cell Analyzer). The cells were grown in the xCelligence and monitored for 24 hours with a subset of cells supplemented with 1 mM sodium pyruvate in the media for protection from reactive oxygen species. After 24 hours of growth arsenic was added at varying concentrations. The cells grown in the presence of sodium pyruvate were continuously exposed to sodium pyruvate upon exposure to arsenic. Another subset of cells was given sodium pyruvate (1 mM) at the start of the arsenic exposure. Upon addition of arsenic, cells were monitored over 24 hours. We found that the addition of sodium pyruvate did not result in a significant dampening of initial cell signaling or cytotoxicity compared with controls. Further experiments yielded an arsenic-induced cytotoxic affect that was dose-dependent. We are currently elucidating the presence of the superoxide anion using a dihydroethidium detection assay for which preliminary results appear to indicate a strong presence at 500 ppb arsenic.

**E-2002**

The ERG1a K⁺ Channel Modulates NF-κB Expression in Mouse Skeletal Muscle, Suggesting ERG1a Modulates Skeletal Muscle Atrophy Through the IKK-β/IκB-α/NF-κB Pathway. CHASE LATOUR1, Christine Jaeger2, and Amber Pond3. 1Carbondale Community High School, Carbondale, IL 62901; 2School of Veterinary Medicine, Purdue University 47907; and 3Anatomy Dept., Southern Illinois University School of Medicine, Carbondale, IL 62901. Email: 1Presenting author: stitch5078@yahoo.com; 2Corresponding author: apond@siusom.edu

The ERG1 gene encodes a K⁺ channel known to be partially responsible for cardiac action potential repolarization in humans and mice. We have shown that the ERG1a channel participates in the onset of skeletal muscle atrophy (muscle...
loss) by up-regulating ubiquitin proteasome proteolysis (UPP) and that ERG1α expression induces expression of MuRF1, a UPP ligase. Here, we explore the mechanism by which ERG1α modulates MuRF1 expression. It is known that Murf1 expression is linked to activation of NF-κB, by which ERG1α modulates MuRF1 expression. We hypothesized, therefore, that ERG1α expression may induce NF-κB expression. Thus, we ectopically co-expressed ERG1α and an NF-κB luciferase reporter in mouse gastrocnemius muscles: 1) treatment group (n=12), left legs received NF-κB firefly luciferase and Renilla luciferase (RL) reporters and a control plasmid while right legs received the same reporters and ERG1α expression plasmid; 2) control group (n=12), both legs received reporters and the control plasmid. The gastrocnemius muscles were electroporated. After 7 days, we performed dual luciferase assays on skeletal muscle homogenates and determined right to left leg luciferase activities ratios per mouse. Skeletal muscle receiving ERG1α had a 52.7% decrease in luciferase activity relative to the controls (p>0.01). Surprisingly, the data show that ERG1α expression decreases NF-κB transcription and suggest that ERG1α might modulate MuRF1 transcription through the IKK-β/IκB-α/NF-κB pathway. Supported by NIH 1R03AR053706-01A2 to ALP.

E-2003

Bactericide: Which Product Is Better? SYDNEY MCNEILL. Dyersburg High School, 125 Highway 51 Bypass, Dyersburg, TN 38024. Email: sydney.mcneill@yahoo.com

Bacteria is a prokaryote that can infect the human skin. Before surgery and on minor cuts, antibacterial products are needed to rid the skin of bacteria, so the best type of antibacterial agent should be used. This project tests Product A, which contains the three antibiotics bacitracin, neomycin, and polymixin B, and Product B, which is made of povidone-iodine, to determine which retards the growth of skin bacteria more efficiently. In order to test these products, two volunteers' arms, hands, legs, and feet were graphed into five different parts. Then, swabs were taken from one grid on all four parts for the control group and for the products. The right side was used for Product A, and the left side was used for Product B. The petri dishes were put into an incubator and measured in square centimeters for the next three days. The total bacteria for Product A on day one is 8.7 cm², on day two is 9.85 cm², and on day three is 11.05 cm². For Product B, the total bacteria for day one, two, and three is 3.3 cm², 10.325 cm², and 22.18 cm² respectively. The data recorded contradicted the hypothesis because it showed that Product A retarded the growth of skin bacteria more efficiently than Product B. The experiment showed that Product A works efficiently over the three day period while Product B only works efficiently on the first day because it allowed a large number of bacteria to grow on the following two days.

E-2004

The Effects of Nicotine on Carcinoma Cell Proliferation. OLIVIA E. SAYER. Warren Tech, 13300 West 2nd Place, Lakewood, CO. Email: oes@precisionvc.com

The purpose of this experiment was to determine whether nicotine increases proliferation of cancer cells. The hypothesis was that nicotine would measurably increase cell proliferation. The original idea was to prove that smoking caused cancer, and progressed to the current form, to test if nicotine promoted proliferation of already cancerous cells. Human lung, colon, pharynx and pancreas cancer cells were tested for proliferation with varying nicotine concentrations. The cells were grown in normal and starved conditions to assess proliferation from nicotine vs. proliferation from media. After treatment and incubation, the cells were assayed and calculated using a micro-plate reader. The results from the plate read were normalized to the control (untreated) wells and averages of each set of wells were taken. The nicotine stimulated proliferation in three of the six cell lines. The plate reader measured cell viability using fluoroscence; the lung, pancreas and one pharynx line showed increased proliferation with the presence of nicotine. The other three lines showed little change in fluorescence between the treated and untreated cells. Little is currently known about the effects of nicotine on carcinoma cell proliferation. It measurably increased proliferation in certain lines, but not others. It is for this reason that much more experimentation is needed. Other carcinoma cell lines should be tested to evaluate whether or not they show increased proliferation with the presence of nicotine. Testing the lung, colon, pharynx, and pancreas lines has given great baseline information and set a standard that testing on other lines might be established.

E-2005

Does \textit{Isaria fumosorosea} (an Entomopathogenic Fungi: No Common Name) Hinder the Growth of Other Fungal Species and If So, to What Extent Does It Hinder the Growth? FAIZAH SHAREEF. Lincoln Park Academy, Port St Lucie, FL 34986. Email: faizahshareef786@gmail.com

The purpose of this experiment was to determine how and to what extent \textit{Isaria fumosorosea} (Ifr) hinders the growth of chosen entomopathogenic fungal species and to determine if the species are compatible. If they are, those fungal species can be used on the same plant plane to combat different pathogenic insects. The researcher plated Ifr with \textit{Trichoderma}, \textit{Fusarium}, \textit{Paecilomyces lilacinus}, and Ifr. The species were plated 6.5 cm away from each other, plated alone, and plated with liquid Ifr on PDA- chlorophenocol agar. These were placed in an incubator for 12 days. The growth was recorded each day after the 2nd day. For the group plated with liquid Ifr, the radial growths were measured after the 7th day and the 12th day. Everything
was incubated at 25°C. Two repetitions were done. The plates labeled Ifr x Ifr were not measured because the colonies began to merge and became immeasurable. Through this experiment the hypothesis was determined correct. Ifr was compatible with Paecilomyces lilacinus but weakly antagonistic towards the other fungal species and did hinder their growths. When liquid Ifr was used (more Ifr spores than the other specie) it was determined that Ifr competes more effectively with increased spore count.

**E-2006**

Stimulants and Inhibitors of VCAM-1 Expression in Vascular Smooth Muscle Cells. MAIREAD TOMS$^{1,2}$, Collin Hensien$^{1,2}$, and Coleen McNamara$^{2}$. 1Albemarle High School Math Engineering and Science Academy, 2775 Hydraulic Road, Charlottesville, VA 22901 and 2Robert M. Berne Cardiovascular Research Center, 415 Lane Rd., Charlottesville, VA 22908. Email: maireadtoms@gmail.com

Background: Factors that promote artery blockages may do so, in part, by activating the expression of adhesion molecules like VCAM-1 on VSMCs. Once expressed, this adhesion molecule may cause the macrophages filled with lipid in the artery wall to stick to the VSMCs and not exit, causing accumulation of fats and cells that lead to the blockages. Purpose: To determine if factors that promote artery blockages (TNFα and LDL cholesterol) may do so by stimulating the expression of an adhesion molecule called Vascular Cell Adhesion Molecule (or VCAM-1) in Vascular Smooth Muscle Cells (VSMCs), and if factors that are felt to protect from artery blockages can inhibit this effect. Procedure: VSMCs were transfected with the human VCAM-1 promoter fused to the firefly luciferase gene. These cells were treated with the individual Ocimum genus infusions, incubated for 45 hours. Trypan Blue staining and microscopic counting of dead cells led to the median lethal dose of H2O2 as 100 μM. MRC-5 cells were pre-treated with the individual Ocimum genus species at 10 ng/ml, LDL(10 μg/ml) and oxLDL (10 μg/ml) cholesterol with and without pre-treatment with the antioxidants Tyrosol and Resveratrol. Light emission from a substrate mixed with the cytoplasm of VSMCs containing the VCAM-1-luciferase gene was measured and normalized to amount of protein in each cell culture plate. Data: Results demonstrated that TNFα, LDL and oxLDL all stimulated a marked increase in VCAM-1 promoter activity in VSMC (> 50 fold, p<0.05). Tyrosol, but not Resveratrol blocked TNFα-induced VCAM-1 promoter activation. Both Tyrosol and Resveratrol inhibited LDL and oxLDL-induced VCAM-1 promoter activation. Although, Resveratrol was more potent at inhibiting oxLDL-induced VCAM-1 activation. Conclusions: These In Vitro studies provide evidence that inflammatory mediators like TNFα, LDL and oxLDL stimulate VSMC to activate the VCAM-1 gene. Anti-oxidants, like Tyrosol and Resveratrol inhibit this effect. Further studies are needed to confirm these results and determine if these factors can modulate in macrophages sticking to VSMCs. Antioxidants such as Tyrosol and Resveratrol may protect from blocked arteries by inhibiting these effects.

**E-2007**

In Vitro Study of Antioxidant Activity of Ocimum Genus Against Hydrogen Peroxide Induced Oxidative Stress and Apoptosis in Human Lung Fibroblasts. SANGAMITHRA VARDHAN. West Shore Jr/Sr High School, 250 Wildcat Alley, Melbourne, FL, 32935. Email: vardhan.sanju@gmail.com

Cell apoptosis, caused by oxidative stress, is an important step leading to Chronic Obstructive Pulmonary Disease (COPD). Chronic bronchitis and pulmonary emphysema are forms of COPD. This project explores natural antioxidants that can help inhibit apoptosis, preventing oxidative lung damage that leads to COPD. The purpose of this project is to find out which of the Ocimum genus species - Ocimum basilicum, Ocimum gratissimum, and Ocimum tenuiflorum - exhibits the most antioxidant properties by inhibiting apoptosis, due to oxidative stress induced by H2O2 in MRC-5 cells. Copper reducing equivalents (CRE) of the Ocimum genus species were determined. CRE is directly proportional to the total antioxidant capacity. The average CRE of O. tenuiflorum (16,229 μM) was the highest, the second highest was that of O. basilicum (15,207 μM) and the least was that of O. gratissimum (14,876 μM). MRC-5 cell lines treated with various concentrations of H2O2, ranging from 450 μM to 0 μM, were incubated for 45 hours. Trypan Blue staining and microscopic counting of dead cells led to the median lethal dose of H2O2 as 100 μM. MRC-5 cells were pre-treated with the individual Ocimum genus infusions, incubated for 60 h, then treated with 100 μM H2O2 concentration, and incubated for 22 h. The ANOVA test showed that the average percentage of viable cells was significantly different among the herbal groups: O. tenuiflorum (90.25%), O. gratissimum (75.49%), and O. basilicum (74.28%). The two tailed p-value tests showed that O. tenuiflorum inhibited apoptosis significantly more than the other two species. O. gratissimum and O. basilicum inhibited apoptosis in similar measure.