**VCU Center on Health Disparities**

**Summer Research Symposium**

August 5th 2015

MMRB Rooms 1009/1011

**Wednesday**

9.30 amIntroduction – Dr Sarah Golding

9.40 am Jaz-munn Johnson – Mentor: Cynthia Cornelissen

9.55 am Lizette Carrasco – Mentor: Kimberley Jefferson

10.10 am Suquoia Mosby – Mentor: Xuichun Ge

10.25 am Michael Kiflezghi - Mentor: Maria Rivera

**Break**

10.45 am Zoe Villamar – Mentor: Paul Wetzel

11.00 am Sarah Izabel – Mentor: Jeff Dupree

11.15 am Joel Roberts – Mentor: Heather Lucas

11.30 am Liam Lewis – Mentor: Andrew Ottens

11.45 am Demetrius Carter – Mentor: Andrew Ottens

**Pizza Lunch Break**

1.00 pm Mark Hairston – Mentor: Joyce Lloyd

1.15 pm Qianni Wu – Mentor: Zendra Zehner

1.30 pm Mariam Sankoh – Mentor: Luiz Shozo Ozaki

1.45 pm Sage McNett – Mentor: Alisa Brewer and Marcie Wright

2 pm Closing remarks

**VCU Center on Health Disparities**

**Vision, Mission, Goals**

**Vision**   
The VCU CoHD will positively impact communities across the commonwealth through the development of programs that are focused on the needs of communities with health disparities.  These programs will reduce disparities through enhancing the skills of both the university community and the disparity community.  **Mission**   
To reduce health disparities by training a diverse and competent workforce, and by serving and engaging disparities communities

**Goals**

1. Enhance research training opportunities for the university  
   **Objectives**
   * Increase the diversity of the health sciences workforce
   * Increase the quantity and quality of health disparities research through research, training and education initiatives
2. Facilitate university and community engagement initiatives to address health disparities  
   **Objectives**
   * Bridge university research with community practice to support relevant research projects that address needs of disparities communities
   * Facilitate university and community partnerships through information sharing and partnership development that enable students, faculty, staff, organizations, and communities to work together to reduce health disparities

**CoHD Research Training Programs**

**The VCU Center on Health Disparities offers several programs and opportunities designed to diversify the workforce in biomedical research. We offer programs for high school students, undergraduates, post-baccalaureates, masters, doctoral and postdoctorals.**

**If you have further questions, contact the VCU CoHD program coordinator of research training programs at COHDTraining@vcu.edu.**

**Initiative for Maximizing Student Diversity Scholars Program**

**PI: Lou De Felice - 2R25GM090084**

The VCU Initiative for Maximizing Student Diversity Scholars Program (IMSD) provides research training in the biomedical sciences for individuals from groups traditionally underrepresented in biomedical research. IMSD Scholars are admitted as early as the end of the freshman year and are involved in program activities through their senior year. The cornerstone of the program is a series of mentored research experiences with VCU faculty members who are leaders in the fields of neuroscience, cancer biology, metabolic diseases, allergy and immunology, microbial pathogenesis, drug addiction or abuse, molecular genetics and others. IMSD Scholars have the opportunity to present their work at local, regional and national research meetings. In addition, IMSD Scholars enroll in a series of courses and workshops that provide instruction in basic concepts in biomedical research, preparation for the Graduate Record Examination (GRE) and career development advice. The IMSD program provides a stipend of approximately $8,000 per year.

**ATTEMPTING TO CREATE A NON-BINDING TBPA MUTANT IN NEISSERIA GONORRHOEAE**

**Jaz-munn Johnson1, Devin Cash2, & Cynthia Cornelissen2**

**1Department of Biology, Virginia Commonwealth University.**

**2Department of Immunology and Microbiology, Virginia Commonwealth University.**

Genital gonorrhea is a very common sexually transmitted disease that affects both sexes. However, it has been demonstrated that women outnumber men as asymptomatic carriers. *Neisseria gonorrhoeae* is the causative agent of the STI gonorrhea and infected individuals fail to develop any protection to the disease. Gonococcal infection in women starts with dysuria and discharge and can progress into more serious matters, including infertility and pelvic inflammatory disease. The disease is becoming resistant to antibiotics and soon there will be no way to successfully treat it, which has caused the demand for a vaccine to heighten immensely. The goal of this study is to investigate gonococcal protein characteristics to contribute to the development of a vaccine. It has been documented that this pathogen has evolved to hijack human proteins and steal vital nutrients including iron from transferrin. The gonococcal transferrin-iron acquisition system is composed of an integral, outer-membrane, TonB dependent transporter (TbpA), and a surface exposed lipo-protein (TbpB). This iron acquisition process is a prime area to study for drug development because it is critical for obtaining nutrients, it is surface exposed, and the protein sequences are well conserved across strains. It has been shown that gonococcal strains without the transferrin receptors do not have the ability to infect humans. This project will utilize site specific mutagenesis of surface exposed loops of TbpA to evaluate key structure-function relationships in their interactions with human transferrin. Specifically, we aim to create a mutant TbpA that cannot bind Tf, but will retain its native shape. This non-binding TbpA mutant is predicted to elicit a more robust, functional immune response by eliminating antigen epitope shielding by the ligand. If successful, this TbpA mutant has potential to contribute to the development of a vaccine.

This work was supported by the IMSD Research Training Program.

**CHARACTERIZATION OF THE ADHERENCE OF *SNEATHIA AMNII* TO HUMAN VAGINAL EPITHELIAL CELLS**

**Lizette I. Carrasco, Amy L. Sanford, and Kimberly J. Jefferson**

**Department of Microbiology and Immunology, Virginia Commonwealth University**

In the United States, infants born to African American mothers are at greater risk of being born preterm (defined as ≤ 32 weeks gestation) than infants born to Caucasian mothers, indicating a problematic health disparity between racial and ethnic groups. While the risk factors contributing to preterm birth vary, previous studies have defined a clear link between preterm birth and intrauterine infections, which are a consequence of bacterial invasion. Certain bacteria such as *Sneathia amnii* can cause amnionitis and we hypothesize that in order to do this, it is equipped with virulence factors that aid it in its traversal of the fetal membranes. Previous work in our lab demonstrated that *S. amnii* adheres to and decreases the viability of chorionic trophoblasts and vaginal epithelial cells. The aim of this study was to further characterize the adherence and cytotoxicity of *S. amnii* in order to better understand how it invades the amniotic cavity. Bacterial adhesins often recognize sugar residues on epithelial glycoproteins, sowe sought to inhibit bacterial adherence by exposing vaginal epithelial cells to *S. amnii* incubated with various monosaccharides and disaccharides. To further characterize the cytotoxicity, the viability of chorionic trophoblasts and vaginal epithelial cells was observed by Trypan blue staining following incubation with *S. amnii* treated with UV, high heat, or Trypsin. From the adherence studies, we found that mannose, a monosaccharide, caused a 4 fold decrease in adherence. Cytotoxicity studies showed that cells remain alive following trypsin and high heat treatment while cells exposed to UV light died. This suggest that a protein was responsible for the cytotoxic activity. Future work will focus on identification of the putative cytotoxin and adhesin.

This work was supported by the IMSD Research Training Program

STREPOCOCCUS SANGUINIS' GENES ROLE IN

BIOFILM FORMATION

Suquoia Mosby and Xuichun Ge

OMCB, Philips Institute VCU School of Dentistry

Streptococcus sanguinis is a normal habitant in the human oral cavity as well as a pathogen of infective endocarditis. The study intended to examine and see which of genes in S. sanguinis were important for biofilm formation. We screened the S. sanguinis mutant library, and preliminarily got a list of genes involved in biofilm formation. Among these mutants, we chose five to further examine their changes in biofilm formation compared to the wild type SK36. The wild type and mutant strains were cultured in BM medium in polystyrene plate wells to form biofilm under anaerobic conditions. To determine the amount change in biofilm formation, the biofilm was stained by crystal violet and then the OD600 was measured to view biofilm change in structure and other properties, and the biofilm was stained by fluorescent dye SYTO 9 and propidium iodide and observed under confocal laser scanning microscopy. The ANOVA and Dunnett's test (the SK36 as the control) was applied to statistically analyze the change in biofilm formation in the mutants. If the p value < 0.05, there is significant difference in the biofilm formation among the mutants or between the mutants and the wild type; otherwise, there is no significant difference. Compared to the wild type, if the biofilm formation decreases in the mutant, that means this genes are positively associated with the biofilm; otherwise, the gene is negatively involved in biofilm formation. The experiments were performed at using three repeats of each mutant. When we looked at the CV staining, we found a significant difference between the wild type and ssk 1127 (P val. Less than 0.0001), ssk 1301 (P val. =0.0397), and ssk 0816 (P val. Less than 0.0001), and ssk 0613 (P val. Less than 0.0001). The confocal laser scanning microscopy data showed that 1301, 1127, 0613 and 0816 gave a clearer and more even biofilm than 0573. These results suggested that 1301, 1127, 0613 and 0816 may be positively involved in the biofilm formation in S. sanguinis but that gene 0573 is negatively associated with biofilm formation.

This work was supported by the IMSD Research Training Program.

**Effect of Opioid Induced Constipation on Intestinal Microbiome Composition**

**Michael Kiflezghi1, Logan Vogley1, Tricia Hardt Smith2,**

**Hamid I. Akbarali3, Maria Rivera2**

**1Center for the Study of Biological Complexity, 2Department of Biology, 3Department of Pharmacology and Toxicology**

Mammals are home to large and diverse communities of symbiotic bacteria otherwise known as microbiomes. The most diverse of these microbiomes is found in the gut and play a key role in the maturation of the immune and nervous systems. Intestinal dysbiosis, or an imbalance in the diversity of intestinal microbiota, can lead to increased intestinal permeability allowing lipopolysaccharides to enter the blood stream thereby inducing an inappropriate inflammatory response throughout the body. Opioid treatment has been shown to induce constipation by altering colonic motility which may lead to or exacerbate intestinal dysbiosis. One of the aims of this study is to determine whether intestinal motility altered by morphine administration has an effect on mouse gut microbiome composition. Metagenomic DNA from four control mice and four mice with opioid-induced constipation was collected and the whole gut metagenome sequenced using Illumina MiSeq. This sequence data is at the initial analysis stage. The whole metagenome sequence data will be complemented by generating the taxonomic profile of the microbiome via the sequencing of small subunit rRNA amplicons (16s amplicons) using an Ion Torrent PGM sequencer. Currently we are working on developing a qPCR method to quantitate the copies of 16s DNA in the metagenome. An approximation of the 16s copy number will help in assessing possible bias during 16s PCR amplification.

This work was supported by the IMSD Research Training Program.

**EFFECT OF MILD TRAUMATIC BRAIN INJURY ON EYE MOVEMENTS DURING PERFORMANCE OF A MEMORY-GUIDED TASK**

**Zoe Villamar and Paul A. Wetzel, Ph.D.**

**Department of Biomedical Engineering**

**School of Engineering**

**Virginia Commonwealth University**

Mild traumatic brain injuries (mTBIs) can cause cognitive impairment and oculomotor dysfunction. The subjects of this study are military personnel who have been diagnosed with mTBI. Using an eye movement tracking system, this study aims to objectively determine how eye movements are affected by mTBI when performing a memory-guided visual task. Eye movement data were recorded (EyeLink 1000, SR Research) at 500 Hz. From this pool, 15 subjects with mTBI and 15 normal controls were selected for analysis based on the quality of the eye movement recording. For the memory-guided test, a practice target pattern was shown to the subject. After a brief delay, the subject attempted to reproduce the target pattern, as accurately as possible, under two conditions: 1) where the possible target positions remained on, and 2) where the target positions remained off. Data were analyzed based on how accurately they reproduced the target pattern as measured by directional errors (misses) and correlation (similarity). It was found that mTBI subjects had significantly lower correlation coefficient averages compared to controls for both target conditions. For both target conditions, mTBI subjects showed a significantly higher number of misses compared to controls. No significant differences were found between the two conditions within each group. However, the variance was greater when the target positions remained off. These data suggest that mTBI subjects performed less well compared to the normal population. The eye movements suggest differences between the two groups, which suggest that mTBI may have an effect on cognitive behavior. The lack of difference within each condition could be due to potential practice effects or the limited sample size. In the future, more analyses will be done to include the entire sample population.

This work was supported by the IMSD Research Training program.

**MECHANISMS THAT REGULATE STABILITY OF AXONAL DOMAINS**

**Sarah S. Izabel and Jeffrey L. Dupree**

**Department of Anatomy and Neurobiology, Virginia Commonwealth University**

Multiple sclerosis (MS) is an autoimmune disease of the central nervous system (CNS) that affects the brain and spinal cord resulting in inflammatory and immunological events. Microglia are immune cells of the CNS that through phagocytosis remove damaged or unused cells while also presenting repairing properties within the CNS. Microglia contact the axonal initial segment (AIS) and may play a role in establishing it. Work from our lab has shown that microglial-AIS contact is increased during inflammatory insults concomitant with AIS breakdown. Based on these correlative observations, we proposed that microglia can target the AIS for degeneration during an inflammatory insult. Here, we hypothesize that these cells may also develop increased contact with other axonal domains—specifically the paranode. We also postulate that AIS instability during an inflammatory attack may result from decreased neuronal activity, which is consistent with several published injury models. To test both hypotheses, I have employed double immunocytochemical labeling combined with confocal microscopy and used collected maximum projected z-stack images to quantify paranode/microglial contact, paranode disruption, integrity of synaptic boutons on the AIS and AIS integrity. My preliminary data indicate that microglia do not contact the paranode either in the presence or absence of inflammation. For quantitation of synaptic integrity, I have determined that the confocal microscope does not afford sufficient resolution for accurate quantitation. Presently I am undergoing training with the SIM system, which will significantly enhance resolution and allow me to quantify synaptic integrity. From these observations, we have proposed that microglia may determine if similar increased contact between microglia and other axonal domains is induced in an inflammatory environment.

This work was supported by a Veterans Affairs Merit Award and the IMSD Research Training Program.

**Synthetic Model of Cytochrome c Oxidase, the Terminal Enzyme of the Mitochondrial Electron Transport Chain**

**Joel W. Roberts and Heather R. Lucas, Ph.D**.

**Department of Chemistry, Virginia Commonwealth University, Richmond, VA**

Parkinson’s disease (PD) and Alzheimer’s disease (AD) are multifactorial neurodegenerative diseases that are characterized, in part, by mitochondrial dysfunction. Mitochondria are responsible for a plethora of biological processes but their primary function is to produce energy as adenosine triphosphate (ATP) via the mitochondrial electron transport chain (mtETC). Because the brain’s energy requirements are very high, precise maintenance of ATP production is crucial for its function within the body. Research has shown that reduced activity of protein complex IV of the mtETC has been detected in PD tissue along with defects in mitochondrial dynamics, all of which are associated with neurodegeneration. As a result, the mtETC decreases in efficacy resulting in electron seepage and decreased ATP synthesis. Protein complex IV, also known as cytochrome c oxidase (CcO), is the mtETC enzyme involved in the four-electron reduction of molecular oxygen to form water, an important terminal step in the mtETC for maintaining the mt transmembrane potential and promoting proper production of ATP. With aging, CcO becomes more susceptible to damage, leading to the production of reactive oxygen species (ROS) and electron seepage by the mtETC. We aim to synthesize a synthetic model of the heterobimetallic CuB – hemea3 active site of CcO. Our model will include an iron-porphyrin and a copper-bound pyridylamine based ligand that mimics the catalytic site of CcO. Once accomplished, we will explore the ability to activate catalysis with exposure to laser light, as absorption of red laser light by CcO has been reported to increase mitochondrial mobility and improve redox capacity. Through this synthetic model, we aim to elucidate the mechanism responsible for light stimulated re-activation of mt complex IV - the last electron transfer step from cytochrome c to CcO.

This work was supported by the IMSD Research Training Program.

**Mitochondrial Energetics goes Awry in Higher-Order Brain Circuitry when Growing up with Secondhand Smoke**

**Liam Lewis, Pretal Muldoon1 and Andrew K. Ottens1**

**1Department of Anatomy and Neurobiology, Virginia Commonwealth University**

Nearly one-in-five children in the United States continue to be exposed to secondhand smoke, otherwise known as environmental tobacco smoke (ETS). Recent studies show that growing up with ETS increases the risk for cognitive and behavioral deficits, even without prenatal exposure. The Ottens’ laboratory developed an ETS exposure model with which to test biological causation for altered behavior. Findings to date demonstrate that juvenile ETS exposure results in impaired attention, action control and impulsive behavior and that ETS effects mitochondrial energetics within cerebellum, an area of the brain involved in modulating errant activity. The present hypothesis is that ETS induced aberrant mitochondrial energetics throughout the higher-order regulatory loop between frontal cortex and cerebellum. Immunofluorescence microscopy of mitochondria and their fission/fusion proteins was performed within orbital cortex and the mediodorsal thalamic relay interconnected with cerebellum. In orbital cortex, we found that mitochondrial proliferation increased with ETS exposure relative to control in the juvenile brain (P24), but then was reduced relative to control in adolescence (P49). A similar, though less pronounced bimodal effect observed in mediodorsal thalamus points to dysregulated mitochondrial energetics throughout the higher-order cortico-thalamo-cerebellar circuit governing executive control functions.

This research is supported by NIEHS/NIH R21ES023060 and the IMSD Research Training Program.

**BREATHING OZONE WEAKENS THE BRAIN’S DEFENSE**

**AGAINST TOXICITY**

**Demetrius Carter, Pretal Muldoon, & Andrew Ottens**

**Department of Anatomy and Neurobiology, Virginia Commonwealth University**

The highly selective blood brain barrier (BBB) regulates molecular access to and from the brain. Often beneficial, the tight junctions within the endothelial wall of cerebrovasculature act to quarantine the brain from circulating blood factors that may prove toxic. Yet recent research in collaboration with the Ottens’ laboratory has shown that acute inhalation exposure to pollutants can degrade vascular function. Here we extend those findings into the central nervous system (CNS) and hypothesize that acute ozone (O3) will increase BBB permeability. O3 is a primary constituent in urban and industrial air pollution and has been shown to immediately react within the lungs to induced reactive species. Work in the Ottens’ laboratory has newly shown that O3 exposure further induces the release of a complex signature of biomolecules into the blood that they have been found to induced vascular dysfunction. These potentially toxic byproducts circulate in our blood stream and make it into the brain’s cerebrovasculature where it may alter BBB function. Here we tested whether O3 exposure weakened the BBB within rat brain samples. We employed immunofluorescence microscopy with antibodies targeting serum albumin, which is normal excluded by the BBB, and ZO-1, the anchoring protein of endothelial tight junctions. Densitometry is utilized to quantify these proteins relative to one another, with statistical assessment used to determine the effect of O3. We further assessed whether Fasudil administration, a Rho-kinase inhibitor that enhances vasodilation, would protect against O3 induced BBB permeability. Findings from these studies will shape our understanding of how pollution can induce toxic exposure across the BBB and influence cognitive diseases, e.g. Alzheimer disease/senile dementia of Alzheimer type (AD/SDAT), and behavioral disorders, e.g. attention deficit hyperactivity disorder (ADHD)/hyperkinetic disorder (HD).

This work was supported the IMSD Research Training Program.

**Repression of Ɣ-Globin Through KLF1 Regulation**

**Mark A. Hairston, Joshnamaithili Seelam, and Joyce A. Lloyd**

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Krüppel-like factors (KLFs) are a family of transcription factors implicated in cell differentiation and erythroid development. This study focuses on KLF1, specifically expressed in erythroid cells and its role in regulating fetal Ɣ-globin gene expression. Ablation of one KLF1 allele leads to high Ɣ-globin by a mechanism involving the Bcl11a repressor. Fetal hemoglobin (HbF) is an oxygen transport protein giving the fetus access to oxygen from the maternal bloodstream. Postnatally the newborn synthesizes mainly β-globin or Adult hemoglobin (HbA). It would be of interest to establish persistent expression of HbF as it clinically benefits management of the symptoms of sickle-cell anemia and β-thalassemia. A prior study looked at mice carrying a human β-globin locus transgene and compared the Ɣ-globin expression of embryonic day 14.5 (E14.5) KLF1+/- and KLF1+/+ (WT) mice. KLF1+/- showed a higher percentage of Ɣ-globin compared to WT. This also holds true in the late fetal stage. Persistence of Ɣ-globin suggests that other repressors, like Bcl11a, may interplay in the silencing mechanism of Ɣ-globin. In mouse KLF1-/- embryonic cells Ɣ-globin is reduced but in KLF1+/- human adult cells Ɣ-globin is increased. Therefore, the laboratory decided to look at umbilical cord blood-derived erythroblasts and see what happens to Ɣ-globin if KLF1 is reduced. It was found in these cells Ɣ-globin is also increased. The experiment will compare the fetal liver of both KLF1+/- to WT as the switch from Ɣ-globin to β-globin normally occurs here. The present mouse model mated KLF1+/- with WT. Females were dissected on E14.5, embryonic tissue collected, digested and DNA amplified by PCR for the purpose of genotyping. Total RNA was extracted from fetal liver samples to quantify gene expression. Future work will utilize microarrays for expression profiling to measure relative expression levels of numerous genes. By doing so, we can compare how expression of other genes change between KLF1+/- and WT providing supporting evidence of other repressors of Ɣ-globin that are regulated by KLF1.

This work was supported by the IMSD Research Training Program.

**Identification of microRNAs in Blood and Urine as Potential Biomarkers for Prostate Cancer Detection**

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MicroRNAs (miRNA) are small noncoding RNAs that are very important in post-transcriptional regulation. MiRs bind mostly to the 3’-UTR of mRNAs to negatively regulate gene expression at the level of translation. Previous research has shown that miRNAs may either serve as tumor suppressors or oncomirs dependent on what role the genes they target play in cancer progression. Interestingly, miRNAs can be secreted in a variety of protective capsules and as such can act as cellular transduction signals affecting gene expression at secondary sites. The aberrant expression of some miRNAs in patient blood and or urine may be diagnostic for specific cancers and/or even contribute to tumorigenesis. Thus, miRNAs can be relevant biomarkers for identifying and staging cancer. Our laboratory has been focusing on identifying miRNAs in blood and urine as potential biomarkers for prostate cancer (pCa), the second leading cause of cancer death in men in the United States. In this study, we use Illumina ® Next Generation Sequencing to generate miRNA profiles in samples from pCa patients compared to normal individuals. Analysis of deep sequencing reads was conducted through Partek Flow, v3.0 software using Bowtie 2 (v2.1.0) short read aligner with miRBase v. 20 as the reference database for alignment and annotation. An average of 85% of total reads after trimming aligned to the miRbase and the average quality of each read was 38.3 where a value above 30 is thought to be relevant. According to the deep sequencing data, a number of miRNAs are significantly up-regulated or down-regulated. Quantitative real-time PCR (qRT-PCR) is being performed on selected miRNAs to confirm the deep sequencing analysis. Ultimately, it is anticipated that the results from this study will provide considerable insight into improving current pCa diagnosis by offering an alternative method for early detection and progression, since current monitoring of PSA levels has led to many false positive and over treatments with dire outcomes.

This work was supported by the IMSD Research Training Program.

**MUTATIONS IN HIGH THROUGHPUT SEQUENCING ANALYSES OF THE *CRYPTOSPORIDIUM* 60 KDa GLYCOPROTEIN (GP60)**

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**\*University of Chile, Santiago, Chile**

Cryptosporidium is a protozoan parasite known to cause diarrhea in animals and birds. Some *Cryptosporidium* species are capable of cross species infection such as *Cryptosporidium parvum*, which is commonly found in bovines and is known to be capable of infecting humans and other mammals. Understanding the genetic diversity of pathogens is crucial for their control and prevention. Genetic diversity within *Cryptosporidium* is studied through some genetic markers such as its 60 KDa glycoprotein (gp60). The parasite is subtyped into families by sequence analyses of the tandem serine-coding repeats and surrounding nucleotides in gp60. A high throughput sequencing (HTS) experiment was performed with DNA from four isolates from humans and calves to study the parasite’s genetic diversity within the different host. A DNA fragment of about 1200 bp, containing the entire gene was amplified and sent for HTS using Ion Torrent™ (Life Technologies). Because of the inherent property of this technology, the amplified DNA had to be fragmented to about 400 base pairs and consequentially the resulting sequences contained reads based on fragments with random start positions, a majority of which did not contain the serine-coding repeats. The project consisted of using bioinformatics techniques to parse sequences containing only the entire serine-repeat, and a specified number of nucleotides upstream and downstream of the repeated sequences. From the approximately 100,000 reads obtained from each isolate we parsed about 4,000 sequences with complete repeats. Preliminary analyses show that indeed many subtypes may be present within a parasite isolate. The parsed sequences are being analyzed for their polymorphism and other features useful for studying the parasite diversity and evaluation of its biological significance.

This work was supported by the IMSD Research Training Program to MS and FONDECYT Chile 1121035 to SPF, RM, FF and LSO.

**A QUALITATIVE STUDY TO ADDRESS HEALTH DISPARITIES: REDUCING INFANT MORTALITY, PRETERM BIRTH, AND CLOSING THE GAP BETWEEN RESEARCH AND PRACTICE**

**Sage McNett, Allison A. Vanderbilt, Alisa E. Brewer and Marcie S. Wright**

**Center on Health Disparities, Virginia Commonwealth University**

Despite the recent advancements in biomedical research, health disparities impacting the outcomes for disadvantaged social groups remain prevalent in the U.S. such as the high rate of preterm birth and infant mortality among African Americans. Community engagement acts as an intermediate to bridge the gap between research and practice. The purpose of this qualitative study was to collect feedback from community engagement research participants regarding their experience related to a computer based health literacy and preterm birth prevention program. One-on-one interviews were conducted with participants after their completion of project activity. The interview questions were designed to determine what the participants wanted to learn more about in the near future and to determine what they perceived was most valuable to learn based on their prior exposure. Findings indicated three themes: personal / community health, health disparities, and social determinants. These three themes indicate that most participants were unaware of the prominent preterm birth health disparity before the computer based health literacy program, however participants showed a drive to address personal and community health issues and to change their environmental conditions. For example, education and employment to promote empowerment and wellbeing. The data collected from these interviews will be used to guide relevant and culturally sensitive workshops on health topics to promote awareness and community resources to help eliminate health disparities in the African American community in an urban area.

This work was supported by the IMSD Research Training Program and P60MD002256