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Abstract Booklet

ABSTRACTS

Oral Presentation Abstracts

Presenters are indicated with “*” next to their names.

O.01

INVESTIGATING THE ROLE OF BACE2 IN MELANOCYTES DEVELOPMENT USING CHEMICAL SCREENING. Talia Guardia*, Yan Zhang, Milena Zimmer, and Richard White, Memorial Sloan Kettering Cancer Center, New York, NY.

Beta-site APP-cleaving enzyme 2 (BACE2) is a membrane bound aspartic protease that shares known characteristics of beta-secretases and is structurally similar to BACE1. Although similar, BACE1 and BACE2 have different expression patterns and physiologic functions. We interrogated the function of Bace2 in melanocyte development as well as in melanoma progression. In zebrafish development, BACE2 is highly expressed in melanocyte and loss of BACE2 results in atypical melanophores, with hyper-dendritic melanocytes in the tail fin, and proliferative melanocytes in the head. However, the mechanism involved in this phenotype is not understood. We decided to interrogate the mechanism using the Sigma LOPAC 1280 chemical library. The chemicals, at 30 μ M concentrations, were applied to BACE2^{-/-} zebrafish embryos 24 hpf and the phenotype scoring was done blind to the identity of the chemicals at 3dpf. The phenotype scoring system ranged from 0-5, with 0 being no suppression and 5 being complete suppression of fin tail phenotype. We pursued chemicals that had an initial phenotype score of 4 or 5, which were only 4 out of the 1,280 chemicals. Using chemical suppressor screening, we identified four top chemical hits that suppressed the BACE2^{-/-} phenotype in zebrafish embryos. LY-294,002 hydrochloride, Wortmannin, and AS605240 are known phosphoinositide 3-kinase (PI3K) inhibitors and Temsirolimus is a mechanistic target of rapamycin (mTOR) inhibitor. These chemicals were validated for reproducibility and optimal conditions that would result in maximum suppression of the phenotype. Collectively, these independent results suggest the involvement of the PI3K/Akt/mTOR pathway in the BACE2^{-/-} phenotype. We are now investigating the mechanism by which loss of BACE2 leads to defects in PI3K signaling. Moreover, given the central role of PI3K signaling in cancer, including melanoma, we are now investigating how loss of BACE2 affects melanoma phenotypes. Having a better understanding of the mechanism involved in BACE2^{-/-} phenotype during development will provide us insight into the role of BACE2 in melanoma.

O.02

HIDRADENOCARCINOMA. Justin Donlan* and Marcia Driscoll, Department of Dermatology, University of Maryland School of Medicine, Baltimore, MD.

Hidradenocarcinoma is a rare and potentially lethal neoplasm of the eccrine sweat gland that presents both diagnostic and therapeutic challenges. The rarity of these cancers and their similar presentation to other skin lesions make them difficult to identify clinically. Hidradenocarcinomas tend to occur as solitary, firm, erythematous, exophytic nodules on the face or upper extremities in men and women in their 5th to 7th decades of life. Histopathology is required for the diagnosis of these lesions as most are confused with Basal Cell Carcinoma. Unfortunately, these tumors are aggressive and roughly 30% of cases show lymph node or visceral involvement at the time of diagnosis. Due to the rarity of these tumors, treatment guidelines have not been established. Wide local excision with sentinel lymph node biopsy has been proposed as a potential therapeutic option, however the post-surgical survival rates are discomfoting. Here we present the case of a 68 year

Caucasian woman recently found to have a Hidradenocarcinoma on biopsy and her subsequent treatment course.

O.03

SEARCHING FOR BACTERIAL DNA INTEGRATION IN A MOUSE MODEL OF ETBF-INDUCED COLON CANCER. Alex Casella* and Julie Dunning Hotopp, Department of Microbiology and Immunology, University of Maryland School of Medicine, Baltimore, MD.

Bacteroides fragilis (*B. fragilis*) is a commensal bacterium found in the mouse colon. Enterotoxigenic *B. fragilis* (ETBF) strains are known to induce colitis and colon tumorigenesis in mouse models. While some of the pathogenesis of these diseases has been attributed directly to inflammatory mechanisms, the genetic factors contributing to the development of colon cancer in these mice have remained relatively unexplored. Given that bacterial DNA has previously been found in the genomes of various human cancer cells and that the colon is an area where bacteria are in close contact with epithelial cells, ETBF induced colon cancer is an ideal model to examine the role of bacterial DNA integration (BDI) in tumorigenesis. A well-described model of this disease process is the ETBF+ *Apc*^{+/Delta716} mouse. These mice are C57BL/6J mice that are heterozygous for an insertional mutation in the *Apc* gene, rendering them highly susceptible to intestinal adenoma formation. In this model Min mice are orally colonized with ETBF, which triggers an inflammatory colitis with rapid development of colonic tumors. We hypothesized that ETBF+ *Apc*^{+/Delta716} mouse would show evidence of bacterial DNA integration into the genome of these colon tumors. We performed exome and RNA sequencing on four tumor samples using the Illumina HiSeq platform, and three normal samples await sequencing. The computational pipeline developed by the Dunning Hotopp group was used for identification of putative sites of BDI. Fifty-one possible sites were identified by the pipeline; however, we did not have enough coverage to be confident that this was due to BDI and not contamination.

This research was supported by MSTP T32.

O.04

CONCURRENT PP2A ACTIVATION AND FLT3 INHIBITION ENHANCE APOPTOSIS INDUCTION IN FLT3-ITD ACUTE MYELOID LEUKEMIA CELLS THROUGH DEGRADATION OF PIM1 KINASE. Sikemi Ibikunle*, Patrick Baldwin, and Danilo Perrotti, and Maria Baer, Division of Hematology and Oncology, Department of Medicine, University of Maryland School of Medicine, Baltimore, MD.

Internal tandem duplication of the *fms*-like tyrosine kinase 3 growth factor receptor (FLT3-ITD) is present in acute myeloid leukemia (AML) cells in 30% of patients and is associated with poor treatment outcomes. FLT3 inhibitors have limited clinical efficacy. Proteins involved in FLT3-ITD oncogenic signaling have been studied as potential additional targets for treatment. The proliferative and anti-apoptotic effects of oncogenic kinases including FLT3-ITD and its downstream target Pim-1 may be countered by the tumor-suppressor phosphatase protein phosphatase 2A. Our preliminary findings indicate that concurrent PP2A activation increases cytotoxicity of FLT3 inhibitors. Here, we seek to develop a model of the activity of the PP2A-activating drug, FTY720, in cells with FLT3-ITD. We hypothesize that FTY720 reduces Pim-1 expression via a posttranslational mechanism, namely an increase in Pim-1 degradation. Overall, this study may assist in the development of new treatment approaches for AML patients with FLT3-ITD.

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O.05

DEVELOPMENT OF A 3D PRINTED BONE RESIN LOADED WITH GROWTH FACTOR LADEN MICROPARTICLES. Dylan Bertoni*, Jesse Placone, Max Lerman, Anjana Jeyaram, and John Fisher, Department of Bioengineering, UM College Park School of Engineering, College Park, MD.

Bone can become severely injured beyond the bodies' abilities to repair it in instances of trauma, cancer, or other diseases. These critically sized bone defects require a surgically implanted graft for healthy bone to regrow. These grafts can be autologous (from the patient) or allopathic (from a cadaver). There are roughly 500,000 autologous graft procedures per year in the USA, the most common method to repair bone injuries due to the natural osteogenic properties of the donor bone, with no risk of host rejection or transmission of disease. These procedures require a secondary operation site, however; and typically longer operations with multiple opportunities for infection. There are 200,000 allografts performed per year, for bone repairs where the graft is too large to be provided by the patient themselves. These are more expensive and, even with proper preparation, still pose a risk for disease transmission. 3D printing bone tissue provides a means to synthetically produce complex and biocompatible bone scaffolds that eliminate the morbidities associated with autologous and allopathic grafts. Polycaprolactone (PCL) is a polymer capable of being 3D printed at elevated temperatures (70°C) to mimic the mechanical properties and porosity of bone. For synthetic grafts to be successful, they must allow for the invasion of host capillaries and stem cells, and allow those cells to differentiate into bone forming tissue. To make PCL more osteoinductive, controlled Vascular Endothelial Growth Factor (VEGF) release could stimulate the vascularization of the graft; release of Bone Morphogenic Protein-2 (BMP-2) could induce stem cell differentiation into osteoblasts. To this end, we have developed a novel 3D printable resin by incorporating a solvent which allowed it to be printed at lower temperatures (50°C), so that alginate microparticles loaded with these growth factors could be added to the material. The loading concentration and mixing requirements were studied to create a homogenous resin, then release profiles of these growth factors were determined with ELISAs resulting in a biocompatible material with the potential to act as an osteoinductive bone graft.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

O.06

A NOVEL COMPUTER ALGORITHM FOR 3D PRINTING A PROSTHETIC NOSE: A PILOT STUDY. Meryam Shikara*, Christopher Rizzi, Brian Zelip, Jewel Greywoode, and Kalpesh Vakharia, Department of Otorhinolaryngology - Head and Neck Surgery, University of Maryland School of Medicine, Baltimore, MD.

Nasal defects are most commonly caused by Mohs surgery resections of basal cell carcinoma, squamous cell carcinoma and melanoma. These defects can lead to a great deal of psychological stress for patients. Reconstruction techniques for the nose require multi-step operations, and the process may take months. Prosthetics are commonly used in maxillofacial rehabilitation in lieu of reconstruction, or as a temporary fix to fill the defect. Three-dimensional (3D) printing techniques using CT scans to create 3D models of organs have revolutionized the field of medicine. Unfortunately, the cost of some of these modeling softwares remain a significant barrier to large-scale availability of these products. We developed a computer algorithm utilizing a commercially available 3D animation software, Blender (Blender Foundation, Inc, USA), and Adobe Photoshop CS6 (Adobe Systems, Inc, USA) to create a 3D model of a nose. Photographs of five subjects were

processed with the computer algorithm to create a virtual 3D model of each nose. The model was then printed using a Desktop 3D printer. Attending physicians, residents, and medical students completed a survey, and were asked to rate the similarity between the subjects' photographs and their 3D printed nose on a Likert-type scale [0- completely different to 10- identical]. Thirty-six survey respondents evaluated four views for each of the five modeled noses. The mean score for the overall similarity between the photographs and the 3D models was 8.41 +/- 1.27. The mean scores for each nasal comparison ranged from 7.97 to 8.62. When asked to match the noses, 97.8 % of respondents were able to match the correct 3D nose to the corresponding subjects' photographs. All clinicians surveyed indicated that they would consider utilizing this tool to create a temporary prosthesis rather than referring to a prosthodontist. This computer algorithm can be utilized to model and 3D print a human nose. The 3D printed models closely depict the actual images of each subject's nose, and can potentially be used to create a temporary prosthesis to fill external nasal defects.

O.07

GLYCOENGINEERING PROMOTES STEM CELL NEURONAL DIFFERENTIATION. Cynthia Xu*, Jian Du, and Xiaofeng Jia, Department of Neurosurgery, University of Maryland School of Medicine, Baltimore, MD.

One of the most prevalent experimental methods to treat neural injury is stem cell therapy since stem cells have a variety of functions, including the release of growth factors into the environment. Alone, this method has not been able to treat neural injury satisfactorily. Metabolic glycoengineering (MGE) is a technology that can change cell glycans, and regulate cell adherence and differentiation properties. This study combines stem cell therapy with MGE in vitro in hopes of producing a modified stem cell that will be more differentiated and therapeutic. Neural stem cells (NSCs) were cultured and subjected to MGE to induce differentiation into a neural phenotype. Morphological changes and stages of differentiation were determined through immunohistochemistry, RT-PCR and western blot. The results demonstrate that differentiation occurred to a significantly greater degree in cells subjected to MGE than in the untreated cells. Most importantly, MGE accelerated stem cell differentiation. Following neural injury, the most crucial point of time to treat is immediately following injury, thus using glycoengineered-stem cells could potentially further enhance nerve recovery. In addition to in vitro studies, a novel nerve staining method was developed to differentiate between motor and sensory nerve fibers using immunohistochemistry staining. This visualization will be useful for future in vivo studies to observe nerve regeneration.

This research was supported in part by the Proposed Research Initiated by Students and Mentors (PRISM) Program, University of Maryland School of Medicine Office of Student Research.

O.08

CHANGES IN LYMPHATIC VASCULATURE IN THE DISEASED HEART. Cherriese Thompson* and Polina Goihberg, Department of Anesthesiology, Brigham and Women's Hospital, Boston, MA.

Heart disease is caused by various conditions, including Type 2 Diabetes Mellitus (T2DM) and prior instances of myocardial infarction (MI). Cardiac lymphatic vessels play an important role in normal heart function and myocardial responses to tissue damage. In the present study, we examined the impact of MI and T2DM on cardiac lymphatic vasculature in mouse models of human diseases. We employed immunolabeling and fluorescence microscopy of human lymphatic endothelial cell (LEC) markers, such as LYVE-1 and podoplanin (PDPN), along with other proteins of interest. For MI, cardiac samples were obtained from wild-type mice subjected to permanent coronary artery ligation. Non-operated (NO) and sham-operated (SO) mice were used as controls.

For T2DM, TallyHo/Jng mice were compared with genetically-matched healthy controls, SWR/J. Additionally, flow-cytometry was utilized to evaluate the phenotype of cardiac cells isolated from mouse hearts after MI. Immunocytochemistry and flow-cytometry assessed LECs treated with insulin, cytokines, and growth factors. Immunohistochemical analysis confirmed robust lymphangiogenesis in the scarred region of the infarcted heart. PDPN labeling identified multicellular aggregates that do not display the LEC phenotype. Flow cytometry revealed a 3-fold increase in the density of PDPN-positive cells two days after MI compared to SO and NO mice. On average 20% of PDPN-expressing cells co-expressed CD11b or F4/80 markers, indicating that the bulk of the PDPN-containing cells is not committed to myeloid lineages. Also, a 3-fold increase in the density of lymphatic vessels was found in the samples from TallyHo/Jng mice compared to SWR/J. Moreover, while PDPN and LYVE-1 were typically co-expressed in the lymphatic vessels of healthy hearts, the amount of LYVE-1 in LECs was reduced in diabetic mice. Consistent with this, LECs exposure to insulin, cytokines, or growth factors reduced LYVE-1 expression. These data indicate that T2DM induced conditions affect cardiac lymphatic vessels. Our findings inform on extensive changes in the endothelium of cardiac lymphatic vessels of the mice models of human diseases, namely, post-MI heart failure and T2DM.

This research was supported by the Brigham and Women's Hospital Center for Faculty Development and Diversity.

O.09

EFFECTS OF GLP-1 INFUSION ON VASCULAR ENDOTHELIUM DURING HYPOGLYCEMIA. Aaron Grubner*, Nino Joy, Lisa Younk, Donna Tate, and Stephen Davis, Department of Medicine, University of Maryland School of Medicine, Baltimore, MD.

Diabetes is a disease characterized by hyperglycemia. The landmark DCCT study in T1DM established the significant and well-known correlation between hyperglycemia (high A1c levels) and the microvascular complications associated with diabetes. However, studies attempting to connect hyperglycemia with the macrovascular complications associated with diabetes (i.e. CVD) have shown mixed results. Recently, some studies in T2DM have associated hypoglycemia, as opposed to hyperglycemia, with CVD. Hypoglycemia has been shown to activate inflammatory and pro-atherothrombotic mechanisms which could help contribute to CVD in people with diabetes. Glucagon-like peptide-1 (GLP-1) agonists, a potent medication used to treat T2DM, has been shown to have beneficial effects on the vascular endothelium and myocardium. These effects would make GLP-1 a most useful and exciting agent in the prevention of adverse effects of hypoglycemia. To date there are scarce data investigating the effects of GLP-1 infusion on pro-inflammatory responses during hypoglycemia (Three reports from Ceriello et al). The specific aim of our study was to determine if GLP-1 could reduce deleterious effects of hypoglycemia on in-vivo vascular biologic mechanisms. Subjects participated in two one day studies consisting of 2 hours of pancreatic clamp followed by 2 hours of hyperinsulinemic/hypoglycemic glucose clamp. At appointed time points before and during the induced hypoglycemia, blood was collected and examined for markers indicating vascular responses. Flow mediated dilation was measured to test endothelial function of the brachial artery. Additionally, microneurography was performed to gauge sympathetic nerve activity and 3-H3 glucose was infused to measure glucose kinetics. We found that during moderate hypoglycemia (50 ± 1 mg/d/L) in healthy individuals, GLP-1 infusion reduced pro-inflammatory, pro-atherothrombotic, and pro-coagulant responses. It also improved fibrinolytic balance and improved endothelial function via both endogenous and exogenous NO-mediated mechanisms.

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O.10

FUNCTIONAL STATUS PREDICTS MAJOR COMPLICATIONS AND DEATH AFTER ENDOVASCULAR REPAIR OF ABDOMINAL AORTIC ANEURYSMS. Connor Oates*, Donald Harris, and Robert Crawford, Department of Surgery, University of Maryland School of Medicine, Baltimore, MD.

Endovascular aortic repair (EVAR) is a lower-risk option for treating abdominal aortic aneurysms (AAA), particularly in patients with poor functional status who are often unfit for open surgical repair. The contribution of preoperative functional status to outcomes after EVAR, however, has been poorly defined. Using data from the National Surgical Quality Improvement Program (NSQIP) database, we aimed to identify whether impaired functional status is associated with worse outcomes after EVAR. Using NSQIP defined preoperative functional status, patients undergoing nonemergency EVAR were stratified as independent or dependent and compared by univariate analyses. The effect of functional status on mortality and major complications was also assessed by multivariable logistic regression. Dependent patients were older and more often minorities. They had higher rates of COPD, heart failure, renal failure. Dependent patients also were more likely to have an American Society of Anesthesiologists (ASA) score of 4 or 5. Preoperative dependent status was associated with higher rates of operative complications (34% vs 11%; $P < .0001$), systemic complications (13% vs 4%; $P < .0001$), and mortality (6% vs 1%; $P < .0001$). Adjusting for demographics and comorbidities, dependent status was an independent risk factor for mortality (OR, 3.4; 95% CI; 2.0-5.5) and major complications (OR, 3.0; 95% CI, 2.4-3.8). In addition, dependent patients had longer hospital lengths of stay (4 vs 2 days; $P < .0001$), and higher rates of reoperation (5% vs 3%; $P = .03$) and readmission (4% vs 2%; $P = .01$). Although EVAR is a minimally invasive method of repairing AAA, preoperative functional status is the leading determinant of postoperative major morbidity and mortality. Functional status may be used as a valuable marker of increased perioperative risk and to identify patients who may benefit from preoperative optimization.

O.11

A COMPARISON OF ANTICOAGULATION STRATEGIES IN VENO-VENOUS EXTRACORPOREAL MEMBRANE OXYGENATION. Aakash Shah*, Ya Zhou, Francis Brigante, Chetan Pasrija, and Zachary Kon, Division of Cardiac Surgery, Department of Surgery, University of Maryland School of Medicine, Baltimore, MD.

Bleeding remains a major source of morbidity associated with veno-venous extracorporeal membrane oxygenation (VV-ECMO). Moreover, there remains significant controversy, and a paucity of data, regarding the ideal anticoagulation strategy for VV-ECMO patients. In this study, all patients undergoing isolated, peripheral VV-ECMO (2009-2014) at a single institution were retrospectively reviewed. Three sequential eras of anticoagulation strategy were compared: activated clotting time (ACT: 160-180 sec), high partial thromboplastin time (H-PTT: 60-80 sec), and low PTT (L-PTT: 45-55 sec). The primary outcomes were number of blood products and major bleeding events per day on ECMO, and oxygenator changes and circuit changes per 100 days on ECMO. Major bleeding was defined as requirement of >2 units of red blood cell (RBC) transfusion in 6 hours, >4 units in 24 hours, or documented gastrointestinal, pulmonary, or cannula site hemorrhage. Thrombosis was defined by requirement for oxygenator change, circuit change, or plasma free hemoglobin >20 . Secondary outcomes were ICU length of stay (LOS), hospital LOS, survival to decannulation, and survival to discharge. 123 patients were evaluated: 53 ACT, 25 H-PTT, and 45 L-PTT. Median age was 46 (IQR 31-57) years. Pre-ECMO APACHE II scores, SOFA scores, and

Murray scores were not significantly different between the groups. Patients in L-PTT group required less RBC than the ACT or H-PTT group (2.1 vs. 1.3 vs. 0.8, $p=0.06$,

O.12

CRISPR-CAS9 EDITING OF THE BCL11A ERYTHROID-SPECIFIC ENHANCER REGION CAN AFFECT FETAL HEMOGLOBIN LEVELS. Oleg Makarevich*, Tami Kingsbury¹, and Curt Civin², ¹Department of Physiology and ²Department of Pediatrics, University of Maryland School of Medicine, Baltimore, MD.

Patients with Hereditary Persistence of Fetal Hemoglobin (HPFH) have been shown to have reduced severity of Sickle Cell Disease (SCD). In these patients, HbS polymerization is inhibited by high levels of fetal hemoglobin (HbF). Recently, HPFH has been shown to be due to a genetic mutation leading to disruption of the erythroid-specific enhancer of the BCL11A gene. Although the BCL11A gene is important in other systems, using gene-editing to disrupt only the erythroid-specific enhancer should lead to selective downregulation of BCL11A function in erythroid cells. Such genetic manipulation of human hematopoietic stem cells (hHSCs) should downregulate BCL11A levels and increase fetal hemoglobin levels in erythroid descendent cells. Successful manipulation could lead to the eventual possibility of significantly improving SCD patients' lives (possibly even eliminating medical problems associated with SCD) through autologous transplant of the patient's own HSCs after similar gene editing. Analysis of single-cell clones created using the CRISPR-Cas9 system with previously identified high-efficiency guide RNAs to disrupt BCL11A function was the focus of this project. Successful disruption was ascertained by sequencing the BCL11A erythroid-specific enhancer in the region of editing, and measuring fetal hemoglobin levels in an erythroid-differentiated hematopoietic stem cell line. The results of these tests indicated that gene editing was successful in K562 cells, an easily transfectable cell line. Subsequent testing also indicated that fetal hemoglobin levels in edited cell lines were significantly higher than in the wild type after erythroid differentiation. These results suggest that CRISPR-Cas9 editing of the BCL11A erythroid-specific enhancer region is possible and can be effectively used to increase fetal hemoglobin levels in erythroid cells.

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O.13

ROLE OF TRICELLULAR TIGHT JUNCTION PROTEIN IN NOISE-INDUCED HEARING LOSS IN MICE. Sergiu Costinas*, Saima Riazuddin, and Eldodie Richard, Department of Otorhinolaryngology - Head and Neck Surgery, University of Maryland School of Medicine, Baltimore, MD.

Epithelial cells rely on tight junctions in order to establish compartments. In the inner ear, they are critically important for maintaining different ionic compositions in the endolymphatic and perilymphatic fluids of the organ of Corti. A key component of tricellular tight junctions is tricellulin, which is particularly enriched at the contact points of three epithelial cells. Mutations in TRIC, encoding Tricellulin, have been associated with autosomal recessive nonsyndromic deafness in humans. Previous studies have shown that the p.Arg497* knockin mutation in Tric leads to rapidly progressing hearing loss in mice. While the impact of the homozygous mutation has been well characterized, the phenotype associated with the heterozygote mutation is still unclear. The aim of this study is to elucidate the effects of a heterozygous TRIC p.Arg497* mutation on the mouse inner ear and the response to a reversible temporary threshold shift (TTS) noise exposure. Auditory brainstem responses (ABR) were assessed seven days prior to noise exposure, 24 hours, seven days, and finally 14 days post exposure. Twenty six-week old mice were exposed to a 8-16kHz bandpass

noise at 94dB sound pressure level for 2 hours. Results showed a similar threshold increase at 24 hours post exposure between heterozygous (het) and wild type (WT) mice for all the tested frequencies (6, 8, 12, 16, 24, and 32 kHz). However, at seven days and 14 days post exposure, TRICR497X/+mice tend to have higher threshold shifts than TRIC+/+ mice, specifically at 24 kHz. Gross morphological analyses, as well as a refined study of the potential hair cell and synapse loss in the organ of Corti, were assessed between the Het and WT mice using Hematoxylin and Eosin staining and immunostaining. Further testing needs to be done to better assess the morphological and functional differences in these Tric mutants. Overall, our results suggest that mutation in Tric predispose to noise-induced hearing loss in mice.

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O.14

IDENTIFYING VARIANT SURFACE ANTIGEN (VSA) EPITOPES ASSOCIATED WITH MALARIA EXPOSURE VIA AN ULTRA-DENSE PEPTIDE MICROARRAY. Albert Zhou*, Department of Epidemiology and Public Health, University of Maryland School of Medicine, Baltimore, MD.

Malaria is a mosquito-borne infectious disease caused by parasitic protozoans of the Plasmodium genus. An erythrocyte infected with *P. falciparum* expresses adhesive variant surface antigens (VSAs) on its surface that play a critical role in sequestration and evasion of host immune responses. These VSAs possess extraordinary genetic diversity. In regions with malaria transmission, adults develop immunity to clinical disease, which is believed to be associated with the development of antibodies to particular VSAs. Although the best studied and largest family of VSAs include *P. falciparum* erythrocyte membrane proteins (PfEMP1s), the other major VSA families, the repetitive interspersed family of polypeptides (RIFINs) and subtelomeric variable open reading frame (STEVORs), have also been implicated in malaria pathogenesis. We aimed to identify epitopes involved in protective natural immunity by utilizing ultra-dense peptide microarrays, a platform consisting of thousands of 16-mer VSA peptides based on the *P. falciparum* reference genome 3D7. Sera obtained from malaria-exposed children and adults both prior to, during, and following a malaria season were examined to determine seroreactivity against a specific RIFIN from a reference genome. We found that sera from adults recognized 145 unique RIFIN peptides compared to 39 for children, suggesting that exposure to malaria parasites is associated with increased recognition of RIFIN peptides. Moreover, sera from adults reacted more intensely to 89 peptides than sera from children. Subsequent steps include identifying particular RIFIN regions of differential seroreactivity and expanding the analysis to compare results for additional RIFINs and STEVORs.

O.15

KETAMINE METABOLITE AND FAST ACTING ANTIDEPRESSANT 2R6R-HNK INDUCES A PRESYNAPTIC POTENTIATION OF SCHAFFER COLLATERAL SYNAPSES OF THE HIPPOCAMPUS. Jonathan Fischell*, Panos Zanos¹, Todd Gould¹, and Scott Thompson², ¹Department of Psychiatry and ²Department of Physiology, University of Maryland School of Medicine, Baltimore, MD.

Major Depressive Disorder (MDD) is the leading cause of disability in the U.S. for people between the age of 15 and 44. It affects roughly 14.8 million Americans or 6.7% of all people over the age of 18. Currently, depression is primarily treated pharmacologically and by far the most common class of drugs used are Selective Serotonin Reuptake Inhibitors (SSRIs). Unfortunately, SSRIs are variably effective and require weeks to months to reliably show any improvement even in successful cases. For this reason the development of new antidepressant drugs that have fast-acting

capabilities is a major focus in the field of depression today. Ketamine, a NMDA receptor antagonist, has been shown to have rapidly acting capabilities however its clinical viability is limited due to its addictive potential and severe adverse side effects. Recent evidence suggests that Ketamine may actually be exerting its antidepressant effects through its active metabolite 2R6R-hydroxynorketamine (2R6R-HNK) and that that effect appeared to be independent of NMDA receptor antagonism. If this is the case, 2R6R-HNK may be able to rapidly treat the symptoms of depression but may not be limited by the same problematic side effects. Although 2R6R-HNK is a promising candidate for a possible next generation of antidepressant treatment, its mechanism of action is still unknown. To investigate this I used electrophysiology to see if 2R6R-HNK would alter AMPA receptor mediated signaling at Schaffer Collateral (SC) and Temporoammonic (TA) synapses in the hippocampus. I found that 2R6R-HNK induced a potent potentiation of AMPA receptor mediated signaling at SC synapses but had no effect on TA synapses. I then used paired pulse analysis and found a decrease in paired pulse ratio following 2R6R-HNK induced potentiation which is suggestive of a pre-synaptic mechanism. This study gives evidence that 2R6R-HNK's antidepressant properties may involve an increase in activation of AMPA receptor mediated signaling in the hippocampus. Understanding how 2R6R-HNK acts on the brain provides us with a possible framework by which antidepressants could modulate the brain to treat the symptoms of depression.

O.16

DRIVING FORCES BEHIND OPIOID ABUSE IN THE ORTHOPEDIC TRAUMA POPULATION: A SCOPING REVIEW. Kaylie Miller*, Nathan O'Hara, and Gerard Slobogean, Division of Trauma, Department of Orthopaedics, University of Maryland School of Medicine, Baltimore, MD.

Prescription opioid abuse has accelerated rapidly in the United States over the past two decades, a problem that has infiltrated nearly all medical specialties. Orthopedic surgeons are the third highest prescribers of opioids, and thus are contributing to this problem at a significant rate. In the past two decades, there has been a surge in the number of publications and the focus in mainstream media on the subject of prescription opioid abuse. However, there has been little emphasis in the literature on opioid abuse among orthopedic trauma patients. The goal of this study was to address this knowledge gap. To achieve this, we employed a scoping review technique due to its ability to successfully address a broad research question. In order to better understand the type of information deemed relevant by opioid researchers, we further analyzed our search results by sorting the publications into the following categories: strengths of the consumer, strength of the suppliers, strengths of substitutes, competition within the field, and regulations (at the institution, profession, and government level). The search strategies generated 8,760 citations; of these, 1,166 publications satisfied our inclusion criteria. 607 of these final abstracts were marked as "extremely relevant" (52%) and the other 559 (48%) were marked "relevant." 36.4% of the total included articles applied to the strength of the suppliers and 19.6% provided information on the consumer. 25.2% of the included papers concerned substitutes for opioids. 15.7% focused on regulatory power in the opioid industry. 14% considered the competition within the industry, including power of both current stakeholders and potential new entrants. The current study provides a thorough summary of existing literature on opioid use in chronic pain and musculoskeletal trauma patients. Furthermore, the categorical division of the literature provides a unique perspective into the epidemic forces that are contributing to opioid use, and may assist in development of effective interventions to reduce excessive opioid use following traumatic injuries.

O.17

PHYSIOLOGIC FEATURES OF BRAIN DEATH. Eno-Obong Essien*, Kristina Fioretti¹, Thomas Scalea², and Deborah Stein², ¹Medical University of South Carolina School of Medicine, Charleston, SC and ²Division of Trauma Surgery, Department of Surgery, University of Maryland School of Medicine, Baltimore, MD.

Brain death is known to be associated with physiologic derangements but the incidence of these pathologies is poorly described. Precise knowledge of the physiologic disturbances that occur at the time of brain death is important for effective management of the potential organ donor thus we sought to characterize the pathophysiologic disturbances that occur at the time of brain death in patients with traumatic injuries. All brain dead patients over a 10 year period were identified from the trauma registry at a level 1 urban trauma center. Patient demographics, injury characteristics, and clinical data for defining organ dysfunction were reviewed for the 24 hours surrounding brain death declaration. 273 patients were identified. Mean age was 38 years (± 17.4). 73% were male. Major mechanism of injury was motor vehicle collision in 33%, penetrating injury in 23% and falls in 21% of the patients. Median injury severity score was 33 (IQR 25-43) with a median head abbreviated injury scale score of 5. Patients were pronounced at a median of 24 hours (IQR 15.7-51.7) following admission. The most common physiological disturbance noted was hypotension with 93% of subjects requiring vasopressors. Thrombocytopenia and acidosis both had an incidence of 77% in the subjects. The next most common disturbances were hypothermia ($<36^{\circ}\text{C}$) in 63% of the patients and moderate to severe respiratory dysfunction ($\text{PaO}_2/\text{FiO}_2 < 201$) in 63% of subjects. Myocardial injury (serum troponin >0.02) was seen in 56% but only 5.7% of patients manifested severe cardiac dysfunction with an ejection fraction of $<35\%$ on echocardiography. Diabetes insipidus was diagnosed in 45% of patients. Interestingly, coagulopathy (INR >1.4) was noted in only 56% and hyperglycemia (glucose $>200\text{mg/dl}$) was seen in 31% despite widespread belief that these occur nearly universally in the setting of brain death.

O.18

EFFECTS OF AEROMEDICAL EVACUATION-RELEVANT HYPOBARIA ON BRAIN INJURY AND MORTALITY FOLLOWING HEAD TRAUMA COMBINED WITH HEMORRHAGIC SHOCK. Wei Quan*, Parisa Rangghran, Julie Proctor, Juliana Medina, and Gary Fiskum, Department of Anesthesiology, University of Maryland School of Medicine, Baltimore, MD.

Approximately 300,000 U.S. combat casualties in recent wars have suffered traumatic brain injuries (TBI). Aeromedical evacuation (AE) is an important component in the care of many traumatic brain injury (TBI) patients and it exposes them to prolonged periods of hypobaria. Our research model looks at the effects of hypobaria on neurologic outcomes and survival in a rat polytrauma (PT) model consisting of controlled cortical impact (CCI)-induced TBI in combination with hemorrhagic shock (HS). We hypothesize that exposure to AE-relevant hypobaria increases mortality and neuropathology following PT. Our PT model consists of CCI to the fronto-parietal cortex of adult male Sprague Dawley rats followed by 30 min of moderate HS, followed by “field” resuscitation with Hextend, and then followed by “in-hospital” resuscitation with re-infused blood. At 24 hr after PT, rats were placed in our altitude chamber for 6 hr, which was adjusted to reach hypobaric conditions approximating those found in airplanes at cruising altitudes, and continuously flushed with either 28% or 100% supplemental oxygen. 30 days later, the brains from the PT rats were processed for quantitative, stereologic determination of cortical lesion volume and activation of microglia/macrophages (ED-1 histochemistry), and oxidative protein modifications (nitrotyrosine histochemistry). Our results indicate that the mortality rate of the rats with PT, followed by hypobaria under 100% O_2 was the highest (70%), compared to hypobaric 28% O_2 or to no

hypobaria (37%). All the rats in the sham group and CCI only group survived to 30 days. All PT and CCI only rats exhibited loss of cortical tissue in the range of 1-24 mm³ and ED-1 immunopositive penumbra ranging from 1-9 mm³. ED-1 immunostaining was also found in the corpus callosum in the range of 0-2.5 mm³. There was no significant cortical tissue loss or ED-1 immunoreactivity observed in the Shams. Additional quantification of cortical contusions, ED-1 immunostaining, oxidative stress, and neurobehavioral outcomes are in progress. The preliminary results demonstrating a high mortality rate for PT rats exposed to hypobaria under 100% O₂ is alarming and brings into question the use of pure O₂ during aeromedical transport of trauma patients. Stained tissues with ED-1 antibodies indicates that this inflammation is an ongoing pathology even after 30 days. Supported by PRISM, the American Academy of Neurology, and the US Air Force Medical Service.

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O.19

MULTIDETECTOR CT PREDICTS THE NEED FOR EARLY DECOMPRESSIVE SURGERY IN TRAUMATIC ABDOMINAL COMPARTMENT SYNDROME. Thomas Battey*, William Chiu¹, Uttam Bodanapally², and David Dreizin², ¹Division of Trauma and Critical Care, Department of Surgery and ²Division of Trauma Radiology, Department of Diagnostic Radiology and Nuclear Medicine, University of Maryland School of Medicine, Baltimore, MD.

To investigate computed tomography (CT) signs predicting the need for early (<24 hour) decompressive laparotomy in post-traumatic abdominal compartment syndrome (ACS). This institutional review board-approved and HIPAA-compliant retrospective study included 86 patients >18 years presenting with traumatic abdominopelvic injury, who developed ACS requiring surgical decompression, and had pre-operative CT between 2004-2015. Abdominopelvic fluid volumes were determined quantitatively using semi-automated segmentation. Additional imaging parameters recorded included largest single bowel wall, smallest inferior vena cava, and smallest aortic diameters; abdominal anteroposterior:transverse ratio; and presence or absence of hydronephrosis, bowel perforation, active arterial bleeding, and inguinal herniation. Relevant laboratory values at time of CT and surgery were abstracted. Patients were separated into early (<24 hours from CT to surgery) and late surgical decompression groups for outcome analysis. Correlation analysis, comparison of means, and multivariate logistic regression was performed. Bladder pressures were measured uncommonly (29%), but significantly more often in patients undergoing late surgery (p=0.03). Comparison of means revealed elevated abdominal fluid volumes (p<0.0001) and bowel wall thickness (p=0.003) in the early surgery group compared to the late surgery group. For laboratory values obtained closest to the time of CT, base deficit was increased (p=0.009), and pH (p=0.016) and bicarbonate level (p=0.013) were decreased in the early surgery group. In multivariate analysis incorporating both laboratory values and imaging features, abdominal fluid volumes (p=0.002;OR:1.003/milliliter) and bowel wall thickness (p=0.011;OR:1.259/millimeter) were independent predictors of early surgery. Segmented abdominopelvic free fluid volumes and increases in single bowel wall diameter together aid in triaging patients most in need of urgent surgical decompression for post-traumatic ACS.

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O.20

CT MARKERS OF FRAILTY IN TRAUMA PATIENTS PREDICT INJURY SEVERITY. Maxwell Raithe*, Joseph Kufera¹, Kathirkamanathan Shanmuganathan², Deborah Stein³, and Margaret Lauer*, ¹Shock, Trauma and Anesthesiology Research Organized Research Center, ²Department of Diagnostic Radiology and Nuclear Medicine, and ³Department of Surgery, University of Maryland School of Medicine, Baltimore, MD.

Age has classically served as a predictor of physiological condition and capacity. There is clinically significant variability in health and vitality for patients with the same chronological age. Frailty, generally defined as a combination of physical and cognitive decline, has been proposed as a superior indicator of patient resiliency than age. Previous studies have identified frailty through surveys and clinical tests, demonstrating correlations to poor surgical outcomes. Determining frailty from radiographic markers may be more advantageous for patients that have sustained traumatic injuries where such assessments are often infeasible. Researchers retrospectively reviewed the Shock Trauma Registry from 01/01/2010 –12/31/2015 to identify men and women over the age of 40 that sustained blunt injuries from motor vehicle collisions. Eligibility was restricted to restrained drivers of motor vehicles with successful airbag deployment that received full body trauma scans upon admission to the Shock Trauma Center (n = 394). Cerebral atrophy, cervical spine degeneration, reduced renal volume, pulmonary bullae formation, sarcopenic obesity, osteopenia, and vascular calcifications were all measured as possible computerized tomography (CT) markers of frailty. Each CT variable was measured by a single trained researcher using TeraRecon Aquarius version 4.4. Bivariate analyses were then completed to compare each potential frailty metric with outcomes of interest including mortality, injury severity, length of stay, and discharge disposition. Early results indicate that certain radiographic markers may have predictive value for injury severity. For example, severe thoracic injury is associated with cerebral atrophy, cervical spine degeneration, pulmonary bullae formation, osteopenia, and vascular calcification. Severe spinal injury is associated with cervical spine degeneration, osteopenia, and vascular calcification. Further study goals include creating a CT frailty score for patient risk stratification.

This research was supported in part by the Proposed Research Initiated by Students and Mentors (PRISM) Program, University of Maryland School of Medicine Office of Student Research.

O.21

ANTERIOR SEGMENT STRUCTURAL CHANGES IN NORMAL EYES FROM PRE-BIRTH TO AGE 25 YEARS. Gianna Stoleru* and Janet Alexander, Department of Ophthalmology and Visual Sciences, University of Maryland School of Medicine, Baltimore, MD.

The use of ultrasound biomicroscopy (UBM) to characterize the eye has grown in clinical application throughout its use in the last 15 years. UBM can be used to image the cornea, iridocorneal angle, anterior chamber, iris, ciliary body, and lens, encompassing the entirety of anterior segment anatomy. Normative values with UBM technology are not well defined, especially in pediatric patients. This study aims to use UBM technology to define a normative data set of the anterior segment from pre-birth to age 25 years. This was a prospective study in which the UBM ultrasound probe was administered on 8 pediatric patients and 6 young adult patients. Image analysis and data collection were completed utilizing ImageJ software according to an existing protocol developed at the University of Maryland Medical Center. Over 30 parameters were measured from the images collected, including ciliary body and iris measurements. Statistical analysis included frequency distributions, correlation coefficients, mixed effects models and repeated measures analysis. The majority of measured parameters in this study confirm an established trend of exponential growth up until age 1, slowed growth from ages 1 to 5, and very minimal growth from ages 5 to 25. These well-established parameters include anterior chamber width distance (ACWD),

anterior chamber depth (ACD), anterior chamber height (ACH), anterior chamber area (ACA), anterior chamber perimeter (ACP), and angle opening distance (AOD500). The study established a consistent ocular growth trend for a variety of other parameters broadly categorized as pediatric iris and pediatric ciliary body measurements. UBM technology is useful as a tool to image and characterize the anterior segment of the eye, and is unique in its ability to provide images of the position, angulation and anatomical variants of the peripheral iris and ciliary body. This data set could be used for comparison in later anterior segment UBM studies of congenital glaucoma, congenital cataracts, for surgical planning, and to identify anatomical variants related to patient outcome.

O.22

VALUE OF ANCILLARY TESTING AFTER INITIAL DIAGNOSIS OF MILD VENTRICULOMEGALY. Alison Mehlhorn* and Stephen Contag, Department of Obstetrics, Gynecology and Reproductive Sciences, University of Maryland School of Medicine, Baltimore, MD.

Detailed prenatal fetal anatomy ultrasound is routinely performed in the second trimester of pregnancy to identify structural abnormalities. The most common fetal brain anomaly identified during these scans is ventriculomegaly. Following identification of fetal cerebral ventricular dilation, a variety of follow-up tests can be performed including additional imaging with MRI, genetic testing consisting of karyotype, FISH, and microarray analysis and congenital infection screening. Currently there are inconsistent guidelines as to the best ancillary tests to perform. The aim of our study was to investigate the utility of these tests following an initial diagnosis of isolated mild ventriculomegaly. We reviewed 121 cases of mild ventriculomegaly identified at a tertiary prenatal referral center between 2009 and 2015. Of these, 56 demonstrated isolated ventriculomegaly and 65 were classified as having complex ventriculomegaly. A variety of ancillary tests were offered and the utility of each test was analyzed. MRI provided additional information in 3 of the 24 cases of isolated mild ventriculomegaly in which it was used. Genetic testing was performed in 9 cases of isolated mild ventriculomegaly, identifying 4 genetic abnormalities. Finally, serology infection screening was found to be negative for maternal infection in all cases. Based on our results we believe ancillary testing is useful. Although cases of complex ventriculomegaly had higher rates of positive ancillary tests compared to isolated ventriculomegaly, testing is useful in all cases and recommend MRI and genetic testing specifically be performed in cases of isolated mild ventriculomegaly.

O.23

EFFECTS OF QUALITY OF DIAGNOSIS-PROGNOSIS CONVERSATION ON PERCEPTION OF HEALTHCARE PROVIDERS, SATISFACTION WITH CONVERSATION & HRQOL OUTCOMES IN TSCI. Stephanie Golob*, Deborah Stein, Dave Hampton, Frances Grissom, and Karen Irizarry, Department of Surgery, University of Maryland School of Medicine, Baltimore, MD.

There are about 12,500 new cases of traumatic Spinal Cord Injury (tSCI) a year in the United States, and with each one of those cases, the physician or another healthcare provider has to deliver the bad news to the patient. This is a crucial skill for physicians to have, yet most physicians feel they are inadequately trained to give bad news. Although some research has been done in the field of oncology on the best way to deliver bad news, there is a lack of research on this in the field of tSCI. Additionally, few studies have used actual patients in evaluating the best way to deliver bad news, and none have attempted to correlate this with patient outcomes. This study will fill that niche by investigating two separate questions. (1) How does the quality of the Diagnosis Prognosis Conversations (DPCs) that a healthcare provider has with the tSCI survivor, measured on 3 separate

“quality-dimensions”, affect the tSCI survivors’ perceptions of their healthcare providers and levels of satisfaction with the conversation? (2) Does quality of DPCs affect Health-Related Quality of Life (HRQoL) outcomes in tSCI survivors? We will investigate these questions through the use of an open-ended interview, a novel DPC Survey created by the experimenters, and two established HRQoL Surveys (Short Form-36 and Spinal Cord Injury Quality of Life-23.) Primary data analysis will include use of a Student T-Test to compare: (1) mean quality of DPCs with mean perceptions of healthcare providers and levels of satisfaction with conversation, and (2) mean quality of DPCs with mean scores on HRQoL Surveys. We expect to find that higher quality DPCs result in more positive perceptions of healthcare providers, higher levels of satisfaction with conversation, and more positive HRQoL outcomes. Limitations to recruitment include small population size due to strict eligibility criteria. This study has the potential to provide an immediate impact in changing how we deliver bad news to survivors of tSCI and presents the possibility to one day improve medical training in the delivery of bad news.

This research was supported in part by the Proposed Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine, Office of Student Research

O.24

HEALTH RELATED QUALITY OF LIFE AND UTILITY ACQUISITION FOR TRAUMATIC PATELLA DISLOCATION IN ADOLESCENTS. Conan So*, Benedict Nwachukwu, Huong Do, Daniel Green, and Emily Dodwell, Department of Orthopaedics, Hospital for Special Surgery School of Medicine, New York, NY.

Patella instability and dislocation in children/adolescents is a significant cause of morbidity with no clear consensus on the role of surgical management. The purpose of this study was to determine the health related quality of life (HRQoL) associated with various stages of pediatric patella instability and the impact of pre-injury functional status on HRQoL assessment. Patients who had been treated for a first time traumatic patellar dislocation between the ages of 10-18, were identified. Utilities for six patella dislocation health state were obtained from patient (self report) and parent (proxy) interviews using the feeling thermometer method (0-100). Sixty pediatric patients and 58 parents were enrolled. Mean patient and parent age was 14.3 (SD+ 1.7) years and 48.1 (SD+ 3.6) years respectively. The majority of included patients had undergone surgery for patella instability (N=52, 86.7%). Mean patient utilities for Injury, Rehabilitation, Post-surgical, Chronic dislocator, Stable return to lower and same function health states were: 26.1, 45.5, 28.9, 24.4, 62.5 and 94.6 respectively. Parent derived utilities for these health states were 28.5, 42.3, 34.7, 22.0, 54.3 and 97.0. Health state correlation between parent and child pairs was poor/fair with the best correlation demonstrated for injury ($r=0.31$) and stable return to same function ($r=0.43$) health states. Patients with higher pre-injury UCLA activity scores assigned a higher utility to stable return to same function ($p=0.04$); there were no other significant differences based on pre-injury level of function. This study demonstrates that patella instability and dislocation is a significant source of decreased quality of life for adolescents. We found that children with higher pre-injury functional status most value a stable return to same level of function (i.e. successful treatment). There is increasing evidence to demonstrate the superiority of surgical management for traumatic patella dislocation compared to non-operative treatment. These findings suggest that for children with a high pre-injury level of function, surgical treatment to restore prior level of function may provide the greatest HRQoL gain.

This research was supported by the Hospital for Special Surgery Medical Student Summer Research Fellowship.

O.25

INCRETIN-BASED THERAPY IN TYPE 2 DIABETES: AN EVIDENCE BASED SYSTEMATIC REVIEW AND META-ANALYSIS. Greer Waldrop*, Jixin Zhong¹, Matthew Peters¹, Bhramar Mukherjee², and Sanjay Rajagopalan¹, ¹Department of Medicine, University of Maryland School of Medicine, Baltimore, MD and ²Department of Epidemiology and Public Health, University of Michigan School of Public Health, Ann Arbor, MI.

Clinically, incretin based therapies such as dipeptidyl peptidase-4 inhibitors (DPP-4i) and glucagon-like peptide-1 receptor agonists (GLP-1Ra) are rarely used in isolation, but rather in combination with other oral anti-diabetic agents (OAD). Prior meta-analytic reviews do not adequately address the impact of background therapy, comparator arms, which often include a variety of different OAD and within class efficacy on glycemia control. Accordingly, we aimed to further investigate the efficacy of incretin based therapies by updating existing reviews by including clinical trial evidence after 2008; estimating the pooled effect of incretin therapies on glycemic efficacy and weight-loss, stratified by comparator therapy (placebo, mono-therapy, etc.), estimating the impact of background OAD and within class (GLP-1Ra or DPP-4i) comparative efficacy, on glycemia control. 82 randomized controlled trials after 2008 with glycemic control and weight loss as primary end-points were included. Both DPP-4i and GLP-1Ra reduced HbA1c, but only GLP-1Ra caused weight loss when compared to either active comparator drugs or placebo. GLP-1Ra were more effective than DPP-4i in glycemia lowering. Long acting GLP-1Ra were more effective in HbA1c lowering than short-acting agents but with similar weight loss effect. The effect of DPP-4i incretin glycemic efficacy was not modified by background therapy used in the study.

O.26

PERIOPERATIVE MANAGEMENT VARIATION AMONG NEONATES UNDERGOING ARTERIAL SWITCH OPERATION: A CALIFORNIA CONGENITAL CARDIAC CONSORTIUM PILOT STUDY. Orestes Mavrothalassitis* and Tara Karamlou, Division of Pediatric Cardiothoracic Surgery, Department of Surgery, University of California, San Francisco Benioff Children's Hospital School of Medicine, San Francisco, CA.

Neonatal arterial switch operation (ASO) is the standard of care for transposition of the great arteries with intact ventricular septum (TGA/IVS), but little is known about the extent of management variation among centers and how that variation impacts clinical outcomes. Through retrospective analysis of neonates undergoing ASO for TGA/IVS at one center between 2004-2014, variation within perioperative process measures was described. Risk factors for increased total hospital length of stay (TLOS), postoperative length of stay (PLOS), and total inpatient costs (TIC) adjusted to 2014 dollars were analyzed by bivariate and multivariable analysis. Inclusion and exclusion criteria were met by 34 neonates. There were no in-hospital mortalities. The complication rate was 52.9% (20.6% without inclusion of delayed sternal closure reoperations). Median TLOS was 18.5 days (IQR, 16.8-23.5) and median PLOS was 11.0 days (IQR, 10.0-16.0). Median adjusted TIC were \$154,188 (IQR, \$122,060-\$187,605). Median age at operation was 7.0 days (IQR, 5.0-9.3). When compared with patients with ASO on or before day of life six, patients with ASO after day of life six had longer TLOS (22.6 days vs. 16.6 days, $p = 0.004$) and greater adjusted TIC (\$178,783 vs. \$131,385, $p = 0.02$). Total duration of postoperative central venous catheter (CVC) placement, total duration of postoperative intubation, and time to exclusive postoperative PO feeds were significantly correlated with TLOS, PLOS, and adjusted TIC (all $p < 0.01$). Management of neonates undergoing ASO is variable and significantly impacts TLOS, PLOS, and adjusted TIC.

This research was supported by the American Association for Thoracic Surgery Summer Intern Scholarship in Cardiothoracic Surgery

O.27

INCREASING THE KIDNEY DONOR POOL BY EXPANDING LIMITS ON BODY MASS INDEX AND AGE. James Tonascia* and Soo Yi, Division of Transplant, Department of Surgery, University of Maryland School of Medicine, Baltimore, MD.

Utilizing donors with advanced age or elevated body mass index (BMI) could increase the number of live donor kidney transplants. Our aim was to evaluate the safety for both donors and recipients when people with elevated BMI or advanced age are donors. We conducted a retrospective cohort study derived from a case-series review of 393 donor-recipient pairs who underwent kidney transplantation at our center between 2011 and 2015. Donors were followed for up to two years and recipients were followed for up to five. Comparing the high BMI (≥ 30 kg/m², n = 121) and low BMI cohorts, the serum creatinine profiles over the course of follow-up were statistically significantly higher for the high BMI donors ($p = 0.05$), but not clinically significant (mean difference over time = 0.04 mg/dL). No significant difference in the profile of the proportion of donors with a GFR > 60 ml/min between the two BMI cohorts was found ($p = 0.36$). Older donors (Age ≥ 60 , n = 50) did not have a significant difference in the serum creatinine profiles compared to younger donors ($p = 0.37$), but the profile of the proportion of donors with a GFR > 60 ml/min over the course of follow-up was significantly lower ($p < .0001$). Having a donor with high BMI or older age was not associated with graft failure (BMI ≥ 30 : $p=.91$; Age ≥ 60 : $p=.60$) or mortality (BMI ≥ 30 : $p=.96$; Age ≥ 60 : $p=.48$) in recipients. For transplants performed at UMMC, older donors or donors with elevated BMI have safety profiles similar to other donors, and graft function is not impaired in their recipients.

This research was supported in part by the Proposed Research Initiated by Students and Mentors (PRISM) Program, University of Maryland School of Medicine Office of Student Research.

O.28

ACTIVATED MICROGLIA RELEASE PRO-INFLAMMATORY MICRORNAS THROUGH EXTRACELLULAR VESICLES. Niaz Khan*, Alok Kumar, Gelarah Abulwerdi, Bogdan Stoica, and Alan Faden, Department of Anesthesiology, University of Maryland School of Medicine, Baltimore, MD.

Extracellular vesicles (EVs) are important for intercellular signaling and their release is often increased with cellular activation or in pathophysiological states. A key element transported by EVs are microRNAs (miRs), which can post-transcriptionally regulate gene expression. miRs play an important role in the modulation of microglial phenotype. Pro-inflammatory miRs, such as miR-155, are increased in M1-like microglia and potentiate pro-inflammatory responses that may be neurotoxic; on the other hand, anti-inflammatory miRs are increased in M2-like microglia and may provide neuroprotection. It is unknown whether activated microglia release pro- or anti-inflammatory miRs through EVs. Here, we hypothesized that microglial cells increase release of EVs containing pro-inflammatory miRs like miR-155 after an inflammatory challenge (i.e. LPS stimulation) and that these EVs can serve to activate naïve microglia in vitro and in vivo. Cultured BV2 microglia cells were stimulated with LPS for 24 hours. EVs were isolated by centrifugation, and their RNA content was extracted for qPCR analysis. We observed a ~30-fold increase in miR-155 levels in EVs isolated from LPS-stimulated microglia (LPS-EVs) relative to those from unstimulated microglia (control EVs). In the next set of experiments, naïve BV2 microglia were incubated with EVs from LPS-stimulated or control cultures for 24 hours. Analysis by qPCR demonstrated a ~2.5-fold increase in miR-155 levels as well as significant increases in mRNA levels of inflammatory activation markers in cells stimulated by LPS-EVs compared to control EVs. Finally, in vivo cortical injection of LPS-EVs demonstrated increased neuroinflammation in the brain relative to control EVs as shown by immunohistochemical staining for activated microglia. Together, our results

demonstrate that microglia can secrete EVs containing pro-inflammatory miRs that can activate naïve microglia. This pathway may be a mechanism for pro-inflammatory microglial seeding and could be a therapeutic target for conditions where neuroinflammation contributes to pathology.

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Poster Presentation Abstracts

Presenters are indicated with “*” next to their names.

P.01

ANALYSIS OF GAIL MODEL FOR BREAST CANCER RISK ASSESSMENT WITH GP-88 BLOOD TESTING. Erica Cranston*, Nancy Tait¹, Ginette Serrero², David Hicks², and Katherine Tkaczuk³, ¹Breast Evaluation and Treatment Program, Marlene and Stewart Greenebaum Comprehensive Cancer Center and ³Department of Medicine, University of Maryland School of Medicine, Baltimore, MD and ²A&G Pharmaceutical, Inc., Columbia, MD.

Glycoprotein-88 (GP-88) is a PC-cell derived growth factor that has been shown to increase tumorigenesis, survival, and proliferation of breast cancer (BC) cells. GP-88 is expressed on the cell surface of breast tumors and can be detected in the circulation. GP-88 is elevated in women with BC but remains in a normal range in healthy individuals. The highly sensitive Enzyme Immunoassay EIA can measure GP-88 in serum and normal GP-88 levels are considered to be < 50 ng/ml. The purpose of this study was to assess the association of baseline GP-88 levels with the Gail model risk of developing BC in 5 years and lifetime. Individual risk factors were collected, including age, race/ethnicity, family history of BC, age of first menstrual period, age of first live birth, history of breast biopsies, breast tissue density, and body mass index (BMI), and utilized in Gail model calculations. Patient characteristics include 415 female subjects, median age of 53 (range 40-87), with 140 of the 415 subjects considered high-risk based on a 5-year predicted BC risk of > 1.6% by Gail model. Preliminary regression data analysis using a significance level of $p = .05$ found a significant positive relationship between Gail model predicted 5-year risk of developing BC and baseline GP-88 levels, as well as age and GP-88 levels. High-risk subjects identified by Gail model also had a significantly higher baseline GP-88 level. Further studies will include multivariate analyses looking at known BC risk factors including race, BMI, and breast tissue density.

This research was supported by the Maryland Industrial Partnerships Grant (MIPS) and by the Proposed Research Initiated by Students and Mentors (PRISM) Program, University of Maryland School of Medicine Office of Student Research.

P.02

ROLE OF P38 MAPK IN THE PRO-APOPTOTIC ACTIVITY OF ARTEMISININS IN ACUTE LEUKEMIAS. Taylor Rosenbaum*, Curt Civin, Xiaochun Chen, and Blake Moses, Division of Physiology, Department of Pediatrics, University of Maryland School of Medicine, University of Maryland School of Medicine, , Baltimore, MD.

Leukemia continues to be a deadly disease. The artemisinins are a class of drugs widely used in the treatment for severe malaria due to their potency and few adverse effects. In addition to their antimalarial activity, artemisinins have also been shown to have potent anti-cancer activity, especially for leukemia. In vitro and in vivo studies have demonstrated that the established artemisinin, Artesunate (AS), and a new dimeric artemisinin derivative, ART-838, are promising agents to repurpose for leukemia treatment, particularly in combination with BCL-2 inhibitors (i.e. ABT-199). A previous study on dihydroartemisinin (DHA, the active metabolite of AS in vivo) in liver cancer cell lines reported DHA-associated downregulation of MCL-1, a proapoptotic protein responsible for drug resistance to BCL-2 inhibitors. Since AS was reported to induce phosphorylated P38 mitogen-activated protein kinase (p38 MAPK) due to reactive oxygen species (ROS) production in embryonal rhabdomyosarcoma (ERMS) cells and p38 MAPK is known to downregulate MCL-1 in BCL-2 inhibitor-resistant chronic lymphocytic leukemia (CLL) cell lines, I therefore hypothesize activation of a ROS-p38 MAPK-MCL-1 signaling pathway is responsible for the synergy that the

Civin lab has found between artemisinins and ABT-199 in leukemia cell lines. In this study, I explored the specific role p38 MAPK has in AS and ART-838 -induced apoptosis, to help elucidate the molecular mechanisms underlying artemisinin action in order to better understand its utility for leukemia treatment.

This research was supported in part by the Proposed Research Initiated by Students and Mentors (PRISM) Program, University of Maryland School of Medicine Office of Student Research and by Dr. Curt Civin.

P.03

THE ROLE OF SUR1-TRPM4 IN GLIOBLASTOMA MIGRATION. Jonathan Na*, Jesse Stokum, Volodymyr Gerzanich, and J. Marc Simard, Department of Neurosurgery, University of Maryland School of Medicine, Baltimore, MD.

Glioblastoma Multiforme (GBM) is a highly invasive and aggressive astrocytic primary brain neoplasm, accounting for approximately 13,000 deaths and for 18,000 new cases annually in the US. (American Brain Tumor Association, 2014). Current therapies for this disease only include surgical resection and chemotherapy, yet post-op recurrence is near, resulting in a median patient survival of approximately 15 months. The sulfonylurea receptor 1 (Sur1)-Transient receptor potential 4 (Trpm4) channel is an ATP- and calcium-sensitive cation channel previously associated with focal cerebral ischemia and subsequent malignant edema. Experiments were designed to examine the presence of the Sur1-Trpm4 channel in glioblastoma and a possible role in migration. To assess the expression of Sur1-Trpm4 in GBM, immunohistochemistry (IHC) was conducted on tissues from the rat RCAS-Tva GBM model. To further validate the role of Sur1-Trpm4 in GBM migration, the U87 and KR158b cell lines were utilized. qPCR was used to assess mRNA expression of Sur1 and Trpm4. Additionally, the levels of Sur1 and Trpm4 protein in these cell lines were determined through immunoprecipitation and immunoblot experiments. To determine the role of Sur1 and Trpm4 in GBM migration, migration analyses using the xCelligence System were performed, using DMEM with 10% fetal bovine serum as a chemoattractant. 9-Phenanthrol, glibenclamide, were used to attenuate Trpm4 and Sur1 activity, respectively. IHC, qPCR, IP, and immunoblot experiments indicate that the Sur1-Trpm4 is expressed in the RCAS-Tva GBM rat model and the KR158b cell line, but not the U87 cell line. Migration experiments indicate that inhibition of Trpm4 with 9-phenanthrol attenuates migration of tumor cells. The present data suggest a role for Sur1-Trpm4 in the migration of glioblastoma cells. However, further investigation is needed to determine the mechanism of this channel in migration in order to illuminate the potential for Sur1-Trpm4 as a therapeutic target for glioblastoma.

This research was supported in part by the Proposed Research Initiated by Students and Mentors (PRISM) Program, University of Maryland School of Medicine Office of Student Research.

P.04

THE ROLE OF E3 LIGASE HECW2 IN CARDIAC STRUCTURAL REMODELING PRECEDING ATRIAL FIBRILLATION. Antanina Voit* and Gopal Babu, Department of Microbiology and Immunology, Rutgers New Jersey Medical School School of Medicine, , Newark, NJ.

Atrial fibrillation (AF), the most common tachyarrhythmia increases with risk of stroke, diabetes, heart failure, aging and genetic disorders or can be idiopathic. AF is characterized by changes in ion channel function and atrial structure. Sarcoplipin (SLN) is an atrial specific regulator of SR Ca²⁺ ATPase (SERCA) and plays an important role in atrial Ca²⁺ homeostasis. We find that the transgenic expression of SLNT5A in mouse atria results in decreased SR Ca²⁺ content and elevated levels of diastolic Ca²⁺ as reported in human AF. In SLNT5A mouse model, the atrial structural

remodeling is progressive and leads to AF. Our preliminary studies also demonstrate the activation of ubiquitin-proteasome system (UPS) in atria of both human AF and SLNT5A TG mice. The UPS is responsible for the turnover of many different cellular proteins and plays major role in a variety of cellular processes under normal and during pathology. Recent studies have shown that the proteasome activity increases in a context of cardiac overload, and that blocking such activation decreases the left ventricular remodeling and preserve the cardiac function. Our preliminary data therefore suggest that UPS activation in AF may have direct relevance for atrial structural remodeling. Restoration of atrial structure and electrical conductivity in the SLNT5A TG mice upon proteasome inhibition further supports this notion. In addition, we find selective upregulation of a novel E3 ligase, Hecw2 in atria of both human AF and SLNT5A TG mice. Co-immunoprecipitation studies indicate that Hecw2 interacts with sarcomeric α -actin and α -tropomyosin. These results suggest that Hecw2 overexpression in AF may facilitate the degradation of contractile proteins through UPS and contributes to myolysis.

This research was supported by funds from Department of Cell Biology and Molecular Medicine, Rutgers - New Jersey Medical School.

P.05

CHARACTERIZATION OF HYPOTHALAMIC-PITUITARY-ADRENAL (HPA) ABNORMALITIES IN PATIENTS WITH POSTURAL ORTHOSTATIC TACHYCARDIA SYNDROME (POTS). Lauren Rosso*, Mansur Shomali¹, and Ramesh Khurana², ¹Department of Medicine, University of Maryland School of Medicine and ²Department of Neurology, MedStar Union Memorial Hospital, Baltimore, MD.

Postural orthostatic tachycardia syndrome (POTS) is a complex and often debilitating medical condition in which individuals experience orthostatic intolerance and an increased heart rate upon standing. Clinical observations have suggested that previously unreported neuroendocrine abnormalities may be associated with POTS. In a series of 32 patients, 19 demonstrated adrenal insufficiency either through inadequate cortisol response to cosyntropin stimulation or insulin-induced hypoglycemia. In addition, 6 out of 9 patients were observed to have inadequate GH secretion in response to insulin-induced hypoglycemia. In this series of patients, 10 individuals who were treated with physiologic doses of hydrocortisone and underwent repeated tilt table testing, experienced a significant reduction in the orthostatic heart rate. These observations suggest that overarching central neuroendocrine defects may contribute to the pathophysiology of a proportion of individuals with POTS.

P.06

DEFINING A SAFE DURATION OF INPATIENT OBSERVATION FOR RESOLUTION OF BRADYCARDIC EVENTS IN PREMATURE NEONATES. Lauren Cosgriff* and Natalie Davis, Division of Neonatology, Department of Pediatrics, University of Maryland School of Medicine, Baltimore, MD.

Apnea of Prematurity (AOP) is a common diagnosis in the Neonatal Intensive Care Unit (NICU) characterized by idiopathic apneic (cessation of breathing for >20 seconds) and bradycardic (heart rate <80bpm) episodes with variable time to resolution. Typically more severe events requiring intervention resolve first, followed by bradycardic events of lessening frequency. Pending self-resolution of these events infants are monitored for heart rate, respiratory rate, and oxygen saturation levels until they demonstrate adequate maturation of their respiratory control system. Although most NICU facilities have guidelines in place defining a bradycardia free period, this observation time ranges widely and reflects the lack of literature available to guide clinicians on appropriate duration of observation. The aim of this study was to define a safe period of inpatient

observation for resolution of bradycardic episodes. We performed a retrospective medical record review of 62 infants born in 2013/14 at <37 weeks gestational age (GA) who were treated with caffeine for AOP. We documented timing and severity of apneic and bradycardic events as well as demographic and clinical risk factors. Of the 62 infants included in the study, 33 were female, the mean birth weight was 1252±444 grams, and the mean GA was 29±2.6 weeks. We then calculated the intervals between the infants' last three bradycardic events, and found a median of 4 days (IQR=5) with a wide range of 1-28 days. Infants in the upper quartile with the longest interval to resolution (≥ 7 days) had significantly younger gestational ages at birth ($p=0.0065$), smaller birth weights ($p=0.0018$), and were more likely to be African American ($p=.0254$). Those in the upper quartile were also more likely to have required surfactant ($p=0.0071$), ventilator treatment ($p=0.0231$), have chronic lung disease ($p=0.0202$), and be older at the time of discharge ($p=0.0250$). We conclude that infants born most premature are at highest risk for prolonged bradycardic events. We are performing ongoing data collection and analysis to identify further clinical risk factors for prolonged bradycardic events.

This research was supported in part by the Proposed Research Initiated by Students and Mentors (PRISM) Program, University of Maryland School of Medicine Office of Student Research.

P.07

ASSOCIATION BETWEEN PRE-ECLAMPSIA AND CONGENITAL HEART DEFECTS.
Breanne Bears*, Sarah Crimmins, Ozhan Turan, and Shifa Turan, Division of Maternal Fetal Medicine, Department of Obstetrics, Gynecology and Reproductive Sciences, University of Maryland School of Medicine, Baltimore, MD.

Emerging research supports a common dysfunctional angiogenic mechanism for fetal impacts in the form of congenital cardiovascular disease including congenital heart defects (CHD) and maternal impacts in the form of preeclampsia/eclampsia (PE.) Fetuses diagnosed with CHD, came from a referred high risk population, occasionally from general antenatal evaluation, assessed by the Fetal Heart Program at UMMC. Cases and controls were identified retrospectively from our ultrasound database, and those with known chromosomal or non-CHD anomalies were excluded. PE was diagnosed according to standard guidelines (ACOG 2013) and categorized as early (delivery <34 weeks) or late. Contributors to early and late PE were assessed and compared with controls (no CHD, no chromosomal and structural abnormalities from the same database) using binary logistics regression and Chi-square analysis. Of 2856 meeting inclusion criteria, 215 (7.5%) had CHD. PE occurred in 182 (6.4%) cases, early PE in 28 (1%) and late PE in 153 (5.4%). CHD cases had no increased risk for early PE (OR:2.07 CI:0.71-6.01) or late PE (OR:0.85 CI:0.44-1.64) compared to controls. In the CHD group, developing late PE was significantly increased by diabetes (OR:7 (1.9-26) $p=0.0005$), or chronic hypertension (CHT) (OR:15 (3.7-63) $p< 0.0001$). In the CHD group, chronic hypertension was the major determinant of late PE (Logistic regression $R^2=0.23$ $p<0.0001$). In those without CHD, late PE was not increased by diabetes (OR:1.1.4 CI: 0.9-0.1) $p=0.11$ or CHT(OR: 1.4 CI: 0.79-2.32 $p=0.26$). No maternal characteristics, medical co-morbidities, or obstetric data significantly correlated with early PE in either cohort. CHT was the major determinant of late PE in CHD group ($R^2=$ $p<0.0001$). Considering those with CHT, CHD was a significant determinate for late preeclampsia (OR:3.43 CI:1.3-9.1 $p=0.001$.) Fetal CHD did not by itself predict an increased risk of pre-eclampsia. However, in women with CHT, when there is evidence of dysfunctional fetal angiogenesis (in the form of CHD), the risk of preeclampsia is greater. Enhanced surveillance and perhaps treatment appear warranted in these women.

This research was supported in part by the Proposed Research Initiated by Students and Mentors (PRISM) Program, University of Maryland School of Medicine Office of Student Research.

P.08

DENDRITIC SPINE DENSITY OF MEDIUM SPINY NEURONS IN THE NUCLEUS ACCUMBENS IN A MOUSE MODEL OF DEPRESSION. Stefanie Zaner*, Tara LeGates, and Scott Thompson, Department of Physiology, University of Maryland School of Medicine, Baltimore, MD.

The nucleus accumbens (NAc) is a key brain structure involved in the reward pathway and is associated with all reward behaviors. Although much research has been done implicating the role of the NAc in depression, the specific underlying neural circuitry remains unclear. In the NAc the main neurons are medium spiny neurons (MSNs). There are two subtypes of MSNs, those expressing the dopamine receptor D1 and those expressing D2. Proper balance of these two subtypes of MSNs is important for mood regulation and is thought to be dysfunctional in depression. Chronic stress, a common factor precipitating depressive episodes weakens excitatory synaptic strength in many brain regions including the NAc. Furthermore, this weakening occurs in parallel to the changes in reward related behaviors. Previous work has shown that D1 expressing MSNs are weakened by chronic stress, while D2 expressing MSNs are not, however the mechanism by which this weakening is occurring is unknown. One possible explanation for this finding would be if there is selective atrophy of D1 expressing MSN dendrites. I investigated the hypothesis that depression-induced weakening of NAc synapses are the result of a reduction of dendritic spine density in D1, but not D2 expressing MSNs. To do this I used chronic multi-modal stress (CMS) to induce a depressive-like state in mice as measured by sucrose preference testing (a commonly used assay to measure anhedonia in rodents). I then used confocal light microscopy to quantify spine density of D1 vs. D2 containing MSNs in the NAc. Although my results were not significant, I found a trend suggesting a decrease in spine density of D1, but not D2 expressing MSNs in the NAc.

This research was supported in part by the Proposed Research Initiated by Students and Mentors (PRISM) Program, University of Maryland School of Medicine Office of Student Research.

P.09

EVALUATING PERI-ICTAL MOOD CHANGES IN PATIENTS WITH EPILEPSY. Sharon Ong*, Jennifer Hopp¹, Scott Thompson¹, Mark Kvata², and Erin Lanzo², ¹Department of Neurology, ²University of Maryland School of Medicine, Baltimore, MD.

Depression and anxiety are among the most common comorbid conditions associated with epilepsy. While the occurrence of these conditions has been studied in the post-ictal and inter-ictal periods, little is understood about mood changes in the peri-ictal period. We hypothesize that patients with baseline depressive moods will show improvements in mood in the peri-ictal period. We also hypothesize that patients with focal onset seizures are more likely to have mood improvements in the peri-ictal period than those with generalized onset seizures. We studied peri-ictal mood changes in the Epilepsy Monitoring Unit (EMU) at the University of Maryland Medical Center. We administer the Beck Depression Inventory and the Beck Anxiety Inventory to patients in the EMU to measure symptoms of depression and anxiety prior to a seizure and at 4 intervals post-ictally. Our data show that epileptic patients show improvement in both mood and anxiety post-ictally. There was significant difference in 4-hour average BDI scores from baseline ($p=0.0086$) and 12-hour BAI scores ($p=0.0152$). We also saw a significant average maximal change over 24 hours in focal and generalized onset epileptic patients ($p=0.02$), and significant anxiety improvement in focal onset patients ($p=0.04$).

P.10

CHARACTERIZATION OF DEXAMETHASONE USE FOR DELAYED CEREBRAL EDEMA IN ACUTE PRIMARY INTRACEREBRAL HEMORRHAGE. Richa Manglorkar* and Wendy Wan-Tsu Chang, Department of Neurocritical Care, University of Maryland School of Medicine, Baltimore, MD.

Management of patients with acute primary intracerebral hemorrhage (ICH) is focused on reduction of secondary brain injury from the mechanical and cellular effects of the hematoma and associated edema. Hemoglobin and its breakdown products have been demonstrated to be neurotoxic, exacerbate acute perihematoma edema, and contribute to disruption of the blood-brain barrier. This suggests that there are components of cytotoxic edema related to cellular death as well as vasogenic edema related to disruption of the blood-brain barrier that contribute to the development of perihematoma edema seen with ICH. Perihematoma edema develops early after ICH and increases within 7 to 11 days after ictus. Corticosteroid such as dexamethasone is commonly used for treatment of vasogenic edema associated with brain tumors with great success. Early studies in the 1970s and 1980s on the use of steroids in ICH showed no benefit in outcomes, instead, increased infectious complications. However, there have been no studies since that examine in detail the time window, dose, and duration of steroid treatment and its effect on perihematoma edema and associated outcomes. There exists a knowledge gap between the pathophysiology of delayed cerebral edema with acute primary ICH and potential treatment options. We aim to characterize the use of dexamethasone for delayed cerebral edema and its potential effects on outcome.

This research was supported in part by the Proposed Research Initiated by Students and Mentors (PRISM) Program, Office of Student Research, University of Maryland School of Medicine.

P.11

POSTERIOR PARIETAL-MOTOR CORTEX PLASTICITY IN WRITER'S CRAMP. Mary Maldarelli*, Sophie Cho, Pattamon Panyakaew, and Mark Hallett, National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, MD.

Focal dystonia is a neurological disorder characterized by excessive muscle contraction, and has been noted to have three main abnormalities: loss of inhibition, sensory dysfunction, and plasticity derangement. Plasticity derangement and altered neuronal circuits have been of particular interest for study to determine the etiology of focal task specific dystonia. Transcranial Magnetic Stimulation (TMS) is a technique used in both the United States and Europe for neurological study and treatment of depression. Paired Association Stimulation (PAS) with TMS allows for study of different connections between sections of the brain. It has been previously established that using TMS PAS, Long Term Potentiation (LTP)-like or Long Term Depression (LTD)-like plasticity can be observed in neuronal circuits. In this study, we examined the possibility of plasticity within posterior parietal (PP)-motor cortex (M1) connections utilizing TMS, an established plasticity intervention strategy, and PAS to determine connection strength. Evoked potentials were collected from the first dorsal interosseous muscle of writer's cramp focal dystonia patients and normal subjects. We were able to identify LTP like plasticity between PP and M1 in writer's cramp patients after intervention, as compared to an observed LTD like plasticity between PP and M1 in normal subjects.

P.12

INVESTIGATING EARLY EVIDENCE OF MULTIPLE SCLEROSIS IN A PROSPECTIVE STUDY OF HIGH-RISK FAMILY MEMBERS. Sonya Steele*, Zongqi Xia¹, Anshika Bakshi², Philip De Jager¹, and Daniel S. Reich², ¹Program in Translational Neuropsychiatric Genomics and Partners Multiple Sclerosis Center, Ann Romney Center for Neurologic Diseases, Department of

Neurology, Brigham and Women's Hospital, Boston, MA, and ²Division of Neuroimmunology and Neurovirology, National Institute of Neurological Diseases and Stroke, National Institutes of Health, Bethesda, MD.

The Genes and Environment in Multiple Sclerosis (GEMS) study is a multiple sclerosis (MS) inception cohort with a prospective design, which investigates factors that increase a person's risk of developing the disease. The aim is to assess the prevalence of brain MRI and clinical abnormalities consistent with demyelination so as to identify and validate predictive biomarkers in populations at risk for MS. Subclinical demyelinating lesions and subtle clinical findings may precede symptom onset. The GEMS participants with ≥ 1 first-degree relative(s) with MS were assigned a genetic and environmental risk score (GERS) based on targeted genotyping of validated risk variants and questionnaires covering known environmental risk factors. Individuals in the top and bottom 10% of the GERS distribution underwent 3-tesla brain MRI with gadolinium. They were also evaluated with standard neurological disability scales and by assessment of vibration perception with 128 Hz and Rydel tuning forks and the Vibratron II device. The primary outcome was lesions on T2 images meeting 2010 MRI criteria for dissemination in space (DIS). Our cohort comprised 41 individuals in the high-risk group (40 women) and 59 in the low-risk group (25 women) with a mean age of 35.1 years (standard deviation: 8.7). To meaningfully compare the two risk groups, we focused our analysis on the female GEMS participants. Five individuals (8%) met the primary outcome, of whom four were in the high-risk group ($p=0.2$). Leptomeningeal contrast enhancement was detected in four cases (11%), three of which were in the high-risk group. Participants in the high-risk group had poorer lower extremity vibration sensitivity by all three tests, most notably Vibratron II ($p=6.1 \times 10^{-5}$). Our results suggest subtle subclinical demyelination, manifesting most clearly as differences in vibration sensitivity, in neurologically asymptomatic family members. The detection of clinically meaningful signs and neuroimaging findings has important implications, as it may pave the way for understanding the earliest events in the disease and potentially preventing the subsequent accumulation of neurological disability.

This project was funded through a grant from the Foundation of the Consortium of Multiple Sclerosis Centers' MS Workforce of the Future program.

P.13

COMPARISON OF KINEMATIC AND CLINICAL MEASURES OF ARM MOVEMENT IN MULTIPLE SCLEROSIS AND OTHER NEUROLOGICAL DISORDERS. Mindy Chen*, Susan Conroy¹, Anindo Roy², Jeremy Rietschel³, and Christopher Bever², ¹Department of Physical Therapy and Rehabilitation Science and ²Department of Neurology, University of Maryland School of Medicine, Baltimore, MD and ³University of Maryland, College Park, College Park, MD.

Movement disorders, such as Multiple Sclerosis (MS), are neurologic syndromes that affect voluntary and automatic movements. Recent studies have revealed that upper extremity (UE) dysfunction in MS patients is widely under-recognized even though a high percentage those patients have some UE dysfunction in the early stages. Loss of function in the UEs leads to increased dependency and reduces quality of life. Early diagnosis of movement disorders leads to earlier treatment, which may prevent further progression of the disease. Clinical measures like the Jebsen Hand Function Test (JHFT) and 9-Hole Peg Test (NHP) are currently the standard for assessing a patient's arm and hand function. However, these measurements are very time-consuming and are susceptible to reliability errors. Unlike clinical tests, kinematic measurements made by rehabilitation robots have been found to be highly repeatable. InMotion2 is a neurorehabilitation robot that guides a patient's UE to deliver therapy (Hogan & Krebs, 1998). Robotic measures from InMotion2 have been correlated to clinical evaluations of patients with ischemic stroke (Krebs et. al, 2014). The current study was a non-randomized, quantitative observational investigation of motor control

examining arm movements in twenty-eight healthy and neurologically impaired subjects. The goal was to determine if robot-derived performance metrics are more sensitive in distinguishing movement features unique to certain neurologic pathologies than currently used clinical tests. Ten age-matched healthy, five multiple sclerosis, three Parkinson's disease, and ten subcortical stroke subjects performed point-to-point and circle drawing movements using InMotion2. Several kinematic metrics were derived from those tasks. Subjects also completed JHFT and NHP clinical tests. Statistical analysis revealed that all kinematic metrics were able to distinguish between those with and without neurologic disease. Smoothness ($U=7, p=0.027$) and path error ($U=6.5, p=0.023$) differed significantly between MS and stroke subjects. Principal Component Analysis revealed that a factor related to quality of movement was able to discern patients with subcortical stroke from those with other diseases. While kinematic and clinical metrics were correlated, clinical tests were only able to distinguish between healthy and disease groups. No statistically significant differences were found for any of the clinical measurements across groups. This preliminary study suggests that robotic measures may serve as a cheaper and more sensitive alternative to assess a patient's UE motor function.

This project was funded through a grant from the Foundation of the Consortium of Multiple Sclerosis Centers' MS Workforce of the Future program.

P.14

PHOSPHORYLATED SIRT1 AS A BIOMARKER OF RELAPSE AND RESPONSE TO TREATMENT WITH GLATIRAMER ACETATE IN MULTIPLE SCLEROSIS. Jonathan Ciriello*, Alexandru Tatomir, Dallas Boodhoo, and Horea Rus, Department of Neurology, University of Maryland School of Medicine, Baltimore, MD.

SIRT 1 is a class III NAD-dependent histone and protein deacetylase that can induce epigenetic gene silencing and modulate cell survival. We have previously shown that the SIRT1 is expressed by peripheral blood mononuclear cells (PBMCs) and found that SIRT1 expression was significantly lower in relapsing MS patients compared to those in remission. Our goal was to longitudinally investigate the role of active, phosphorylated SIRT1 (p-SIRT1) as a potential biomarker of relapse and predictor for response to glatiramer acetate (GA) treatment in patients with relapsing remitting MS. We also want to investigate the downstream effects of SIRT1 by measuring the level of trimethylation of histone 3 lysine 9 (H3K9me3). A cohort of 15 GA-treated patients was clinically monitored using the Expanded Disability Status Scale and PBMCs were collected at 0, 3, 6, and 12 months after initiation of the therapy. P-SIRT1 and H3K9me3 levels were assayed by western blot using specific antibodies. Statistically significant lower levels of p-SIRT1 protein ($p=0.0047$) and H3K9me3 ($p=0.0013$) were found during relapses when compared to stable MS patients. Non-responders to GA treatment were defined as patients who exhibited at least two relapses following initiation of GA treatment. Statistically significant lower levels of p-SIRT1 protein ($p=0.0289$) was found in GA non-responders compared to responders. We did not find a significant difference in H3K9me3 ($p=0.093$) levels between GA responders and non-responders. Receiver operating characteristic (ROC) analysis was used to assess the predictive power of p-SIRT1 and H3K9me3 as possible biomarkers of relapse and response to treatment. Area under the curve for ROC analysis for p-SIRT1 was 0.708 ($p=0.0225$) and for H3K9me3 was 0.85 ($p=0.0045$) for prediction of relapse. Area under the curve for ROC analysis of H3K9me3 was 0.755 ($P=0.0174$) for prediction of responsiveness to GA treatment. Our data suggest that p-SIRT1 and H3K9me3 could serve as potential biomarkers for MS relapse. Our data also suggest that H3K9me3 could serve as possible biomarker to predict response to GA treatment.

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P.15

OPTIMIZED GENE PROFILING METHODS PROVIDE INSIGHT INTO THE MECHANISM OF ORGAN REJECTION IN CYNOMOLGUS MONKEY CARDIAC ALLOGRAFT RECIPIENTS. Emily Bergbower*, Agnes Azimzadeh, and Richard Pierson, III, Department of Surgery, University of Maryland School of Medicine, Baltimore, MD.

Organ transplant rejection is significant issue to the field of transplantation, and the molecular mechanisms that govern this process are incompletely understood, particularly in the context of novel immunomodulatory drugs. Here, we profiled a specific array of genes by RT-qPCR to determine if gene expression levels of T cell subset markers, B cell markers and costimulatory pathway molecules are associated with rejecting vs. nonrejecting cardiac allografts in monkeys treated with various immunomodulatory approaches. Specifically, we have shown that treatment of heart allograft recipients with FR104 (a selective, non-activating CD28 blocking antibody) or Belatacept (which blocks CD28's ligands, CD80 and CD86), both are effective to prevent acute rejection, but are associated with differing gene expression profiles in sampled heart tissue over time after transplant. Interestingly, prematurely rejected tissues displayed unique genetic profiles compared to cardiac tissues from hearts that did not reject in the same treatment group. Finally, we identified optimal methods for isolating and amplifying RNA from difficult cardiac tissue samples while comparing techniques for quantification of transcripts in graft tissue (standard curve, the traditional $\Delta\Delta C_t$ method, as well as the more novel Nanostring technique). Our study identifies candidate specific immune cell "biomarkers" likely to accurately predict premature rejection in cardiac allograft tissue under novel immunomodulatory treatment regimens, and illustrates that improved RNA isolation and newer gene profiling methods can optimize data acquisition.

This research was funded by the American Association of Thoracic Surgeons through the Summer Intern Scholarship.

P.16

THE EFFECT OF *PEAR1* RS12041331 GENOTYPE ON CIRCULATING BIOMARKERS OF ENDOTHELIAL FUNCTION IN THE CONTEXT OF ASPIRIN THERAPY. Varun Ayyaswami*, Joshua Backman¹, Richard Horenstein¹, Keith Tanner¹, and Joshua Lewis², ²Division of Endocrinology, Diabetes, and Nutrition, ¹Department of Medicine, University of Maryland School of Medicine, Baltimore, MD.

Platelet endothelial aggregation receptor 1 (*PEAR1*) is a transmembrane receptor that is highly expressed in both endothelial cells and platelets. Previous investigations demonstrated that genetic variation in *PEAR1* (i.e. rs12041331) significantly influences platelet- and endothelial-related phenotypes including on-aspirin platelet aggregation, endothelial cell (EC) adhesion, and risk of experiencing recurrent cardiovascular events. Thus, we hypothesized that carriers of the rs12041331 minor allele may have altered levels of endothelial adhesion biomarkers and that aspirin administration would modify the relationship between genotype and biomarker level. We measured levels of circulating biomarkers of endothelial adhesion (vascular cell adhesion molecule-1 [VCAM-1] and intercellular adhesion molecule-1 [ICAM-1]) by rs12041331 genotype pre- and post-aspirin administration (324 mg/d for 7d) in serum samples from 70 healthy Amish subjects. In addition, using Western blot analysis, we assessed *PEAR1* expression by rs12041331 genotype in human umbilical vein endothelial cells (HUVECs). Before and after aspirin administration, we observed no evidence of association between *PEAR1* rs12041331 and levels of either VCAM-1 ($P = 0.24$ and

0.15, respectively) or ICAM-1 (P = 0.62 and 0.68, respectively). Interestingly, we observed that rs12041331 minor allele homozygotes had a non-significant reduction in VCAM-1 levels (-6.6 ng/ml \pm 13.1) and an increase in ICAM-1 levels (16.9 ng/ml \pm 13.8) following aspirin administration that was not observed in other groups. Western blot analysis revealed that the *PEAR1* rs12041331 minor allele resulted in a trending reduction in PEAR1 expression. While no significant differences were detected in biomarkers by *PEAR1* rs12041331 genotype pre- and post-aspirin administration or in endothelial expression by *PEAR1* rs12041331, nonsignificant trends suggest that the statistical power of the study was insufficient. We suggest future studies repeat these experiments with greater sample size. Moreover, future studies should examine endothelial-derived biomarkers that may be more significantly affected by *PEAR1* during aspirin therapy.

This study was supported by National Institutes of Health grants NIH K23 GM102678, U01 GM074518, and U01 HL105198.

P.17

MANIPULATION OF HUMAN ERYTHROPOIESIS VIA RAB GTPASE 5. Ariel Siegel*, Min Jung Kim¹, and Curt Civin², Department of Pediatrics, University of Maryland School of Medicine, Baltimore, MD.

The generation of blood transfusion products in ex-vivo cultures from CD34+ hematopoietic stem progenitor cells (HSPCs) has been investigated as an alternative therapy to improve transplantation and transfusion. However, the clinical utility of these products has been limited due to poor yields and high costs of current inefficient ex-vivo methodologies. Previous experiments have shown that erythroid-expressed microRNAs (miR), miR-144 and miR-451, enhanced erythropoiesis in human CD34+ HSPCs by targeting the expression of RAB GTPase 14 (RAB14). In contrast to the inhibitory role of RAB14 in erythropoiesis, the Civin laboratory found that RAB5 protein expression levels increased during erythropoiesis. The RAB proteins are members of the Ras-related superfamily of small membrane-bound GTPases that play key roles in intracellular trafficking, receptor recycling and signal transduction. Based on this previous work, we anticipated that RAB5C, an isoform of RAB5, acts as a positive regulator of erythropoiesis. However, through overexpression of different RAB5C lentivector constructs in TF1 cells, a human erythroleukemia cell line, we unexpectedly found reduced erythroid differentiation. Further research may better elucidate the mechanisms underlying the induction of erythroid differentiation, a pathway with future therapeutic implications.

This research was supported by the American Society of Hematology HONORS Award.

P.18

TOLEROGENIC POTENTIAL AND EFFICACY OF COSTIMULATORY PATHWAY BLOCKADE IN PRESERVING ADAPTIVE IMMUNITY FUNCTION. Subhashree Nayak*, Marquise Singleterry¹, Elizabeth Kang², Agnes Azimzadeh², and Richard Pierson, III², ¹University of Maryland, College Park, College Park, MD and ²Department of Surgery, University of Maryland School of Medicine, Baltimore, MD.

A prominent modality of chronic rejection in cardiac allograft transplant recipients is host T cell-mediated immune injury and induction of cardiac allograft vasculopathy. Inflammatory responses that reduce graft survival require T cell activation, a process contingent upon multiple, simultaneous receptor-ligand interactions between T cells and antigen presenting cells (APCs) such as the major histocompatibility complex – T cell Receptor interaction, as well as several costimulatory pathways such as the CD40 (APC)/CD40L (T cell) and CD28 (APC)/B7 (T cell) receptor-ligand interactions. The Pierson/Azimzadeh lab has shown that in a monkey model undergoing anti-CD40L therapy, graft survival is dramatically increased. However, initial translation of this therapy in patients was

halted due to thrombotic complications. Thus, this project aims to target the CD40 receptor on APCs via an anti-CD40 monoclonal antibody therapy (2C10R4) and measure its effects in inducing graft tolerance and maintaining adaptive immune function in cardiac allograft recipients. Additionally, there is therapeutic potential in modulation of the CD28/B7 costimulatory pathway. Belatacept, a clinically used fusion-protein based therapy is a B7 blocker that simultaneously blocks CD28 and CTLA-4 (T cell ligands) interactions with B7 (APC), has been shown to be less toxic than CsA (a classical immunosuppressive drug) in transplant patients. However, it was associated with a higher risk of acute rejection. In order to preserve CTLA-4/B7 interactions (a down-regulator of T cell activation), it is postulated that selectively blocking CD28 (via FR104, an mAb therapy) will be a more efficacious in blocking T cell activation than broadly targeting the CD28/B7 pathway. Here, we seek to gauge the efficacy of antibody production (a marker of adaptive immune function) in monkey cardiac allograft recipients undergoing anti-CD40 or anti-CD28 monoclonal antibody treatment following exposure to tetanus toxoid. Following cardiac allograft transplantation, mAb-based biologic regimen, and tetanus toxoid vaccinations, blood samples were collected at post-immunization intervals. Analysis of these samples to quantify antibody production using ELISA will elucidate the capacity of mAb-based costimulation blockade therapy to maintain adaptive preformed immune function.

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P.19

THE EFFECT OF GESTATIONAL RUNNING WHEEL EXERCISE ON THE METABOLIC PHENOTYPE OF OFFSPRING EXPOSED TO MATERNAL HIGH FAT DIET. Veronica Son*, Seva Khambadkone¹, Miranda Johnson², Timothy Moran², and Kellie Tamashiro², ²Division of Behavioral Sciences, Department of Psychiatry, ¹Johns Hopkins University School of Medicine, Baltimore, MD.

Obesity is a major health concern worldwide that increases the risk for co-morbidities such as hypertension, heart disease, and type 2 diabetes. More alarming is the rate of obesity in children, which is dramatically on the rise and contributes to the earlier onset of type 2 diabetes during childhood and adolescence. Recent studies have demonstrated that the early life environment, particularly the intrauterine environment during pregnancy, can influence the development of obesity and diabetes in offspring. In a rodent model, offspring born to mothers consuming a high fat diet during pregnancy have increased body weight and adiposity, as well as impaired glucose tolerance, insulin sensitivity and leptin sensitivity, all of which promote diabetes. We hypothesized that voluntary exercise during pregnancy will attenuate or reverse the adverse metabolic effects of maternal high fat diet on offspring. Using a rat model, we assessed food intake, body weight, body composition, and glucose tolerance in offspring of dams fed a high fat maternal diet throughout gestation with or without access to a running wheel. Gestational running wheel activity in high fat diet dams had greater effects on offspring in adulthood than in early life (at weaning). At weaning, glucose tolerance was significantly improved by running wheel activity. There was no significant effect of running wheel activity on plasma leptin levels. However, in adulthood, offspring of high fat diet dams with running wheel access had significantly decreased food intake and body weights, decreased total and visceral fat mass, and faster glucose clearance in an oral glucose tolerance test. These data suggest that exercise during pregnancy may be an effective intervention to prevent or reverse the adverse consequences of maternal high fat diet on offspring energy balance and metabolic parameters. Interventions such as maternal exercise may help address the startling increase

of childhood obesity and early onset of type 2 diabetes, as well as lead to positive long-term metabolic effects for offspring that extend into adulthood.

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PREDICTIVE MODEL FOR OVERT HYPOGLYCEMIA FOLLOWING BORDERLINE HYPOGLYCEMIA IN NON-CRITICAL INSULIN-TREATED HOSPITALIZED PATIENTS. Shuvodra Routh*, Susan Langan¹, Sherita Hill Golden², and Nestoras Mathioudakis², ¹Johns Hopkins Bloomberg School of Public Health and ²Division of Endocrinology and Metabolism, Johns Hopkins School of Medicine, Baltimore, MD.

In inpatient settings, insulin therapy accounts for the majority of hypoglycemic events. The objective of this study was to develop a predictive model for identifying hospitalized patients at risk of developing overt insulin-associated hypoglycemia (blood glucose of ≤ 70 mg/dL) following a borderline hypoglycemic event (blood glucose 71-99 mg/dL) in the antecedent 24 hours (day -1). We hypothesized that the following predictors can affect the risk: age, weight/BMI, insulin total daily dose (TDD), nutritional status, kidney disease, liver disease, steroid use, glycemic variables and patterns (blood glucose variability, mean, peak/nadir), and relative insulin doses in the 24 hours before and after the overt hypoglycemic episode. This retrospective cross-sectional study was conducted using EMR records of hospitalized adults with insulin-associated hypoglycemia after the first 24 hours of admission. Multivariable logistic regression models were developed and the Akaike's Information Criterion (AIC) was used for model selection. According to on the model with the lowest AIC, glycemic variability ($P < 0.001$) and Type I diabetes ($P < 0.05$) were associated with the highest odds ratios of 1.57 (95%CI: 1.37-1.82) and 1.52 (95%CI: 1.03-2.23), respectively. Steroid use ($P < 0.001$) and race other than Black or White ($P < 0.001$) were associated with the lowest odds ratios of 0.60 (95%CI: 0.52-0.73) and 0.52 (95%CI: 0.37-0.74), respectively. The model correctly classified 75.7% of the cases, had greater specificity than sensitivity (44.1% and 29.9%, respectively) and a high negative predictive value (91.6%) but a low positive predictive value (22.3%). Due to the low positive predictive value of the model, we concluded that it would have limited effectiveness if utilized in a real-world setting in the format of an informatics alert integrated into the EMR. The model's classification accuracy could potentially be improved by using a dataset not restricted to patients with antecedent borderline hypoglycemia and one that includes all the blood glucose readings during the entire length of hospitalization for both insulin and non-insulin treated patients who developed overt hypoglycemia.

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VARIATION AMONGST PEDIATRIC ORTHOPAEDIC SURGEONS WHEN TREATING MEDIAL EPICONDYLE FRACTURES. Meghan Hughes*, Karan Dua, Nathan O'Hara, Joshua Abzug, Department of Orthopaedics, University of Maryland School of Medicine, Baltimore, MD.

Medial epicondyle fractures are a common pediatric injury due to a child falling on an outstretched arm or direct trauma to the elbow. These fractures account for up to 12% of all pediatric elbow fractures and can be associated with concomitant upper extremity fractures, incarcerated fracture fragments, elbow dislocations, and ulnar nerve injuries. Currently, there is no true standardization of treatment and management leading to discordance amongst providers. Depending on the fracture, associated factors, and patient characteristics, some orthopaedic surgeons believe non-operative treatment is the best approach. However, other orthopaedic surgeons recommend surgery even though there is only anecdotal evidence in the literature, provided by expert opinion, which validates this method. Using a discrete choice experiment methodology, this study aims to assess the variability seen amongst pediatric orthopaedic surgeons

regarding the management of medial epicondyle fractures in children and adolescents. The research team is specifically interested in identifying where the treatment variation comes from, looking at both patient and rater (surgeon) characteristics, to try and develop a standardized approach for managing these fractures.

This research was supported in part by the Proposed Research Initiated by Students and Mentors (PRISM) Program, University of Maryland School of Medicine Office of Student Research.

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A PULSE CHECK ON TEAMWORK AND LEADERSHIP: A DESCRIPTION OF THE FIRST FIVE MINUTES OF RESUSCITATIVE CARE IN A PEDIATRIC EMERGENCY DEPARTMENT. Emily Grimsley*, Niall Cochrane, Ryan Keane, Brian Sumner, and Karen O'Connell, Department of Emergency Medicine, The George Washington University School of Medicine and Health Sciences, Washington, D.C.

Pediatric resuscitations are stressful, rare events that require coordinated care by multiple providers in order to provide timely life-saving interventions. Recent guidelines have established 'best practices' for pediatric resuscitations, including critical tasks to be completed within the first five minutes of patient arrival to obtain the best outcome possible. This study aimed to assess resuscitation team performance at a pediatric tertiary care hospital during cardiopulmonary resuscitation (CPR). This a descriptive study of teamwork and task performance during the initial treatment of pediatric patients presenting in cardiopulmonary arrest (CPA). Video recordings of out-of-hospital pediatric CPAs that had resuscitative care in resuscitation rooms at Children's National Medical Center were reviewed. Only cases where the team had pre-arrival (PA) notification of a patient in CPA were included for the assessment of PA variables. Between January 1, 2014 and June 30, 2015, 24 videos met the inclusion criteria. 4 patients were subsequently excluded due to the lack of a CPR in progress upon patient arrival. All cases had the team assembled and briefed and the majority of roles delineated prior to patient arrival. Team leaders verbalized role assignments 60-90% of the time, while team members verbally assigned themselves to tasks the other 10-36% of the time. Critical tasks such as oxygen delivery, bag-mask ventilation, continuation of CPR, and interosseous line placement occurred within the first minute of patient arrival (median times in seconds: 41.4, 36.8, 25.4, and 124.1 respectively). Areas for improvement included AED/defibrillator pad application (25% within 5 minutes) and administration of the first dose of Epinephrine (80% within 5 minutes). This study highlighted the successful completion of critical interventions within the first five minutes of pediatric CPR. Areas in need of improvement were identified and can serve as valuable data when developing team-focused educational interventions. Studies investigating teamwork communication and performance on actual patient outcomes for out-of-hospital pediatric CPA is needed.

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ASSOCIATION BETWEEN RED BLOOD CELL TRANSFUSIONS AND SEVERITY OF RETINOPATHY OF PREMATURITY IN VERY LOW BIRTH WEIGHT INFANTS. Leah Schecter*, Faeq Al-Mudares¹, Janet Alexander², Alexandre Medina de Jesus¹, and Sripriya Sundararajan¹, ¹Division of Neonatology, Department of Pediatrics and ²Department of Ophthalmology and Visual Sciences, Baltimore, MD.

Red blood cell (RBC) transfusions are frequently administered to very low birth weight (VLBW) infants in the neonatal intensive care unit (NICU). While RBC transfusions are lifesaving, there are risks associated with them. Retinopathy of Prematurity (ROP), a vasculoproliferative eye disease associated with prematurity, is a leading cause of blindness in neonates. In recent years, studies have found evidence to support a relationship between ROP and RBC transfusions. However,

prospective trials have yet to establish a causal link. Our study aims to better characterize the relationship between both the frequency and timing of RBC transfusions and the severity and aggressiveness of ROP. We hypothesize that a positive correlation exists between frequency of RBC transfusions and the severity of ROP. We also hypothesize that the earlier the neonate is exposed to RBC transfusions, the more likely they are to develop treatment requiring ROP and the more rapid the progression of the disease. To test these hypotheses, we conducted a retrospective chart review of 631 VLBW infants admitted to the University of Maryland Medical Center NICU between 1/01/10 and 12/31/2014. Of those 631 neonates, 436 met inclusion criteria. The frequency and timing of RBC transfusions for each neonate during their hospital course until discharge was recorded and statistical analyses were used to evaluate the relationship between number of transfusions and severity of ROP, including t-tests, ANOVA, and multivariate regression. A subset of 386 inborn VLBW infants were studied to analyze the relationship between timing of RBC transfusions and the progression to prethreshold vs. threshold ROP and to laser treatment requiring ROP. Preliminary results show a statistically significant correlation between frequency of RBC transfusions and severity of ROP measured both by stage and by Type 1 or 2 ROP classification. Analysis of timing revealed a cumulative effect on severity of disease but little effect on how quickly the disease progressed.

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PREDICTORS OF POST-NECROTIZING ENTEROCOLITIS STRICTURE FORMATION IN PRETERM NEONATES. Meghan Gray* and Natalie Davis, Division of Neonatology, Department of Pediatrics, University of Maryland School of Medicine, Baltimore, MD.

Necrotizing enterocolitis (NEC), is a severe intestinal disorder affecting 5% of all NICU admissions and 10% of very low birth weight infants, and is associated with a mortality rate of approximately 50%. Patients who survive acute NEC have the added risk of possible stricture formation during the healing process following ischemic injury to the intestine, requiring an additional surgical intervention. It is widely accepted that breast milk feeds are protective against the development of NEC, but little data is available to determine what effect feeding type has on post-NEC stricture formation. The protective effect of breast milk (BM) on development of NEC has led to encouragement of donor breast milk (DBM) when the mother is unavailable to provide BM. DBM is beneficial, but comes with disadvantages, such as cost and availability. We aimed to evaluate potentially modifiable risk factors for post-NEC strictures including treatment type, treatment duration, type of feeding, and evaluate predictors of post-NEC strictures including maternal factors and laboratory profiles that could help providers make earlier diagnoses. This study is a retrospective medical record review including neonates born between 2007 and 2015 diagnosed with definite NEC (stage II or higher). We then evaluated for the diagnosis of stricture and other clinical and demographic risk factors for stricture development. When comparing the stricture group to the non-stricture group, we found no significant effect of gestational age, birth weight, race, or gender. Interestingly, we found no significant effect of type of feeds on post-NEC stricture formation. 100% (n=12) of the stricture group had some exposure to BM, while 82.1% (n=64) of the non-stricture group had exposure to BM (p=.1103). There was also no effect of formula exposure on post-NEC stricture formation, 41.7% (n=5) of the stricture group was exposed to formula, while 37.2% (n=29) of the non-stricture group was exposed to formula (p=.7653). This is the first study evaluating effects of feeds on stricture development, and although we know BM is protective against NEC, no effect is seen on subsequent stricture formation.

P.25

THE ADEQUACY OF OUTSIDE EMERGENCY ROOM AND URGENT CARE CENTER RADIOGRAPHS WHEN PRESENTING TO A PEDIATRIC ORTHOPAEDIC OFFICE SETTING. Eric Margulies*, Karan Dua, Nathan O'Hara, and Joshua Abzug, Department of Orthopaedics, University of Maryland School of Medicine, Baltimore, MD.

Emergency room and urgent care center (ER/UCC) providers are often the first evaluators of pediatric orthopedic injuries. Radiographs are conducted to diagnose the patient, who is then commonly referred to an orthopedic surgeon for further assessment. The aim of this study was to determine the adequacy of these images, and what factors lead to the need to repeat radiographs at the initial visit. A prospective study was performed to enroll all pediatric patients that presented to the pediatric orthopaedic clinic following an acute injury where radiographic studies had previously been obtained at an outside ER/UCC. Demographic and mechanism of injury data was obtained. Adequacy of the initial radiographs was determined in a binary fashion with images deemed adequate if no additional radiographs were obtained, and images considered inadequate if the chief resident or attending physician ordered new radiographs. Radiographs obtained to assess potential loss of reduction were excluded. The amount of time each patient spent in the office from check-in to check-out was also noted. 51 patients were enrolled in the study with 52.9% (n=27) of ER/UCC radiographs being adequate and 47.1% (n=24) being inadequate. Patients with inadequate radiographs required an average of 3.4 (95% CI: 2.7-4.0) additional images. The most common reasons for repeat radiographs were missing views (n=8, 33.3%), an inadequate lateral view (n=7, 29.2%), and poor image quality (n=4, 16.7%). Patients with adequate initial images had a significantly shorter mean clinic visit time (P<0.0001) compared to patients with inadequate radiographs, with a mean difference in clinic time of 32.0 minutes (95% CI: 22.4-41.6). ER/UCC diagnostic imaging is often insufficient to permit the adequate diagnosis and treatment decision making by pediatric orthopaedic surgeons. Repeating initial injury radiographs are costly in terms of radiation exposure to the patient, additional time required of the healthcare system and patient/family, and increased financial cost to the overall healthcare system. Education of ER/UCC providers regarding optimal imaging techniques and views may reduce these burdens.

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EFFECT OF WELLNESS CHAMPIONS FOR CHANGE PILOT INTERVENTION ON THE DEVELOPMENT OF SCHOOL-LEVEL WELLNESS TEAMS. Hengyi Guo*, Megan Lopes¹, Yan Wang², Doris Yimgang², and Erin Hager², ¹Department of Epidemiology and Public Health, Maryland State Department of Education and ²Division of Growth and Nutrition, Department of Pediatrics, University of Maryland School of Medicine, Baltimore, MD.

Schools have been a main focus of childhood obesity prevention efforts for years, with mixed evidence of effectiveness of specific programs on obesity outcomes. Federal legislation has addressed health promotion in schools by mandating school system-level written local wellness policies (LWP) in 2004. LWP effectiveness depends on the level of implementation. However, it is unknown how well schools comply with the unfunded LWP implementation mandate. We conducted a pilot intervention study Wellness Champions for Change to promote LWP implementation. 63 schools from 5 Maryland school systems were randomized, stratified by school system and type (Elementary, Middle, and High), into three arms: schools that received an intervention to train staff to be Wellness Champions, assistance from trained specialists, and financial resources (arm A), schools that received staff training and resources (arm B), and schools that received resources only (arm C). We hypothesize that (1) A schools do better than B schools and (2) B schools do better than C schools in the following areas: 1) percent with wellness teams in place, 1a) activeness and cohesiveness of wellness teams, and 2) level of LWP implementation. A mixed methods approach was used, including quantitative surveys (baseline and one-year after

baseline) and semi-structured interviews to gather feedback on the intervention. Up to 5 respondents were chosen by a school administrator from each school to fill out the online quantitative survey. Interviewees were randomly selected for the qualitative survey. Multi-level linear regression modeling will be conducted for quantitative analyses, adjusting for repeated measures within schools and clustering of schools within school systems. Preliminary analyses among 57 schools (n=57/63, 90%), show a positive trend for A schools of having a greater increase in percentage of wellness teams post-intervention than C schools, although not statistically significant. Next, we will test the hypotheses with the full sample (n=63). Based on the findings, we anticipate developing and disseminating tools to help other school systems more effectively implement their school wellness policies.

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ASSESSING CLINICIAN COMPLIANCE WITH NATIONAL GUIDELINES FOR PEDIATRIC HIV CARE AND TREATMENT IN RWANDA. Laura Sirbu*, Sanchari Ghosh¹, and David Riedel², ¹University of Maryland School of Pharmacy and ²Department of Medicine, University of Maryland School of Medicine, Baltimore, MD.

Children infected with HIV in resource-limited settings such as Rwanda do not fare well; it is estimated that, without treatment, more than half of HIV-infected children in sub-Saharan Africa will die before age two. Over the past decade, Rwanda has made great strides in increasing access to antiretroviral therapy (ART). However, obstacles remain, particularly for children, including difficulties with early HIV diagnosis, commencement of a treatment plan, and retaining children in long term care. A retrospective cohort of 932 pediatric patients (<15 years old) who commenced ART between 2007 and 2009 was analyzed for adherence to National HIV Treatment Guidelines, specifically whether standard protocols were followed for: recording weight before and during ART treatment; prescribing Bactrim prophylaxis to all; screening and providing treatment of tuberculosis (TB); meeting eligibility criteria for starting ART; and whether the correct ART regimen was prescribed. 90% compliance with these measures is the minimum expected threshold for providers in the country. While 97.1% of patients had their weight checked at ART start, only 47.5% had their weight checked at every subsequent visit (i.e., 6, 12, 18, and 24 months and the most recent visit). For Bactrim prophylaxis, 94.8% of patients were correctly prescribed medication, but 3.0% did not have documentation. 92% of children were screened for TB at ART initiation. Of those that screened positive, 25.1% were treated for active TB. Overall, only 73.4% of patients met all of the eligibility criteria for starting ART according to the national guidelines. Of those that did not meet the eligibility criteria, 79.0% started ART early and 21.0% did not have adequate documentation. Additionally, only 67.0% of ART regimens were correctly prescribed based on national guidelines. For example, of TB co-infected children, only 53.5% received the correct medication regimen. Although Rwanda has surpassed many other sub-Saharan African countries for scaling up ART, further efforts focused on educating providers about current national protocols will be necessary to obtain the best HIV-related outcomes for the population.

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FOOD INSECURITY AND OBESITY IN CHILDREN < 4 YEARS: ASSOCIATIONS WITH WIC AND SNAP PARTICIPATION. Chloe Drennen* and Maureen Black, Division of Growth

and Nutrition, Department of Pediatrics, University of Maryland School of Medicine, Baltimore, MD.

Food insecurity and pediatric obesity are significant public health issues, especially among low-income children. Food insecurity may be linked to obesity through a reduction in the quality of food consumed, resulting in the consumption of low-cost, energy-dense, nutrient-poor meals. Food assistance programs, including WIC and SNAP, seek to reduce the burden of food insecurity and improve diet quality. Findings regarding relations between food insecurity and obesity among children are mixed. Most studies focus on children ages 4-18. The few studies among children < 4 often fail to stratify by age, potentially masking important developmental differences in young children's experience of food insecurity and susceptibility to obesity. The goal of this study is to assess in a sample of children < age 4 from low-income families 1) age related differences in the risk for obesity; 2) age related differences in relations between food insecurity and obesity; and 3) the impact of food assistance program participation on these relations. Data were collected by Children's HealthWatch. Trained research assistants administered a survey to a sample of low-income, multiethnic caregivers from January 2009 – December 2015. Child weight was measured. Four age categories were used: <13 months, 13 - 24 months, 25 – 36 months, and 37 - 48 months. The analysis plan included unadjusted logistical regression, followed by adjusted logistic regressions. Rates of obesity increased with age and were significantly higher among the two oldest age groups (12.3% age 25-36 months, 14.5% age 37-48 months), compared with < 13 months (7%). In unadjusted analyses, rates of obesity did not differ between food secure/insecure status among children < 24 months, but among 3 year-olds, food insecure children had higher rates of obesity compared to food secure children. However, differences were not significant in adjusted analyses. Food assistance program participation was not associated with increased risk for obesity, but did not reduce the risk among food insecure children. Additional research is necessary to identify the confounders associated with the increased risk among 3 year-olds.

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THE X-RAY CRYSTAL STRUCTURE OF HNRNP A18 RNA RECOGNITION MOTIF.
Katherine Coburn*, Zephan Melville¹, Braden Roth¹, France Carrier², and David Weber¹,
¹Department of Biochemistry and Molecular Biology and ²Department of Radiation Oncology, University of Maryland School of Medicine, , Baltimore, MD.

Skin cancer is the most common form of all cancers and malignant melanoma (MM) accounts for over 72% of the skin cancer deaths each year. Stage IV MM, the most advanced stage of MM, is challenging to treat with only a small subset of patients responding positively. The five and ten year survival rates for stage IV are 15% to 20% and 10% to 15%, respectively. One characteristic of MM is the up-regulation of a heterogeneous ribonucleoprotein termed hnRNP A18 (A18). A18 is a RNA binding protein found in malignant melanoma cell lines and patient tumors. A18 is predominantly located in the nucleus, but translocates to the cytosol upon assault by cellular stressors such as UV or hypoxia. In the cytosol, A18 binds to and stabilizes mRNA transcripts for pro-survival genes that code for proteins such as thioredoxin and the hypoxia-inducible factor 1 alpha (HIF-1 α). For these reasons, and because of its overexpression in MM, A18 is being targeted via structure-based drug design approaches. Towards such a goal, the x-ray crystallography structure of the RNA recognition motif (RRM) of A18 was solved and is described here. First, protein crystals were obtained by sitting drop diffusion and then diffraction data were collected remotely from the Advanced Photo Source at Argonne National Laboratory. These data were used to solve the crystal structure and demonstrated that A18 has a tertiary structure observed in many other RRM, which includes two

alpha helices and four beta sheets. A18 has high sequence identity with other hnRNPs, especially in the RRM and as expected beta sheets 1 and 3 residues were conserved in 3D space and likely involved in RNA binding. The next step for this research is to develop small molecule inhibitors for A18 using this structure and structure-based drug design approaches once early stage leads are identified.

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EFFECTS OF PRENATAL HYPOXIA ON MITOCHONDRIAL PROTEIN EXPRESSION OF GUINEA PIG OFFSPRING LIVERS. Hannah Khan*, Hannah Khan, Song Hong, and Loren Thompson, Department of Obstetrics, Gynecology and Reproductive Sciences, University of Maryland School of Medicine, Baltimore, MD.

A healthy pregnant woman provides the environment for the developing fetus. However, intrauterine stress such as hypoxia (HPX), undernutrition, and overnutrition, can negatively impact fetal growth and development. We now know that fetal growth restriction, a maladaptive response to intrauterine stress, increases the risk of chronic disease such as cardiovascular disease, metabolic syndrome and obesity in adults through programming. Animal studies have been developed to study the underlying mechanisms contributing to programming effects. Recent studies in our lab have shown that gestational hypoxia induces maternal hypertension, compensatory placental growth and fetal growth restriction, and exhibit a variety of changes in gene expression the fetal heart and liver. In HPX fetal livers, we measured reduced activity levels of the fatty acid oxidation enzyme, Medium Chain Acyl-CoA Dehydrogenase (MCAD), and increased levels in the offspring exposed to prenatal HPX compared to their respective controls. Therefore, we hypothesized that prenatal hypoxia alters mitochondrial enzyme expression and programs altered mitochondrial function in the offspring liver. My study investigates the programming effect of prenatal HPX on expression of the mitochondrial complex proteins (I-V) in the electron transport chain to test the effect of prenatal HPX on mitochondrial function. Pregnant guinea pigs were exposed to either normoxia (NMX) or HPX (10.5%O₂) during late gestation (50-65d, term =65d) and the offspring were raised in a NMX environment and studied at 90d old. Male offspring livers were extracted, weighed, and frozen tissues were assayed for protein expression of Complexes I-V using Western blot analysis. Prenatal hypoxia had no effect on expression of any of the mitochondrial proteins measured. We conclude that prenatal HPX may selectively alter mitochondrial protein expression in the offspring liver.

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A PHASE II STUDY EVALUATING THE EFFICACY AND TOXICITIES OF TREATMENT MODALITIES IN PATIENTS WITH HIGH RISK, NON-METASTATIC PROSTATE CANCER. Gloribel Le*, Young Kwok, and Stephanie Rice, Department of Radiation Oncology, University of Maryland School of Medicine, Baltimore, MD.

High risk prostate cancer remains a challenging entity to treat. Current standard of care with radiation therapy includes long-term androgen deprivation and radiation therapy given as entirely external beam radiation therapy (EBRT) or EBRT with a brachytherapy boost. Even with this therapy, many patients will suffer a biochemical recurrence. This study was aimed at evaluating the benefit of adjuvant docetaxel with regard to disease free survival as well as acute and chronic toxicity. After IRB approval, this phase two trial accrued 38 patients to undergo treatment with neoadjuvant, concurrent and adjuvant androgen deprivation along with EBRT to a total dose of 45 Gy to the whole pelvis and a Cs-131 brachytherapy boost and 4 cycles of docetaxel chemotherapy. Acute and chronic gastrointestinal and genitourinary toxicities were monitored at follow up visits, and disease-free survival (DFS) was monitored based on biochemical recurrence as evidenced by a

nadir + 2 rise in PSA. The average age of enrolled patients at the time of prostate cancer diagnosis was 62 years. At a median follow up of 83 months, 84.2% of patients (32/38 patients) completed the treatments specified in the GCC -0605 protocol. From the cohort who completed the treatments as specified, 12.5% of patients experienced grade 2 or higher acute gastrointestinal toxicity, 21.9% of patients experienced grade 2 or higher acute genitourinary toxicity, 3.1% of patients experienced late (1 year post-treatment) grade 2 or higher gastrointestinal toxicity, 3.1% of patients experienced late (1 year post-treatment) grade 2 or higher genitourinary toxicity, and only 9.4% had biochemical recurrence of their prostate cancer. Median DFS was 66 months for all patients treated per protocol. Our results show promising early results in this challenging disease entity at a relatively short follow up duration. Further maturation of data is necessary, but early results are promising that the addition of docetaxel may offer additional benefit to high risk prostate cancer patients in the upfront setting.

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INJURY SEVERITY SCORE ASSOCIATED WITH CONCURRENT BLADDER INJURY IN PATIENTS WITH BLUNT URETHRAL INJURY. Eric Eidelman*, Ian Stormont¹, Deborah Stein², and M. Minhaj Siddiqui², ¹Division of Urology, Department of Surgery and ²Department of Surgery, University of Maryland School of Medicine, Baltimore, MD.

Blunt urethral trauma is generally caused by significant force, with many injuries attributable to motor vehicle accidents. High impact trauma increases the likelihood of concurrent injury in other organs, such as the bladder. The mechanism of concurrent bladder injury with blunt force urethral trauma has recently been described in the literature. Common indicators of bladder injury are gross hematuria and pelvic fracture. However, in a patient with multitrauma these indicators are not always reliable. To properly diagnose bladder trauma a cystogram must be performed. In a trauma scenario, it is not feasible to give every patient with blunt urethral trauma a prompt cystogram. The decision to receive the additional imaging is often based on the surgeon's discretion. A proven prognostic indicator to evaluate the likelihood of bladder injury in these patients has not yet been established, but would be useful in determining care. This purpose of this IRB approved retrospective study was to examine the potential of using Injury Severity Scores as an indicator of this injury type. The Injury Severity Score (ISS) is a commonly used prognostic indicator in trauma centers with a higher score indicating a more serious injury. A cohort of 98 trauma patients who presented with blunt urethral trauma over a 12 year period was evaluated, with 28 of these patients presenting with concurrent bladder injury. Using univariate analysis multiple factors, including ISS, were studied to determine if any showed a positive correlation with concurrent bladder injury. The results show that there was a statistically significant correlation between ISS and concurrent bladder injury ($p=0.0001$), with an $ISS \geq 34$ showing a greater than 50% chance of bladder involvement; an $ISS < 34$ showed a 13% chance of bladder involvement. None of the other analyzed factors showed significance. Using ISS as an added indicator of bladder trauma could potentially be used in clinical practice with high utility in determining which blunt urethral trauma patients receive additional imaging.

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IMPACT OF TWITTER ON CITATION BASED METRICS IN UROLOGY. Solomon Hayon* and M. Minhaj Siddiqui, Division of Urology, Department of Surgery, University of Maryland School of Medicine, Baltimore, MD.

From 2012 to 2013 the number of tweets at the AUA annual meeting rose from 811 to 4,951, calling into question if there is a measurable relationship between academic urology and Twitter. This paper sought to quantify the relationship between Twitter mentions and the number of citations a paper receives. 213 papers from 7 prominent urology journals were examined. Analysis was performed 18 months after publication to allow ample time for accumulation of social media posts and academic citations. Each paper was evaluated with 3 metrics – Scopus, Google Scholar, and Altmetric. Altmetric software allowed for individual tweets to be examined, and the frequency of authors self-tweeting their own papers was tracked. Analysis showed that even a single mention on Twitter significantly affected the number of academic citations a paper received. The average Scopus and Google Scholar scores of a paper with ≥ 1 Twitter mention were 5.60 and 10.48 respectively, compared to 2.82 and 5.07 for papers not mentioned on Twitter ($p=0.008$ and $p=0.002$). Furthermore, there was a positive correlation between the number of Twitter mentions and the number of citations for Scopus ($R= 0.328$, $p=<0.001$) and Google Scholar ($R=0.348$, $p=<0.001$). This relationship remained significant when controlling for journal impact factor. Finally, it was determined that an author self-tweeting a paper significantly affected the number of citations, with a 4.62 and 6.49 citation increase for Scopus and Google Scholar scores ($p=0.005$ and $p=0.020$) in self-tweeted papers. This data suggests that Twitter plays a role in predicting the popularity of a paper in both the social media and academic domains, which is interesting considering the vastly different timelines of Twitter (minutes to days) and academic publications (months to years). The fact that author Twitter mentions significantly impacts citation number poses an interesting dilemma in that it shows authors are capable of self-promotion, but also of potential “gaming” of paper metrics. This is important since both citation and alternative metrics are sometimes used as a proxy for research quality and impact.

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TB OR NOT TB IN THE ABDOMEN: A CASE REPORT OF PERITONEAL TUBERCULOSIS IN AN IMMUNOCOMPETENT PERSON WITH NO RISK FACTORS IN BALTIMORE CITY. Soraya Chanyasubkit*, Patricia Tellez-Watson, and John Warren, Division of Infectious Diseases, Department of Medicine, University of Maryland School of Medicine, Baltimore, MD.

Extra-pulmonary tuberculosis (EPTB) composes approximately 20% of all tuberculosis (TB) cases and peritoneal tuberculosis is the sixth most common form of EPTB worldwide. Peritoneal tuberculosis poses a significant diagnostic challenge because of its uncommonness, non-specific symptoms, and weaker association with traditional TB risk factors such as homelessness, HIV status, or incarceration. A 25 year old African American woman who has never left Baltimore City and denied any history of homelessness or incarceration presented with one month of abdominal pain and fevers, initially concerning for carcinomatosis. An omental biopsy showed necrotizing granulomas and no evidence of malignancy. Given the high clinical suspicion for peritoneal TB because of positive quantiferon TB gold, necrotizing granulomas, and negative rheumatology and malignancy work-up, she was started on empiric RIPE therapy. After discharge, *Mycobacterium tuberculosis* was confirmed via DNA probe from an acid-fast bacilli culture of the omental biopsy. Though it is a rare disease, peritoneal TB should be considered as part of a differential diagnosis for abdominal pain and distension regardless of immune state and traditional pulmonary TB risk factors. Empiric treatment should also be started if clinical suspicion is high as it can take many weeks for cultures and results to be finalized.

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STATISTICAL COMPARISON AND MORTALITY TRENDS BETWEEN QSOFA AND SIRS AMONGST HAITIAN ICU PATIENTS. Ali Aneizi*, Rafaela Izurieta, Claire Staley, and Alfred Papali, Division of Pulmonary and Critical Care, Department of Medicine, University of Maryland School of Medicine, Baltimore, MD.

Mortality from sepsis remains substantial across the world, but resource-constrained countries face the highest burden. Lack of recognition and diagnosis of sepsis at the time of triage has been identified as one of three major delays in adequate treatment. Recently, an international task force has redefined clinical standards for diagnosing sepsis using physiologic variables that are easy to calculate by the bedside. This physiologic scoring is known as the “qSOFA score.” The Quick Sequential Organ Failure Assessment (qSOFA) score was developed and validated in high-income countries. Its validity in resource-constrained settings, particularly in low- and low-middle income countries, is unknown. This study evaluates qSOFA and compares it to SIRS (Systemic Inflammatory Response Syndrome) in the ICU of the low-income country of Haiti. qSOFA patients are significantly different at the time of ICU transfer than SIRS patients in that they have significantly systolic and diastolic blood pressures ($p=0.01$ and $p=0.02$, respectively) and significantly lower Glasgow Coma Scores ($p<0.001$). No significant differences in mortality rate were found between qSOFA patients, SIRS patients, or the general ICU cohort. qSOFA exhibited 42.0% sensitivity in diagnosing sepsis, as defined by SIRS, and a specificity of 95.1%. qSOFA is not an appropriate screening tool for sepsis in this Haitian ICU. qSOFA may be better in identifying patients who are further along in their sepsis progression, but more research needs to be done in order to fully define the role of qSOFA in low-income countries.

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THE EFFECTS OF CLOSED INCISION NEGATIVE PRESSURE WOUND THERAPY IN PANNICULECTOMY PATIENTS. Eseigboria Ikheloa*, Yvonne Rasko¹, Silviu Diaconu², Colton McNichols¹, ¹Division of Plastic Surgery, Department of Surgery, and ²Department of Surgery, University of Maryland School of Medicine, Baltimore, MD.

Abdominal wall hernias in the obese population of the United States are a surgical challenge to surgeons across the nation. An extensive abdominal panniculus has negative implications on a patient's quality of life including limited appropriate hygiene, increased risk of chronic dermatitis, cellulitis, candidiasis, skin ulceration, and limited mobility. These risks have led some surgeons to believe that simultaneous panniculectomy at the time of ventral hernia repair in morbidly obese patients should be performed. The benefits of panniculectomy include removal of poorly vascularized and inflamed tissue. After undergoing panniculectomy, patients may exhibit increased mobility and attain a better quality of life. However, a negative aspect of panniculectomy is a resultant large wound. As with any wound, there is potential for complications including infection, seroma, hematoma, skin necrosis, and skin/fascial dehiscence. Prior to the last twenty years, it was standard for surgeons to close an incision using primary intention such as sutures or staples. A newer technique, incisional negative pressure wound therapy was introduced and is largely utilized within plastic surgery. Negative pressure wound therapy uses a vacuum source to create negative pressure inside of a wound. This creates a tight seal in hopes for better healing. Previous studies of the effectiveness of negative pressure wound therapy have shown varying results based on procedure. This study looks at patients at the University of Maryland Shock Trauma who received simultaneous panniculectomy and ventral hernia repair and their outcomes in the presence and

absence of negative wound pressure therapy. Using comparison of means, outcomes were compared and analyzed in an effort to help eradicate gaps in clinical knowledge regarding the benefit or disadvantage of negative pressure wound therapy in patients who receive simultaneous ventral hernia repair and panniculectomy.

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THE EFFECT OF SOLID, SEMI-SOLID AND LIQUID DAIRY PRODUCTS ON SATIETY, FOOD INTAKE AND POST-MEAL GLYCEMIA IN NORMAL WEIGHT AND OVERWEIGHT SENIORS. Sara Fard*, Shirley Vien¹, Harvey Anderson², and Marron Law¹, ¹University of Toronto School of Nutritional sciences and ²Department of Medicine, University of Toronto School of Medicine, Toronto, Canada.

Seniors have become the fastest growing population at risk of overweight, obesity and diabetes. Higher dairy consumption has been associated with less obesity and diabetes in adult populations, and short-term studies show they improve post-prandial glycemia, satiation and prolong satiety. However, dairy consumption has decreased markedly over the past few decades, such that in 2004, only 26% of men and 20% of women aged 51 to 70 years met the three servings recommended in Canada's Food Guide. The objective of this study was to compare the effects of consuming two servings of dairy products on blood glucose (BG), appetite-regulating hormones, satiety and later food intake in seniors (n=30). To date, 12 healthy males and females (age: 63.5 ± 0.60 y; BMI: 24.8 ± 0.78 kg/m²) completed the study. At baseline (0 min), the participants were fed a treatment of either 500 ml water (calorie-free control), 0% MF milk, 3.25% MF milk, 350 g plain Greek yogurt (2% MF, with additional 150 ml water), or 60 g regular fat cheddar cheese (with additional 425 ml water), to have isovolumetric treatments. At 120 min, participants were given an *ad libitum* pizza meal. At 0, 15, 30, 45, 60, 75, 90, 120, 140 and 170 min, finger pricks were administered for blood glucose (BG) measurement, and subjective appetite was assessed using visual analog scales. Yogurt and cheese resulted in lower post-treatment BG and appetite compared to skim and whole milk, and water ($p < 0.0001$). BG was highest after skim and whole milk, compared to other treatments and water ($p < 0.0001$). Food intake at the test meal was lowest after yogurt, but not different from other treatments, except water ($p = 0.01$). Yogurt and cheese resulted in lower appetite and BG following consumption, and lower food intake at a later *ad libitum* meal, compared to skim and whole milk, and water. Thus, semi-solid and solid dairy products may be a preferred source of dairy for seniors at a breakfast that usually contains a high glycemic carbohydrate.

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FOOD HEDONIC RATINGS IN AN OBESE POPULATION AND ASSOCIATION WITH CHRONIC DISEASE AND WEIGHT LOSS. Jacob Quaytman* and Nanette Steinle, Division of Endocrinology, Diabetes, and Nutrition, Department of Medicine, University of Maryland School of Medicine, Baltimore, MD.

Obesity has grown to become one of the major public health issues in the United States, and nutrition and food intake are important predictors of obesity risk. Hedonic measures have been shown to be an accurate way of measuring habitual food and drink intake and risk for chronic disease as an alternative to dietary recall. Obese people are known to have greater overall liking of sweet and fatty foods compared to normal weight individuals but less is known about how the food

hedonics of obese people changes following weight loss. In this study, we analyzed the variation in food hedonics using a general Labelled Magnitude Scale in patients enrolled in weight loss programs at the Baltimore Veterans Administration and University of Maryland Medical Center. We compared subjects' baseline mean hedonic values of six different food groups (sweet, salty, sour, bitter, umami, fatty) with obesity comorbidities and tasting sensitivity of the bitter compound 6-n-propylthiouracil (PROP). Nontasters of PROP significantly preferred fatty foods and trended toward liking bitter foods more than tasters of PROP. Of note among obesity comorbidities, patients with hyperlipidemia significantly liked fatty foods at baseline more than patients without hyperlipidemia. Going forward, we will compare how the mean hedonic values of different food groups changed after 12 weeks and 12 months in the weight loss program for subjects who successfully lost weight with those who did not. We hypothesize that participants who lose weight will have lower hedonic scores for sweet and fatty foods than those who did not lose weight. Recognizing how food preferences are related to chronic disease diagnoses and weight loss will help us better understand taste plasticity and the determinants of obesity and weight loss.

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EFFECTS OF WEIGHT LOSS AND AEROBIC EXERCISE TRAINING ON ADIPOSE TISSUE ZINC α 2-GLYCOPROTEIN AND ASSOCIATED GENES IN OBESITY. Shealinna Ge*, Alice Ryan¹, and Shawna McMillin², ¹Division of Gerontology and Geriatric Medicine, ²Department of Medicine, University of Maryland School of Medicine, Baltimore, MD.

Zinc α 2-glycoprotein (ZAG) has been implicated in the mobilization and utilization of fatty acids from adipose tissue, and is lower in obese and higher in cachexic adults than normal weight adults. Previous studies suggest that ZAG binds to the beta3-adrenergic receptor (B3AR) to influence fatty acid metabolism in adipose tissue by regulating hormone sensitive lipase (HSL) and fatty acid synthase (FAS). The purpose of this study is to investigate the effects of a six month weight loss (WL) or aerobic exercise (AEX) intervention on adipose tissue and skeletal muscle ZAG mRNA levels and protein expression, as well as the expression of B3AR, FAS, and HSL in 26 men (n=10) and women (n=14) (BMI=21-45 kg/m², age=50-81 years). Abdominal and gluteal adipose tissue and vastus lateralis muscle were obtained before and after WL (n=13) or AEX (n=13). ZAG and B3AR expressions were determined by RT-PCR, and ZAG plasma levels by ELISA. Body weight decreased 11% in men and 8.5% in women (p<0.001) in WL and did not change with AEX. VO_{2max} increased 9.2% in men and 18% in women (p<0.005) after AEX. RMR decreased only in men after WL (-9.0%). Men have lower abdominal and gluteal mRNA expressions in ZAG (p<0.05) and HSL (p<0.05) than women. AEX increased gluteal ZAG expression (+16%, p<0.05) and abdominal HSL expression (+31%, p<0.05) in men, and not in women. Abdominal ZAG and HSL mRNA levels did not change significantly with WL in either sex. There were no changes in plasma ZAG and adipose tissue B3AR and FAS mRNA levels. ZAG mRNA expression is associated with resting metabolic rate (abd r=-0.53, glut r=-0.51, p<0.05) and basal fat oxidation (abd r=-0.43, glut r=-0.40, p<0.05), suggesting that ZAG regulates energy expenditure and substrate utilization. Aerobic exercise is a potential lifestyle strategy to modify ZAG and HSL expression that may be sex dependent. Further work is needed to elucidate the role of ZAG in the propensity for weight gain and the ability of exercise to mitigate these responses.

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POWER INJECTION THROUGH ULTRASOUND-GUIDED IVS: SAFETY AND EFFICACY UNDER AN INSTITUTIONAL POLICY. Kathy Dunning* and Michael Witting, Department of Emergency Medicine, University of Maryland School of Medicine, Baltimore, MD.

A case of contrast-associated compartment syndrome at the University of Maryland Medical Center (UMMC) sparked a concern for using ultrasound-guided IV lines for power injection which was followed by a policy limiting high speed injection to ultrasound guided IVs. Here, we estimate the safety and efficacy of high-speed contrast injection using this protocol in patients with ultrasound-guided access. In this ambispective study, we enrolled prospective cohorts of emergency department patients with high-speed radiographic contrast media injection (≥ 3.5 ml/sec) into two groups, IVs placed with ultrasound guidance and IVs placed using traditional inspection and palpation. We performed a separate retrospective review including these defined groups. Furthermore, we reviewed all hospital records of patients with compartment syndrome during the period between January 2010 and December 2011. Between November 2013 and August 2014, 32 patients were referred to radiology for high-speed injection through ultrasound-guided IVs. Of these, 25/32 (78%) had successful injection (7 failed in radiology) versus 26/27 (96%) with catheters inserted using traditional methods (risk difference = 0.18, [95% CI (-)0.01-(+)0.38]). Based on retrospective records, we estimate that 79 additional cases were done during the period between 2010-2011. We noted no cases of compartment syndrome during either period, for an incidence estimate of 0 per 100 cases (95% CI 0-3). A hospital policy for high-speed contrast injection through ultrasound-guided IVs has a safe record. Ultrasound-guided IVs are refused in 22% of trips to radiology.

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PULMONARY ACTINOMYCOSIS: A CASE REPORT AND REVIEW OF THE LITERATURE. Grace Maldarelli* and Danielle Baek, Division of General Internal Medicine, Department of Medicine, University of Maryland School of Medicine, Baltimore, MD.

The actinomyces are a genus of anaerobic and microaerophilic Gram-positive filament-forming rod-shaped bacteria. The various *Actinomyces* species can be found in the normal healthy microbiota of the human mouth, gastrointestinal tract, and reproductive tract. *Actinomyces* can cause disease, however, when bacteria pass through a breach in the mucosa of any of those areas. The most common presentations of *Actinomyces* infections are cervicofacial actinomycosis, pelvic actinomycosis, and pulmonary actinomycosis. Pulmonary actinomycosis is frequently associated with aspiration of either oral secretions or foreign bodies. The various manifestations of actinomycosis are often mistaken for malignancy on imaging, and a tissue biopsy is typically required for final diagnosis. Here, we report the case of a 66 year old man with a past medical history of chronic obstructive pulmonary disease, chronic alcohol use, and a 12.5 pack-year smoking history who presented with altered mental status, difficulty ambulating, and a cough productive of thick white sputum. He was subsequently found to have a right lower lobe mass by CT scan. His early hospital course was complicated by acute respiratory failure requiring intubation, and by subsequent cardiac arrest with rapid return of spontaneous circulation. The patient was eventually diagnosed with pulmonary actinomycosis by biopsy of the lung mass, and penicillin treatment was initiated. This case illustrates some classic history, imaging, and biopsy findings of pulmonary actinomycosis, as will as demonstrates the possible severity of *Actinomyces* infection.

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