The Janus particles were fabricated from 21 μm polystyrene spheres deposited on glass slides and covered with a top aluminum thin film. These Janus particles were then deposited in a specially fabricated cell and their alignment was controlled with AC electric fields at low frequency of 50 Hz.

POLARIMETRIC OBSERVATIONS OF HD 197770. G. A. Topasna, Department of Physics and Astronomy, Virginia Military Institute, Lexington, VA 24450. Observations of the polarized star HD 197770 were made in the *V* band using the using the optical polarimeter on the 0.5 meter telescope at the Virginia Military Institute observatory located at McKethan Park in Lexington, VA. Observations of other standard stars were made for comparison of the degree of polarization and the polarization position angle. The analysis shows that the HD 197770 shows less variability than the other standards stars. The mean values of the normalized Stokes parameters q and u were analyzed and the null hypothesis of equality of population means was tested using the Welch test. The weighted average of the normalized Stokes parameters were used to calculate the degree polarization and position angle and found to be 4.087 ± 0.003 % and $131.16 \pm 0.05^{\circ}$ respectively. Given the stability of HD 197770 we recommend that it be studied further across multiple wavelengths for possible use as a secondary standard star.

OPEN SOURCE TEXTBOOKS. Thomas C. Mosca III, Rappahannock Community College, Department of Mathematics, Warsaw, VA 22572. With textbooks costing hundreds of dollars per class, there is considerable motivation to examine "open educational resource" (OER) materials. This presentation provides a brief introduction to open source teaching materials for math and physics classes, and touches briefly on copyright. The purpose of this talk is more to stimulate discussion of the pros and cons, and also to solicit sources from members of the audience, for the benefit of other members.

Biology with Microbiology and Molecular Biology

INTIMATE ATTACHMENT: THE INTIMIN STORY OF PATHOGENESIS. Abigail Lenz, Department of Biology and Chemistry, Liberty University, Lynchburg VA 24502. Enterohemorrhagic *Escherichia coli* (EHEC) is a bacterium causing of mortality via hemolytic uremic syndrome. The virulence EHEC causes in humans is modeled in mice with *Citrobacter rodentium* (CR), which is a normal murine pathogen causing transmissible murine colonic hyperplasia but is not pathogenic to humans. The two most widely used strains of CR are ICC168 and DBS100, but only ICC168 has been sequenced. Therefore, genetic differences between these widely used strains have yet to be determined. We sequenced DBS100 and found it to be different from its presumably clonal ancestor by almost 400,000 nucleotides. Colonization strategies are thought to be among genetic difference in strains of CR. The process of attachment is important to colonization and involves adhesins, the primary of which is intimin in EHEC and CR. An intimin mutation divorces attachment from pathogenesis in colonization, thus addressing the importance of competition in pathogenesis. Similarly, the introduction and removal of antibiotics in the course of colonization creates a

competitive environment. We found that a more competitive environment increased pathogenesis of CR in the murine intestine.

INTERACTIONS BETWEEN NANOPARTICLES AND MACROPHAGES. M. Bani-Hani[1], C. Osgood[1] & M. Stacey[2], [1]Department of biological sciences, Old Dominion University, Norfolk VA 23529 and [2]Frank Reidy research center for bioelectrics, ODU, Norfolk VA 23529. The unique characteristics of Nanoparticles (NP) make them suitable for a wide range of applications. However, the use of NP raises concerns regarding their potential impact on health. NP could lead to enhanced or suppressed immune functions and they may damage unintended target tissues. To assess the safety and potential impact of NP, we investigated interactions between macrophages and nanocapsules (NC). We used mouse cell line, J774A.1, incubated with different concentrations of NC. The NC were tagged with near infrared (NIR) NC suitable for live animals imaging and flow cytometry analysis. The same NC were tagged with Alexa-488 Fluor for microscopic analysis. The flow cytometry analysis showed the increased fluorescence from cells incubated with these NC for up to 5 hours. From the microscopy images, we found that most (but not all) macrophages engulfed the NC in a time- and dose-dependent manner and most of these NC were localized to the cytoplasm. In addition, some NC entered the nucleus which was clear from the DAPI stain after the NC treatment. Cell viability were not affected by the NC even with the higher NC concentrations and for longer times as it appeared from the MTT assay. Our results, in addition to some preliminary data about NC toxicity, indicated that these NC are not killing the cells but may induce macrophage response. Our next work investigates macrophage function in response to NC treatment.

INDUCTION OF P53 ACTIVITY IN NIH 3T3 CELLS FOLLOWING EXPOSURE TO NON-IONIZING RADIATION. Yoshinori Takeda & Rosemary Barra, Dept. of Biology, University of Mary Washington, Fredericksburg, VA 22401. Radiation biology has been of interest in recent years with the primary focus on ionizing radiation and its effects on DNA. However, there is another, larger part of the electromagnetic spectrum, the non-ionizing radiation section. Recent articles suggest that free radical oxygen forming reactions are possible with exposure to 60Hz frequencies, and that p53 expression is increased in cells exposed to infrared radiation. In this study, NIH 3T3 cells were exposed to a helium-neon laser (4mW, 632.8nm) for 3-5 hours for 2-5 days. To observe the effects by the laser, p53 expression and radical oxygen presence were measured as indicators of DNA damage or response to DNA damage. Two-way ANOVA was performed to test for significance on hours of exposure, radiation exposure versus control, and effects of distance on the treated cells grown in a slide chamber. p53 expression was found to be significantly higher (23% increase in expression of p53) in radiation-treated cells compared to the control ($N=28$, $p=0.028$). Radical oxygen levels were not significantly different ($N= 6$, $p>0.05$), but this may be due to a small sample size and warrants further study. Overall, the data suggests that non-ionizing radiation affects fibroblast cells and p53 expression. Further studies will focus on various radiation sources and the mechanism for p53 activation following exposure to non-ionizing radiation.

IFNL4 GENE EXPRESSION IN HEPATITIS C VIRUS INFECTION. Kellie Perry,[1,2] Ancha Baranova[1,2] & Michael Estep[1], [1]Betty and Guy Beatty Center for Integrated Research, Inova Health System, Falls Church, VA, [2]Center for the Study of Chronic Metabolic Diseases, George Mason University, Fairfax, VA. HCV is associated with long term chronic health conditions such as cirrhosis of the liver, cancer of the liver, and is the primary reason for liver transplants. Our aim was to examine the expression of interferon response genes during IFN+Ribavirin treatment in the context of the IFNL4 genotype. IFNL4 is a SNP that induces an immune response in the presence of HCV infection occurs via activating the Janus kinase signal transducer and activator of transcription (STAT) pathway. The rs368234815 gene that encodes for INFL4 and its variants are located on chromosome 19q13 just upstream of the (rs12979860) IL28B gene that encodes for IFNL3. There are two genetic variants of IFNL4, "?G" and the other nonsense mutation "TT". IFNL4 (rs368234815) has been linked with inducing an immune response and sustained virologic response (SVR) like its neighbor IFNL3 (IL28). In this study, IFNL4-TT showed a strong linkage to IL28B-C allele. Gene expression increased with IFNL4 associated genes on Day 0 and Day 7. Expression differences were observed with IFNL4 associated genes linked to IFNg pathway associated genes on Day 0 and Day 7. The top 5 pathways linked to IFNL4 were apoptotic pathways. Decreased expression with apoptotic genes were correlated with the nonsense IFNL4-TT allele. Relevant time points of gene expression differences were Pretreatment and Day 7. Pathways associated with IFNL4 were predominately apoptotic.

EPIGENETIC ALTERATIONS IN A TRANSGENIC ALZHEIMER'S DISEASE MODEL. Matthew Baker, Michael Carson, Rebecca Haraf, Noor Taher, Amanda Hazy & Gary D. Isaacs, Dept. of Biology and Chemistry, Liberty University, Lynchburg VA. Genetic contributions leading to the development of Alzheimer's disease are mostly obscure, however evidence suggests that epigenetic modifications may play a role. Cytosine methylation has been shown to play a role in several biological disease states, and several AD-associated genes are regulated through this mechanism. Therefore, we set out to determine if methylation can provide genetic markers for AD development. The methylation levels of DNA promoter regions from transgenic mice, which overexpress beta amyloid as seen in AD, were compared to that of control transgenic mice. Two methodologies were used for determining promoter methylation statuses. First, using methylation sensitive (HpaII) and insensitive (MspI) restriction endonucleases, which both target the sequence 5'-CCGG-3', we determined the methylation status of genomic promoter regions through LM-PCR and subsequent microarray analysis. These genomic regions showing Aβ-induced methylation modifications were then narrowed down to the 0.1% most changing regions for stringency purposes. The other methodology used was Methylative DNA Immunoprecipitation, which uses antibodies to isolate methylated genomic fragments that are then hybridized to a microarray. The regions undergoing significant change were linked to their ontological function, revealing correlation with ontologies related to neurogenesis and apoptosis. This evidence seems to indicate an epigenetic contribution to AD pathology. This work is supported by the Jeffress Memorial Trust (Grant J-998), the ARDRAF Grant, and the Virginia Academy of Science.

DETERMINING THE RATIO OF THE MITOCHONDRIAL DNA IN BROWN ADIPOSE TISSUE TO WHITE ADIPOSE TISSUE. Amir Mohammadhasani[2], Maria Keaton[2], Lei Wang[2], Zobair M. Younossi[1,2,3], Ancha Baranova[1,2], Aybike Birerdinc[1,2]. [1]Betty and Guy Beatty Center for Integrated Research, Inova Health System, Falls Church, VA, [2]Center for the Study of Chronic Metabolic Diseases, George Mason University, Fairfax, VA, [3]Center for Liver Diseases, Department of Medicine, Inova Fairfax Hospital, Falls Church, VA. Many recent studies show obesity is now a global public health problem due to the increase in mortality associated with increasing BMI. In adults, visceral adipose is mostly comprised of lipid storing white adipose tissue (WAT) with limited amounts of lipid expending capable brown adipose tissue (BAT) which may play a role in obesity induced chronic diseases. Due to the high number of mitochondria expressing UCP1 BAT cells are capable of using lipids to produce heat. RNA and DNA was extracted from visceral adipose samples collected from obese patients during elective gastric bypass. Mitochondrial and genomic DNA were compared in ratios. Relative expression of PRDM16 and UCP1 were measured with validated primers. Spearman's correlation reveals UCP1 and PRDM16 are strongly positively correlated ($r= 0.6032$, $p<4.035e-09$). However the genomic and mitochondrial ratios were could only be analyzed within a range since BAT activation is regulated by low temperatures. Further research will include differentiation of hTERT immortalized adipose derived Mesenchymal stem cells into BAT and WAT to use as positive and negative ends of the ratio spectrum.

UTILIZING NONTHERMAL PLASMA BASED ON SHIELDED SLIDING DISCHARGE IN AMBIENT AIR FOR DELIVERY OF PLASMID DNA TO MAMMALIAN CELLS. C.M. Edelblute[1,2], L.C. Heller[1,3], M.A. Malik[1], & R. Heller[1,3], [1] Frank Reidy Research Center for Bioelectrics, Old Dominion University, Norfolk, VA,[2] Department of Biology, Old Dominion University, Norfolk, VA, and [3] Medical Diagnostic and Translational Sciences, Old Dominion University, Norfolk, VA. In the current study, we assessed the efficacy of a novel non-thermal plasma reactor based on shielded sliding discharge (SSD) producing cathode-directed streamers generated in ambient air for the delivery of plasmid DNA to mammalian cells. *In vitro* experiments were performed with mouse melanoma cells (B16F10) and human keratinocytes (HaCaT) inoculated with plasmid DNA encoding luciferase (pLUC) and green fluorescent protein (pGFP). Quantitative results measured over a 72-hour period displayed luciferase expression levels as high as 10-fold greater in SSD plasma-exposed cells than levels obtained from the inoculation of plasmid DNA alone ($p<0.05$, $p<0.01$). No effect on viability was observed as consequence to plasma exposure in either cell line. SSD plasma-mediated pGFP delivery to HaCaT cells seeded on polycaprolactone scaffolds was confirmed by immunostaining. SSD plasma exposure significantly enhanced pLUC expression in an *in vivo* murine model over 28 days compared to intradermal pLUC injection alone ($p<0.05$). Our findings suggest SSD plasma is an attractive non-viral, non-contact DNA transfer method that warrants further exploration as an alternative or supplemental molecular delivery approach.

POSITIVE SELECTION OF SPERMATOGONIAL STEM CELLS. Eoin C. Whelan[1], A. Nwala[2], S. Arif[2], S. Olariu[2] & C. Osgood[1], [1]Department of Biology ODU, Norfolk VA 23508, [2]Department of Computer Science ODU, Nofolk VA 23508. The paternal

age effect is when the incidence of certain genetic diseases is associated with father's age. A model has been proposed that explains the exponential increase in disease incidence with father's age via the positive selection of mutations in spermatogonial stem cells while in the testes. We developed a computer model that simulates the stem cell division within the niche and accounts for mutation accumulation in a stochastic manner. The goal was to match the model to existing clinical data and estimate the minimum selection pressure required to explain the disease incidence rates. Our model assumed homeostasis of the stem cell niche and that each seminferous tubule represented a single niche. Each niche began with zero mutant cells. We estimated mutation rate based on the average human mutation rate and the rate of spermatogonial stem cell division. The selective advantage was the probability assigned that if a mutant cell divides it will undergo a symmetric division and produce an additional mutant stem cell. Each run of the simulation showed clumping of mutations consistent with the selective model. The data from the model output was averaged and used to produce a simulated population in order to compare the model to the clinical data. The model agreed with data from clinical studies of paternal age effect syndromes with a selective advantage of 0.005 to 0.01. The paternal age effect incidence rate could not be explained by the model with mutation rate alone but by adding a selective advantage to the mutations our data closely aligns to the clinical incidence. (Supported by Sigma Xi Grant-in-Aid of Research and Virginia Academy of Science)

UTILIZATION OF ENTERIC SIGNAL AUTO-INDUCER 3 BY *ESCHERICHIA COLI* TO REGULATE MOTILITY OF *FLHDC* THROUGH QUORUM SENSING. [1]Ryan N. Montalvo & [2]E. D. Richards, Liberty University [1]Department of Biology and Chemistry & [2]Department of Health Sciences. The interplay of enteric bacteria involves communication utilizing quorum sensing mechanisms. *Escherichia coli* regularly utilize the histidine kinase receptor QseC to regulate motility in the *flhDC* operon. QseC autophosphorylates in response to stress signals within the intestine and is stimulated by the adrenergic hormones epinephrine and norepinephrine as well as a quorum sensing molecule AI-3. The producer(s) of AI-3 within the intestine for *E. coli* are unclear. qPCR of fecal samples quantified concentrations of intestinal bacteria for streptomycin-treated mice and streptomycin-treated mice colonized with *E. coli* MG1655, HS, and Nissle. *Bifidobacterium* display fold increases of 7.5, 6.6, and 11 respectively, at 7 days post-infection. Our results implicate *Bifidobacterium spp.* in the changing motility phenotype. Over a 60-days, we observe population instability in relation to motility and the *flhDC* locus. Sequencing reveals analogous deletions within the *flhDC* loci of non-motile MG1655, HS, and Nissle that. Through motility assays, we observe that MG1655, HS, and Nissle fluctuate from 100% motility to 20-60% motility at 60 days. While the HS and Nissle strains lack the same promoter as MG1655, they retain the capability to exhibit non-motility phenotypes. Distinct mechanisms regulate *flhDC*. Murine and biosynthetic colonization with MG1655, HS, and Nissle strains display the nature of bacterial population fluctuations within the intestine that help define colonization drifts over time.

SEASONAL SHIFTING OF THE TROPHIC NICHE IN A GENERALIST APEX PREDATOR. P. A. P. deHart[1], L.E. Hurd[2], J.M. Taylor[2], M.M. Shearer[2], & M. C. Campbell[2], [1]Department of Biology, Virginia Military Institute, Lexington VA 24450

and [2] Biology Department, Washington & Lee University, Lexington VA 24450. Apex predators influence community composition and diversity through direct and indirect interactions with other species, but they may not occupy the top trophic level throughout their lives. The question remains: are top predators feeding in a frequency-dependent manner, or are they varying their prey in accordance with different dietary demands as they grow and mature? We investigated this question by examining the feeding relationships throughout the lifespan of a well-studied apex predator, the praying mantis *Tenodera aridifolia sinensis*, using stable isotope analysis. We measured the $\delta^{13}C$ and $\delta^{15}N$ of lab-reared mantids fed known diets of arthropods from the key feeding guilds (leaf chewers, sap feeders, and carnivores), of their prey items, and of field-caught mantids over a growing season. Lab-raised mantids generally increased in both $\delta^{13}C$ and $\delta^{15}N$ with the consumption of successively higher trophic level prey, but field-caught mantids were highly variable in this regard. These signatures indicated that mantids did not take prey from trophic guilds in proportion to their abundances, so are not frequency-dependent predators. There was a clear pattern of ^{15}N enrichment during growth and development of mantids, with adults and eggs exhibiting highest values, decreasing from egg hatch to third instar as nymphs fed on lower trophic levels and increasing steadily thereafter, indicating increased carnivores in the diet as nymphs grew. Our results suggest that variability in the community impact of apex generalist predators is strongly influenced by ontogenic shifts in dietary demand.

Posters

PHYLOGENETIC ANALYSES OF *STREPTOCOCCUS PARAUBERIS* FROM FISH AND CATTLE STRAINS. Keaira L. Thornton & Ashley N. Haines, Dept. of Biology, Norfolk State University, Norfolk, Va. 23504. *Streptococcus parauberis* is a gram positive lactic acid bacterium that is found locally and globally and recently has become an emerging pathogen. *S. parauberis* infects cattle and fish, which can negatively impact dairy and aquaculture fisheries. The genomes of multiple isolates from fish were sequenced and compared to the available published genomes from one fish and one cattle isolate. The genomic sequences of various housekeeping genes were used to construct phylogenetic trees using the software Geneious 6.1.5 via Neighbor-Joining and Unweighted Pair Group Method with Arithmetic Mean methods. These phylogenetic analyses will contribute to our understanding of the evolutionary relationships between the strains of this bacterium in different animal host species and predict the closest relative to *S. parauberis*. These data will help explain the molecular epidemiology of *S. parauberis* and help us understand how it spreads from host to host and why it is becoming an emerging pathogen.

PREPS ANALYSIS OF VIRAL PROTEINS POTENTIALLY PRENYLATED BY HUMAN CELLS. Ekateina Marakasova[1], Birgit Eisenhaber[2], Frank Eisenhaber[2], Elena Yarigina[3], Ancha Baranova[1,4], [1]School of Systems Biology, George Mason University, Fairfax, VA 22030, [2]Bioinformatics Institute, Agency for Science, Technology and Research, Singapore 138671, [3]K.I Skryabin Academy of Veterinary Medicine and Biotechnology, Moscow 109472, Russia, [4]Moscow Institute of Physics and Technology, Moscow Region, Dolgoprudny 141700, Russia. Prenylation is a lipid

posttranslational modification of *de novo* synthesized proteins, when a farnesyl or gernylgeranyl group is added to specific site on C-terminal end of a protein. Prenylation of mammalian proteins plays an important role in different processes, such as cell proliferation, differentiation, apoptosis and general metabolism. Recent studies suggest that intracellular microorganisms can use eukaryotic prenylation machine for lipidation of its own proteins. This has been experimentally showed for S. *typhimurium* protein SifA, *L. pneumophila* protein AnkB, and hepatitis delta virus large antigen. In this study we perform analysis of all viral proteins that may be prenylated by human cells. Designed databases consist of human viral proteins both predicted from genome and experimental protein sequences from UniProt and NCBDI protein databases. Prenylation prediction suite (PrePS) predicted 35 viral proteins to be recognized by farnesyltransferase and 9 proteins by geranylgeranyltransferase 2. Interestingly, the vast majority of all identified prenylation events were found in RNA rather than DNA viral proteins.

NKG2D RECEPTOR COSTIMULATION DIFFERENTIALLY ACTIVATES SIGNAL TRANSDUCTION PATHWAYS DOWNSTREAM OF AKT IN MURINE EFFECTOR CD8 T CELLS. Kelsey M. Trace & Amorette Barber, Dept. of Biol. and Env. Sci., Longwood Univ., Farmville VA 23909. Effector CD8 T cells have potent anti-viral and anti-tumor functions. In order to become activated, naive CD8 T cells must be activated through the T cell receptor along with costimulation through the CD28 receptor. After activation, costimulation is no longer required but can greatly affect CD8 T cell functions. One costimulatory receptor that is likely to be stimulated on activated CD8 T cells is NKG2D because the ligands for this receptor are upregulated upon DNA damage, cells stress, and viral infection. Costimulation with NKG2D or CD28 has been shown to activate Akt, however it is unclear what signal transduction pathways downstream of Akt are activated by these receptors. Murine effector CD8 T cells were stimulated through the T cell receptor with and without NKG2D or CD28 costimulation and the activation of Akt and downstream pathways were analyzed using an In-Cell ELISA and RT-PCR. Compared to CD28 costimulation, stimulation through the T cell receptor and NKG2D lead to an increased phosphorylation of AKT and GSK-3β. Gene expression of members of the β-catenin, mTOR, and other pathways was also increased after NKG2D receptor stimulation. These data show that NKG2D costimulation of activated CD8 T cells could potentially enhance their anti-tumor and anti-viral effector functions. (Supported by: Longwood University's Faculty Research Grants and BES Department).

THE USE OF THAWED, ISOLATED LIVER CELL CYTOPLASM AS A NUCLEAR IMPORT MEDIUM. Jeffrey Branson, Haris Mirzada & Stephen Gallik, Ph. D., University of Mary Washington. Fluoresence microscopy studies have shown that proteins lacking a nuclear localization signal (NLS) are excluded from the nucleus. These studies have shown that the fluorescence emission of tetramethylrhodamine isothiocyanate (TRITC)-labeled serum album (BSA) is never detected in nuclei when the labeled protein is incubated with isolated nuclei from various sources. This contradicts the results of autoradiographic studies that clearly show proteins the size of bovine serum albumin can penetrate the nuclear envelope and enter the nucleus very slowly through what is likely a simple diffusion process. This contradiction suggests

that the TRITC fluorophore may not be sensitive enough to detect the low concentrations of albumin that diffuse into the nucleus. The specific objective of this preliminary study is to assess the effectiveness of Alexa Fluor 488 fluorophore-labeled BSA in detecting low concentrations of BSA in the nucleus during its diffusion into the nucleus. Crude homogenates of rat liver cells were incubated for 45 minutes with Alexa Fluor 488-labeled histone H1, which contains an NLS, and Alexa Fluor 488-labeled BSA, which lacks an NLS. The results showed that Alexa Fluor 488 is sensitive enough to detect the very high concentrations of histone H1 that accumulated in nuclei as the result of an NLS-dependent nuclear import process as well as the very low concentrations of BSA that slowly accumulate in the nuclei as the result of simple diffusion.

HOST PLANT PHENOLIC ACIDS: OVIPOSITIONAL STIMULANTS OR DETERRENTS FOR CABBAGE WHITE BUTTERFLY, *PIERIS RAPAE*? Rebecca E. Dey, Skyler T. Carpenter, & Mary E. Lehman, Dept. of Biological and Environmental Sciences, Longwood University, Farmville, VA 23909. Cabbage white butterflies, *Pieris rapae*, lay their eggs on plants of the Brassicaceae family. Females may identify suitable hosts through the detection of various chemicals produced by the plants. Of particular interest are phenolic acids because they are found in high concentrations in Brassicaceae plants, yet little is known about the role they play in ovipositional choices. This study evaluated the potential of four phenolic acids to serve as ovipositional stimulants or deterrents for *Pieris rapae*. The phenolic acids tested were sinapic, vanillic, caffeic, and cinnamic acids. Treatments of phenolic acids and deionized water controls were applied to cabbage disks that were equal in leaf surface area and color. Each treated disk was matched with a DI H20 control and placed within an experimental enclosure with a single mated female. After 24 hours, the number of eggs laid on each disk was observed and recorded. Caffeic and sinapic acids both acted as significant deterrents. Cinnamic acid acted as a significant stimulant. Vanillic acid had no significant effect on oviposition. The combined results of this study and previous research suggest that it may not be possible to make broad generalizations about the effects of phenolic acids on ovipositional decisions and underscores the importance of assessing individual chemicals.

ANTIBIOTIC DYNAMICS OF *ESCHERICHIA COLI* POPULATIONS IN A STREPTOMYCIN-TREATED MOUSE MODEL. Hannah J. Drown & Andrew J. Fabich, Ph.D., Dept. of Biology and Chemistry, Liberty University, Lynchburg VA 24515. *Escherichia coli* infect both the urinary and gastrointestinal tracts in mammals. However, we note that the time required for a population to acquire antibiotic resistance has never been evaluated. By assessing antibiotic resistance at specific time points, we hope to mark the rate of *E. coli* antibiotic resistance *in vivo*. A previous *in vivo* study of *E. coli* fitness measured dynamic changes in motility of several populations over two months, discovering that changes surfaced in the commensal strain of *E. coli* Nissle. We hypothesize that the Nissle strain of *E. coli* within the murine intestine will similarly express spontaneous mutations that produce resistance to antibiotics. In this study, we examined the ability of *E. coli* to gain antibiotic resistance to nalidixic acid, chloramphenicol, novobiocin/SDS, rifampicin, streptomycin, and tetracycline. Each antibiotic was selected to highlight a separate mechanism of action by which resistance

should develop. Individual colonies from mouse feces colonized with the Nissle strain of *E. coli* were screened for genes associated with nalidixic acid, chloramphenicol, novobiocin/SDS, rifampicin, streptomycin, and tetracycline resistance. We compared the wild type *E. coli* growth on each antibiotic to colonies collected on day 7 and day 30 post-infection. The chloramphenicol, novobiocin/SDS, rifampicin, and tetracycline showed no changes in expression after growing *in vivo* for 30 days. However, increased resistance was noted in both the nalidixic acid and streptomycin by day 7 post-infection.

A COMMUNITY ANALYSIS OF THE INTESTINAL MICROBIOTA WITH *CITROBACTER RODENTIUM* COLONIZATION AND PATHOGENESIS. Cassandra E. Black & Andrew J. Fabich, Dept. of Biol. & Chem., Liberty Univ., Lynchburg, VA 24515. Current research does not clearly define how the intestinal microbiota affect gastrointestinal health, including protection from pathogens. EHEC O157:H7 cause severe sickness and is life-threatening, but is difficult to study *in vivo*. *Citrobacter rodentium* (CR) is used to model EHEC infection in mice, but the intestinal microbiota response during CR colonization is not currently well-defined. CR were observed in both conventional and streptomycin-treated mouse models, and microbial population abundances were quantified using genus-specific primers and quantitative PCR. The results indicate that CR colonize better in conventional mice than in streptomycin-treated mice. *Bacteroidales* and *Bifidobacteria* are unaffected by CR colonization, and *Clostridia* and *Lactobacilli* fluctuate during CR colonization with *Lactobacilli* ultimately decreasing. These outcomes may lead to EHEC prevention and clinical care on a global scale.

EVALUATION OF ALTERED GENE EXPRESSION PATTERNS IN BRAIN AND BLOOD FOR AN ALZHEIMER'S DISEASE MOUSE MODEL. A. Házy, M. Baker, R. Haraf & G. D. Isaacs, Department of Biology and Chemistry, Liberty University, Lynchburg VA 24515. Gene expression patterns are known to affect the development and pathology of Alzheimer's disease (AD). To determine the specific changes in expression involved in AD, differential gene expression in brain and blood for an AD mouse model was determined using RNA sequencing. RNA was isolated from brain and blood of AD and control mice (n=3), reverse transcribed to cDNA, and sequenced to determine genome-wide expression. Sequence data was analyzed using the Galaxy platform to identify differentially expressed transcripts. The analysis found 5053 transcripts differentially expressed in the brain for an AD model, including multiple genes known to affect AD progression. Expression data was compared to previously determined changes in methylation and hydroxymethylation to evaluate epigenetic effects on gene expression. While many genes showed a definite increase or decrease in expression with epigenetic changes, other genes displayed splice variant specific changes in expression—certain isoforms of the same gene increased expression and others decreased. This data suggests that epigenetic changes may alter splice variant selection at specific genes. Expression patterns in blood were also determined for comparison to those found in the AD brain. (Supported by: the Jeffress Memorial Trust and the Alzheimer's and Related Diseases Research Award Fund).

A STUDY OF CHLOROQUINE'S ANTIRETROVIRAL CHARACTERISTICS. Justin E. P. Sahs and Lynn O. Lewis, Dept. of Biol. Sci., University of Mary Washington, Fredericksburg VA 22401. Mouse Mammary Tumor Virus, known as MMTV, is a retrovirus transmitted in mice through their breast milk which has been proven to lead to multiple different types of cancer, including breast, ovarian, and prostate cancer. Due to its resemblance to other retroviruses and a relative level of safety towards humans, MMTV is an ideal candidate to act as a research model for retroviruses such as HIV, making it highly relevant in the study of human medicine.The virus enters host cells through receptor mediated endocytosis, which is initiated by the acidification of the vesicle. Chloroquine, which is a weak base, acts as a buffer to minimize acidification and greatly the reduce infectivity of the virus. In research performed last year by UMW student Ryan Green, chloroquine was shown to reduce viral infectivity in MMTV-infected cell cultures most effectively at doses between 2.095E-04 and 2.095E-05 μM. The aim of this study was to prophylactically treat MMTV-infected cells with chloroquine, induce replication with the hormone dexamethasone, and then to isolate and visualize the viral RNA through a spin cycle followed by rtPCR techniques. We found that we were able to visualize the viral RNA via rtPCR followed by gel electrophoresis. Because the chloroquine-treated group was able to be visualized, we can conclude that chloroquine does not eliminate all viral reproduction in hormonally-stimulated MMTV-infected cells.

BIOINFORMATICS AND TRANSCRIPTION FACTORS IN ALZHEIMER'S DISEASE. John R. Davy, John Lawson & Gary D. Isaacs. Dept. of Biology, Liberty University, Lynchburg, VA. 24515. Alzheimer's Disease (AD) is characterized pathologically by neurofibrillary tangles and amyloid-beta plaques, but underlying these processes lie epigenetic changes. Specifically, methylation and hydroxymethylation of cytosine residues in gene promoters changes during the disease process. These changes, either an increase or a decrease in methylation or hydroxymethylation, modulate the binding of transcription factors to the promoter region. We hypothesize that the epigenetic alterations affect specific transcription factors, which alter the gene expression profile seen in AD. Bioinformatics allows high throughput of data, comparing the transcription factors enriched in genes affected by epigenetic changes and those altered in gene expression. Linking these two changing components in AD through TFs provides a dataset for further research into how these TFs are involved in the disease process. We found that a number of TFs, such as USF1, USF2, and ARNT are enriched in both genes changing drastically in epigenetic modifications and in genes changing in expression. Our study also demonstrated that TFs already linked to AD (such as AP1, USF1, and ARNT) are also enriched in epigenetically modified genes, showing the involvement of methylation and hydroxymethylation in the disease process. This work is supported by the Jeffress Memorial Trust (Grant J-998), the ARDRAF grant, and the Virginia Academy of Science.

VISCERAL ADIPOSE DEPENDENT INDUCTION OF AUTOIMMUNE RESPONSE. Allison M. Heath, Katherine Doyle, Aybike Birerdinc & Ancha Baranova, School of Systems Biol., George Mason Univ., Fairfax VA 220304444. One of the numerous adverse effects of the accumulation of excess visceral adipose is that

resulting alteration of cytokine signaling. A state of morbid obesity results in the interference of the body's pro and anti-inflammatory pathways resulting in a systemic pro-inflammatory state and are regulated by such cytokines as IFN-g, TGF-b, TNF-a, IL-1, IL-4, IL-10, IL-12 and IL-8, regulate TH1 and TH2 pathways, which are associated with immune and inflammatory responses. Alteration of the normal function of these pathways may be linked to the development of autoimmune disorders in the morbidly obese. For example in animal models of multiple sclerosis, mice with defective TRIF or type I IFN receptors developed more severe experimental allergic encephalomyelitis than mice with normally functioning receptors and showed an increase in IL-17 production. It has also been shown that embedded in visceral adipose tissue are CD4+ T lymphocytes that govern insulin resistance in mice with diet-induced obesity. Given the role of these proinflammatory regulatory cytokines in both obesity and autoimmune diseases, it appears that the excessive accumulation of visceral adipose tissue may trigger a cascade of events that induces various manifestations of autoimmune diseases particularly in morbidly obese patients.

QUERCETIN AS AN ANTI-INFLAMMATORY AGENT RELEVANT TO THE MANAGEMENT OF METABOLIC SYNDROME. Anna Zhang[2], Ancha Baranova[1,2], Aybike Birerdinc[1,2], [1]Betty and Guy Beatty Center for Integrated Research, Inova Health System, Falls Church VA and [2]Center for the Study of Chronic Metabolic Diseases, School of Systems Biology, George Mason University, Fairfax, VA. Obesity-associated deaths have risen to more than 300,000 a year in the United States, and have been reported as having the second highest mortality rate, second to only tobacco-related deaths. An extensive list of morbidities, such as Metabolic Syndrome (MS), cardiovascular disease (CVD), diabetes mellitus type II (DMII), stroke and cancer is heavily linked to obesity. Complications from obesity and its comorbidities are what makes obesity such a great financial burden. Extensive research has implicated chronic-low grade inflammation induced by dysfunctional adipocytes as a key associate of Metabolic Syndrome, a term coined to cluster major risk factors that are etiologically relevant to the development of cardiovascular disease (CVD) and type II diabetes mellitus (DM), including abdominal obesity, atherogenetic dyslipidemia, hypertension, glucose intolerance. Quercetin, a flavanol ubiquitous in fruit, tea, wine and stems and leaves of vegetables, have been shown to exert an anti-inflammatory action by scavenging adipocytic secretions of pro-inflammatory cytokines from macrophages, activating peroxisome proliferator-activated receptors (PPAR)-gamma, and thus suppressing the activation of pro-inflammatory transcription factor nuclear factor-kappa (NF-κB), and enhancing expression of anti-inflammatory cytokines. The aim of this study is to assess the potential health benefits of quercetin as it relates to the management of metabolic syndrome via its anti-inflammatory mechanisms.

THE EXPRESSION OF KCTD FAMILY GENES IN ADIPOSE TISSUE OF PATIENTS WITH DIET INDUCED OBESITY. Kuan Yao[1,2] , Kataline Anderson[1,2], Aybike Birerdinc[1,2], & Ancha Baranova[1,2], [1]Betty and Guy Beatty Center for Integrated Research, IHS, Falls Church VA 22042, [2]Center for the Study of Genomics in Liver Diseases, Molecular and Microbiology Department, GMU, Fairfax, VA. Potassium Channel KCTD proteins have been suggested in the development of diet induced obesity. KCTD interferes with the normal assembly of the voltage-gated potassium

channels causing the suppression of the voltage-gated channels. Diet-induced obesity was shown to impair endothelium-derived hyperpolarization via altered K channel signaling. Previous studies of KCTD15 have revealed the repression of AP-2α, which inhibits the activity of C/EBPα, an early inducer of adipogenesis. However there is little data on the levels of KCTD15 and other KCTD proteins in the visceral adipose (VAT) of morbidly obese patients. The aim of this research is to determine the relative expression levels of KCTD family genes, such as KCTD1, 9, 12, 15, and 18 in the VAT of a cohort of obese and morbidly obese patients. Currently 12 adipose samples had been extracted and analyzed using qRT-PCR. The delta-delta-ct method was used to investigate the relative levels KCTDs in samples. All KCTD genes have shown a decrease in expression in non-NAFLD samples. However, an increasing trend of gene expressions was observed in KCTD9, KCTD12, and KCTD15 as the levels of NAFLD increase. KCTD1 and KCTD18 gene expressions were variable due to diversity in patients' age, BMI, disease state, and other factors. The assessment of several KCTD family of proteins in the visceral adipose of morbidly obese patients will shed some light on their involvement in the etiology of obesity and its comorbidities.

THE OBESITY LINKED IMMUNE FUNCTION. Momina Tariq,[1] Ancha Baranova[1,2] and Aybike Birerdinc[1,2], [1]Betty and Guy Beatty Center for Integrated Research, Inova Health System, Falls Church, VA, [2]Center for the Study of Chronic Metabolic Diseases, George Mason University, Fairfax, VA. Immune and metabolic systems are among the most primary requirements for survival of a human being. Research in the field of metabolic diseases over the last decade have demonstrated a clear association between weight regulation and inflammation .The immune response and metabolic regulation are highly integrated and the proper function of each is reliant on the other. Dysfunction of one can lead to numerous chronic metabolic disorders, diabetes, fatty liver and cardiovascular diseases. The aim of this present systematic review is to explain the detrimental effects of obesity on the immune system and how it spearheads towards other immune-allied complications. A systematic PubMed literature search was designed. Studies were included that explained the inflammation of adipose tissue as an important protagonist in the development of obesity related problems and studies that interconnected the link between obesity-immune system and systemic inflammation. These preliminary findings indicate that obesity leads to the change in the number of leucocyte count, which instigates impaired immunocompetence and may result in a weakened immune system. Inflammation is characterized by changes in immune cell populations giving rise to altered adipo/cytokine profiles, which in turn induces/stimulates insulin resistance and other diseases such as NAFLD and NASH.

GRIFFONIA SIMPLICIFOLIA ISOLECTIN I-B4 AS A MOLECULAR TAG FOR IMMUNOGLOBULIN. P. Tedrick, E. Marakasova & A. Baranova, School of Systems Biology, George Mason University, Fairfax VA 22030-4444. Lectins are proteins that have high specificity for carbohydrates and can bind reversibly with them by carbonate recognition domains. The isolectin I-B4 (GSI-B4) from *Griffonia simplicifolia* has a marked specificity for a non-reducing terminal μ-D-galactose. We hypothesized that GSI-B4 carbonate recognition domain will bind to immunoglobulin G, because it has an N-glycosylation site that would allow the binding. Therefore an ELISA-like single step assay could potentially be developed to test the binding of GSI-B4 to

immunoglobulins. Partial gene GSI-B4 was synthesized and used for site specific cloning in pQE-30 and pQE-RFP plasmids. Cloning used two restriction enzymes BamHI and SacI for pQE-30, and KpnI and HindIII for pQE-RFP plasmid. T4 DNA ligation reactions were transformed to *E. coli* DH5μ, and positive samples were verified by DNA sequencing. Expression in *E. coli* BL-21 shows at low levels at 10-hour time point after IPTG induction. Expression does not increase substantially by the 24-hour time point. After optimization of expression and verification by Western Blotting, next steps will be to purify GSI-B4. This purified product will be used to develop an ELISA-like assay to test binding of GSI-B4 to immunoglobulin.

A COMPARISON OF GENE EXPRESSION IN RESPONSE TO BPA EXPOSURE IN PROSTATE AND MAMMARY TISSUES. Daniel E. Miller & Deborah A. O'Dell, Dept. of Biol. Sci., Univ. Mary Washington, Fredericksburg, VA 22401. Bisphenol A (BPA) is a synthetic carbon-based compound that has been shown to exhibit hormone-like properties in its ability to act as a ligand to estrogen and androgen receptors. This study aims to investigate similarities in the transcription-altering pathways of BPA in mammary and prostate cells with regards to oncogenes and tumor suppressor genes in untreated control and BPA-treated experimental groups. Previously extracted RNA from human mammary and prostate cells was assessed for integrity and reverse-transcribed to cDNA via PCR. Genomic profiles were obtained through microarray analysis (Oncogenes and Tumor Suppressor Genes PCR Array, SA Bioscience) and analyzed for fold-change in expression from control using software supplied with the assay. Using a threshold fold-change of ± 2.00, BPA exposure resulted in primarily opposite responses in gene expression between the cell lines (i.e. genes up-regulated by BPA in the mammary group tended to be down-regulated in the prostate group). However, there were two oncogenes (JAK2; MYCN) that were up-regulated and two tumor suppressor genes (BRCA1; TP53) that were down-regulated in response to BPA exposure in both mammary and prostate cells. Although JAK2 and MYCN have not been directly linked to mammary or prostate cancers, mutation in both the BRCA1 and TP53 genes is known to be implicated in breast, ovarian, fallopian tube, and prostate cancers, as they play an important role in DNA repair in these cell types. These results suggest a mechanism for the conversion of mammary and prostate epithelial cells to the cancerous state.

CELL PHONE RADIATION INDUCED GENE EXPRESSION IN HUMAN CLIOBLASTOMA CELLS. V. L. King, D. Delfino & D. A. O'Dell, Dept. of Biol. Sci., Univ. Mary Washington, Fredericksburg, VA 22401. The average American spends 25 minutes a day talking on a cell phone. Cell phones emit electromagnetic radiation that ranges from 10^6 to 10^{12} Hertz, which has been shown to produce free radicals that can induce changes in gene transcription. This study examined gene expression changes in human glioblastoma cells over time after cell phone radiation exposure. Human glioblastoma cells were cultured and when confluent were exposed to 25 minutes of cell phone radiation. The cells were lysed and the RNA was extracted immediately, 20 minutes, 24 hours, and 48 hours after exposure. The RNA was converted to cDNA and placed on a commercial RT PCR 96 well plate containing the primers for 84 oncogene and tumor suppressor genes (SABiosciences Corporation: Frederick, MD). RT PCR was performed using a Stratagene 3005p Thermocycler, and

the data was analyzed using the SABioscience PCR Array Data Analysis tool. The results showed that the effects of radiation on gene expression changed as time post-exposure increased. Cell cycle regulation was altered by exposure temporarily allowing increased proliferation that was then reversed. Alteration in genes promoting the cancerous state were up-regulated at 24 hours and then the expression was almost reversed by 48 hours post-exposure. These results provide implications for how constant exposure to cell phones can lead to cellular changes in susceptible cells potentially producing cancerous conditions. Future studies may incorporate larger sample sizes as well as increased and decreased time after exposure for RNA extraction such as 36 and 72 hours after exposure.

COMPARATIVE ANALYSIS OF LUXR REGULATION OF THE QUORUM SENSING IN MESOPHILIC AND PSYCHROPHILIC BACTERIA. Ilya Manukhov[1,3], Ancha Baranova[1,2] & Svetlana Khrulnova[3], [1]Department of General Physics, Moscow Institute of Physics and Technology (State University), 9 Institutsky Lane, Dolgoprudny, 141707, Russia, [2]School of Systems Biology, George Mason University, Fairfax, VA, [3]State Research Institute of Genetics & Selection of Industrial Microorganisms (GosNIIGenetika), Moscow, Russia. Luminescent psycrophiic marine bacteria *Aliivibrio logei* are assumed to be widely distributed in cold waters of northern seas. Here we characterize the structure features of *lux* operon of psychrophilic KCh1 and BM1strains of the marine luminescent bacteria *A. logei* isolated from the intestines of goby fish indigenous to the White Sea and Sea of Okhotsk, Kamchatka. The *A. logei lux* operon was cloned in *Escherichia coli* cells and the structure of this operon and its nucleotide sequence were determined. In the structure of the *A. logei lux* operon, the the luxI gene is absent in front of luxC, and a fragment containing luxR2-luxI genes is located immediately after luxG gene. Thus, it resembles the lux operon of *Aliivibrio salmonicida*. We compared bioluminescent characteristics of this operon with that of mesophilic marine luminescent bacteria *Aliivibrio fischeri*. In spite of structural differences, these two operons exhibited similar sensitivities to autoinducer.

REMOVAL OF OXIDIZED CIRCULATING DNA MAY AUGMENT EFFECTS OF CANCER THERAPY. A. Baranova[1,2], S. V. Kostyuk[1], T. D. Smirnova[1], L. A. Kameneva[1] & N. N. Veiko[1], [1]Research Centre for Medical Genetics, Russian Academy of Medical Sciences, Moscow, [2]Center for the Study of Chronic Metabolic Diseases, School of System Biology, George Mason University, Fairfax, VA. Cell-free DNA fragments (cfDNA) are biologically active as they are enriched in 8-oxo-dG. Short-term exposure to oxidized DNA results in both single- and double strand DNA breaks. Longer treatments evoke a compensatory (adaptive) response. In breast carcinoma model MCF-7, an exposure to oxidized extracellular DNA stimulates the survival and an increase in genome instability. Hence, the oxidized DNA is a stress signal released in response to oxidative stress in the cultured cells and, possibly, in the human body; in particular, it might contribute to systemic abscopal effects of localized irradiation treatments. The mass release of oxidized DNA that accompanies apoptotic and necrotic processes in radio- and chemotherapy treated tumors may aid survival of residual cancer cells and even instigate their resistance to further treatment. It is likely that the selective antibody-guided removal of oxidized DNA from the bloodstream or the block

of respective oxidized DNA-dependent signaling may be developed as an adjuvant treatment for antitumoral therapy.

GLOBAL VARIATIONS IN HBV ENDEMICITY, VACCINE COVERAGE, AND GENOTYPE DISTRIBUTION. Max Marzouk, Ancha Baranova & Aybike Birerdinc, College of Science, George Mason Univ., Fairfax VA. 22030-4444. Despite the advent of an effective vaccination in the 1980's against the hepatitis B virus (HBV), chronic HBV infection remains the world's leading viral cause of hepatocellular carcinoma (HCC). Based on a nucleotide divergence greater than 8%, HBV can be classified into 10 genotypes (A-J) and several subgenotypes with distinct geographic distributions. Natural history studies have indicated that genotype influences clinical outcome and interestingly, the genotypes associated with more severe liver disease predominate in regions with relatively high hepatitis B surface antigen (HBsAg) seroprevalence - an indicator of HBV infection. Here we attempt to characterize global variations in HBV genotype distribution, endemicity, and vaccine coverage – so that emerging nonsynchronous trends can be used to identify regions where HBV resource and education allocation should be prioritized. Recent studies and joint reports by the World Health Organization (WHO) and the United Nations Children's Fund (UNICEF) were the primary sources selected for the analysis of HBV vaccine coverage. Genotyping studies were systematically selected from references in highly cited and relevant publications and Pubmed/Medline keyword searches. Regional seroprevalence and vaccine coverage levels were clustered, mapped, and evaluated. West, East, and Southern Sub-Saharan Africa consistently reported high HBV vaccine coverage during periods of very high endemicity. Similar trends were identified in Central and East Asia. The predominant genotypes in these regions are B, C, and E.

AN ASSOCIATION BETWEEN THE DEPRESSION AND SYSTEMIC INFLAMMATION IN NHANES POPULATIONS. Rachel Smith, Maria Stepanova, Ancha Baranova, & Aybike Birerdinc, Betty and Guy Beatty Center for Integrated Research, Inova Health System, Falls Church VA and Center for the Study of Chronic Metabolic Diseases, George Mason University, Fairfax, VA. A population-based study was conducted to determine the association of individuals with chronic diseases and depression from five cycles of the National Health and Nutrition Examination Survey (NHANES) data. The presence of depression was determined from a depression screener questionnaire, while the presence of chronic diseases were gathered from medical conditions questionnaires (3,751 and 26,225 subjects respectfully). Interestingly, individuals with depression and those with multi-morbidity had the same statistically significant increased inflammatory markers as assessed by HOMA scores, C-reactive proteins, white blood cell counts, and percentages of segmented neutrophils and glycohemoglobin ($p<0.05$). Additionally, both had statistically significant decreased levels of HDL-cholesterol, lymphocyte percentages, and monocyte percentages ($p<0.05$). The increased inflammatory markers suggest that low-grade chronic inflammation may be the connection between depression and chronic diseases in individuals. Examining these inflammatory markers may be used as predictors in the treatment of patients.

NKG2D RECEPTOR ACTIVATION OF NF-KB ENHANCES INFLAMMATORY CYTOKINE PRODUCTION IN MURINE EFFECTOR CD8+ T CELLS. Emily Whitman & Amorette Barber, Dept. of Biol. and Env. Sci., Longwood Univ., Farmville VA 23901. To induce strong immune responses, naïve $CD8^+$ T cells require stimulation through the TCR and costimulatory receptors. However, the effect of activating costimulatory receptors on activated T cells is unclear. The NKG2D costimulatory receptor is expressed on $CD8^+$ T cells with its ligands expressed on tumor cells and during some infections. In order to determine how activation of costimulatory receptors alters effector $CD8^+$ T cell functions, this study compared the activation of the NF-κB signaling pathway by two costimulatory receptors, CD28 and NKG2D. Activation of murine effector $CD8^+$ T cells through CD3 and NKG2D receptors enhanced activation of NF-κB as shown by increased phosphorylation of IKKα, IκBα, and NF-κB and IκBα degradation. Activation of the NF-κB pathway lead to increased secretion of pro-inflammatory cytokines, including IFN-alpha and IFN-gamma, and decreased secretion of anti-inflammatory cytokines, including IL-10 and CCL2. NF-κB activation also increased the effector molecules TNF-alpha, lymphotoxin alpha and beta, and Fas ligand. These data show that stimulation through NKG2D leads to the differential activation of signaling pathways and potentially enhances the anti-tumor and anti-viral functions of effector $CD8^+$ T cells. (Supported by: Longwood University's PRISM program, Faculty Research Grants, and BES department).

Biomedical and General Engineering

STORMWATER RUNOFF FILTRATION MEDIA FOR TRACE METAL CAPTURING. Gail M Moruza, Zach Goehring & Brittany Toney, James Madison Univ., Harrisonburg VA, 22801. Nationwide, stormwater runoff has been identified as the leading cause of water-quality impairment due to the trace metals found within that accumulate over time and create potentially toxic chemical imbalances in ecosystems. Effective stormwater runoff filtration is a key part in preventing dangerous contaminants from being released into the environment and causing harm to the health of aquatic and terrestrial organisms and eventually humans. In particular, the accumulation of trace metals in ecosystems and eventually human drinking water has been known to cause cancer, high blood pressure, liver disease, kidney disease, growth reduction, growth abnormalities, and chronic anemia in the organisms which ingest the metals. The hazardous amassing of trace metals can be alleviated through the study of the most effective filtration media such as zeolite, perlite, and peat. Essential factors affecting filtration efficacy, which are covered in this study, include mass of media and media type used, which in turn determines the mechanisms by which contaminants are captured from solution. From the initial data of this research, it was determined using a one-way ANOVA procedure that no significant difference in filtration capabilities existed between the media types examined. The ANOVA procedure did, however, determine that a greater media mass captured a significantly greater amount of trace metal from solution than did the smaller media mass.

EX VIVO DNA ASSEMBLY. Zachary B. Canfield, Adam B. Fisher & Stephen S. Fong, Department of Chemical and Life Science Engineering, Virginia Commonwealth