Curtis L. Parker, Ph.D.
1942-1998

Curtis L. Parker was chairperson of the Department of Anatomy from 1989 until his death in 1998. Dr. Parker served as Associate Dean for Basic Science and Research from 1992 to 1996, and acting Dean from July 1995 to December 1995. He was chairperson of the Student Academic Progress and Promotion Committee and served as chairperson or a member of virtually every major committee at Morehouse School of Medicine (MSM).

Dr. Parker joined our institution in 1983 as Associate Professor of Anatomy and rose to the rank of Professor in 1985. Prior to coming to MSM, he was an Associate Professor at (Clark) Atlanta University from 1981 to 1983 and an Assistant Professor at Bowman Gray School of Medicine from 1975 to 1980.

Dr. Parker was an outstanding biomedical research scientist and a primary facilitator in providing opportunities for aspiring research students to interact with scientists of national and international acclaim. His untiring efforts made it possible for some ninety medical students to participate in the Fellows Program at the Center for Disease Control and Prevention. This program was the catalyst for a Student Research Day Symposium at MSM. The MSM-based Symposium was designed to provide a venue for MSM trainees from all programs, and trainees from other institutions from across the State of Georgia, to present their research findings and experiences.

On November 6, 1998, Dean E. Nigel Harris proclaimed Student Research Day at Morehouse School of Medicine as the ***Curtis L. Parker Student Research Symposium***.

Foreword

All student participants are commended for their industrious efforts in the advancement of science. The Research Symposium Committee encourages your continued pursuits and optimistically anticipates your success.

*Awards**

*The **Curtis L. Parker Award** is given to the most outstanding oral or poster presenter in the Ph.D. student category.*

*The **Graduate Education in Biomedical Science Award** is given to the most outstanding oral or poster presenter in the master student category.*

*The **Graduate Education in Public Health Award** is given to the most outstanding oral or poster presenter in the public health category.*

*The **Honorable Louis B. Stokes Research Award** is given to the most outstanding oral or poster presenter in the undergraduate student category.*

*The **Jay Romans Medical Student Research Award** is given to the most outstanding oral or poster presenter in the medical student category.*

*The **Postdoctoral Research Award** is given to the most outstanding oral or poster presenter in the postdoctoral category.*

**In the circumstance where there are no competitors in a presentation category, the sole presenter will receive a certificate of participation.*

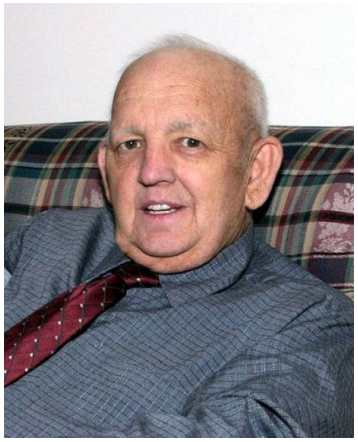
The Honorable Louis B. Stokes, 1925 - 2015

The Honorable Louis B. Stokes, U.S. Congressman (retired) began his political career when he was elected to the U.S. House of Representatives in 1968 making him the first African American member of Congress from the state of Ohio. The thrust of Congressman Stokes' career focused on advocacy for the poor and disadvantaged, especially those in urban America. He served under six Presidents during his 30 years in Congress.

Congressman Stokes led pioneering efforts for minority health; the education of minority health professionals, minority science and engineering professionals; the enhancement of science and engineering infrastructure for research and education at HBCUs; and K-12 mathematics and science education programs focusing on state, urban and rural school districts with significant minority enrollments. Some of the programs for which Congressman Stokes is the architect and sustained sponsor and advocate include the Minority Biomedical Support (now MBRS) Program, Research Centers of Excellence in Minority Institutions, the Office of Minority Health, Research and Minority Health Professions Training Act, and the K-12 Summer Science Camps Program.

Congressman Stokes played a pivotal role in the quest for civil rights, equality and social and economic justice throughout his tenure in the United States Congress. His work in the area of health led to his appointment as a member of the Pepper Commission on comprehensive Health Care, and he was the founder and chairman of the Congressional Black Caucus Health Braintrust.

When the Honorable Louis Stokes retired in 1999, he became the first African American in the history of the U.S. Congress to retire having completed 30 years in office. He was appointed to serve as vice chairman of the PEW Environmental Health Commission at the Johns Hopkins School of Public Health and was appointed by former Health and Human Services Secretary Donna E. Shalala as chairman of the Advisory Committee on Minority Health. Congressman Stokes was the recipient of innumerable distinguished service awards, recognitions, certificates of appreciation, and honorary degrees.



Reverend Earl Jay Romans, 1927 - 2004

Chaplain (Lieutenant Colonel) (retired) Earl Jay Romans was a minister in the Christian Church and served as senior minister at First Christian Church of Jonesboro, GA at the time of his death.

Reverend Romans was known as a "soldier's chaplain" and was loved and respected for his sense of humor and commitment to the men and women who served with him. Always leading by example, he could be found "in the ditches or in the air" with his troops. His trademark at all times, good or bad, was "Thumbs Up!"

During his career Reverend Romans received the following awards: Meritorious Service Medal (4), Army Commendation Medal (2), Master Parachutist Badge, Pathfinder Badge, Army Achievement Medal, National Defense Service Medal, Armed Forces Expeditionary Medal, Army Service Ribbon, and Overseas Service Ribbon.

Reverend Romans wanted to donate his body to the Morehouse School of Medicine for student teaching and research. However, because his body could not be utilized for this purpose, the Jay Romans Medical Student Research Fund was established in an attempt to carry out his wish to support student research at MSM.

KEYNOTE SPEAKER

Barney S. Graham, M.D., Ph.D.

Dr. Barney S. Graham is an immunologist, virologist, and clinical trials physician with an extensive background in basic and translational research applied to vaccine development. He obtained an undergraduate degree from Rice University, a medical degree from the University of Kansas, and completed internal medicine residency, chief residencies, ID fellowship, and Ph.D. in Microbiology and Immunology at Vanderbilt University where he was an R01-funded investigator before joining the NIAID Vaccine Research Center at NIH Vaccine Research Center (VRC) at NIH as a founding member in 2000.

He retired as Deputy Director of the VRC in 2021 and is now an independent consultant and Professor of Medicine and Microbiology, Biochemistry, and Immunology and Senior Advisor for Global Health Equity at Morehouse School of Medicine in Atlanta.

Dr. Graham is an elected member of the American Society of Clinical Investigation, American Association of Physicians, and National Academy of Sciences and has been awarded two honorary doctorate degrees. He is a recipient of the Robert M. Chanock Award for lifetime contributions to RSV research, the Albert B. Sabin Gold Medal Award for contributions to vaccinology, the Albany Medical Center Prize in Medicine and Biomedical Research, the National Academy of Sciences John J. Carty Award for the Advancement of Science, the Charles Mérieux Award for Achievement in Vaccinology and Immunology, the Maxwell Finland Award for Scientific Achievement from the National Foundation for Infectious Diseases, and the New York Academy of Medicine John Stearns Medal for Distinguished Contributions in Clinical Practice. He was named one of the world's 100 most influential individuals and one of the Heroes of the Year in 2021 by Time Magazine and recognized as the Federal Employee of the Year by the Partnership for Public Service.

Dr. Graham is an author on more than 500 scientific publications, and a thought leader on emerging viral diseases and pandemic preparedness. He is best known for his research on RSV pathogenesis, structure-based vaccine design, and application of mRNA delivery technology. He was involved in the advanced evaluation of vaccines and monoclonal antibodies for HIV, Ebola, and Chikungunya, and developed novel vaccines for RSV, influenza, Zika, paramyxoviruses, and coronaviruses including the first COVID-19 vaccine and monoclonal antibody to enter clinical testing and that subsequently achieved Emergency Use Authorization and licensure.

PROGRAM

Wednesday, February 8, 2023

Morehouse School of Medicine
National Center for Primary Care

8:30am - 9:00am **CONTINENTAL BREAKFAST**

9:00am – 9:10am **WELCOME**
Rick Kittles, Ph.D.
Senior Vice President for Research
Professor, Department of Community Health and Preventive Medicine

ACKNOWLEDGEMENTS AND PROCEEDINGS
Gianluca Tosini, Ph.D.
Chief Scientific Research Officer
Professor and Chair
Department of Pharmacology and Toxicology

ORAL PRESENTATIONS

9:15-9:30am	**Xiting Lin**	Abstract #O-01
9:35-9:50am	**Jared Bailey**	Abstract #O-02
9:55-10:10am	**Jamirah Chevrin**	Abstract #O-03
10:15-10:30am	**Leah Arms**	Abstract #O-04
10:35-10:50am	**Karen Aikhionbare**	Abstract #O-05
10:55-11:10am	**Darlene Bio**	Abstract #O-06
11:15-11:30am	**Lasha Clarke**	Abstract #O-07
11:35-11:50am	**Morgan Coleman**	Abstract #O-08
11:55-12:10pm	**Hamdi Abdi**	Abstract #O-09

12:15pm-1:00pm **LUNCH**

1:00pm-2:45pm **POSTER SESSION**

3:00pm **INTRODUCTION OF KEYNOTE SPEAKER**

Valerie Montgomery Rice, M.D.
President and CEO, Morehouse School of Medicine

"Rapid COVID-19 Development and the Future of Vaccinology"

Barney S. Graham, M.D., Ph.D.
Professor, Department of Microbiology, Biochemistry, and Immunology
Senior Advisor, Global Health Equity
Morehouse School of Medicine

4:15pm **PRESENTATION TO KEYNOTE SPEAKER**

4:20pm **THREE MINUTE THESIS® COMPETITION**

1. Kiam Preston, Jr.
2. Kaylin Carey
3. Xiting Lin
4. Melayshia McFadden
5. Aliyah Anderson
6. Mikaili Abdullah

4:45pm **CLOSING REMARKS AND PRESENTATION OF STUDENT AWARDS**

J. Adrian Tyndall, M.D. MPH
Executive Vice President for Health Affairs and Dean
Morehouse School of Medicine

Rick Kittles, Ph.D.
Senior Vice President for Research

ORAL ABSTRACTS

O-01 Title*: Emerging Antibiotic Resistant Patterns in Pediatric Staphylococcus aureus Skin and Soft Tissue Infections*

Authors: Xiting Lin [1], Casey Cazer [2], Robert C. Jerris, PhD [3,4], Traci Leong, PhD [5], Peter T. Baltrus, PhD [6], Mark Gonzalez, PhD [3], and Lilly C. Immergluck, MD, MS, FAAP [3,7]

1. Morehouse School of Medicine, Doctor of Medicine/PhD in Biomedical Sciences Program
2. Cornell University College of Veterinary Medicine, Depart. of Population Medicine and Diagnostic Sciences
3. Children's Healthcare of Atlanta
4. Emory University, Department of Pathology and Laboratory Medicine
5. Emory University, Rollins School of Public Health, Depart. of Biostatistics and Bioinformatics
6. MSM Department of Community Health and Preventative Medicine
7. MSM Clinical Research Center, Department of Microbiology, Biochemistry, and Immunology

Mentor: Lilly C. Immergluck, MD, MS, FAAP, Morehouse School of Medicine

Background/Significance: In 2019, it was estimated that 1.27 million deaths in the US were due to antibiotic-resistant bacteria. Methicillin-resistant *Staphylococcus aureus* (MRSA) is one bacterium that has contributed to the increasing antibiotic resistance, partly driven by its ability to cause both community-onset and hospital-acquired infections. Despite recent reports of decreasing proportion of MRSA infections in children, the antibiotic landscape is evolving with increasing resistance to clindamycin. The most common clinical presentation for community-onset *S. aureus* infections in children is skin and soft tissue infections (SSTIs). We hypothesize that antibiotic resistance patterns, specifically those involving clindamycin, in pediatric community-onset *S. aureus* SSTIs have changed over time.

Methods: This retrospective study (2002-2017) analyzed electronic medical records from patients with *S. aureus* infections seen at Children's Healthcare of Atlanta. Unique patient-year with community-onset SSTIs, < 19 yo, living within the 20-county Atlanta metropolitan statistical area, and complete antimicrobial susceptibility testing results for eight antibiotics (clindamycin, erythromycin, gentamicin, oxacillin, rifampin, tetracycline, trimethoprim-sulfamethoxazole, vancomycin) were included in the analysis. Association mining was applied to single years using the *arules* R package (v1.7.3).

Results: 8,436 cases met the inclusion criteria. Across the 16 years, 27 antibiotic patterns were identified. The set OXA-ERY (i.e., MRSA also resistant to erythromycin) is decreasing in frequency over time and closely matches the temporal trend for MRSA. Prevalence of CLI-ERY is increasing since 2005, mostly contributed by methicillin-sensitive *S. aureus* (MSSA).

Conclusions and Implications: Resistance to clindamycin and erythromycin has been increasing, more so in MSSA than MRSA. Clindamycin is recommended as a first-line therapy for *S. aureus* SSTIs as it works against both MRSA and MSSA. However, our findings suggest more research may be needed to determine whether this antibiotic would be the most appropriate and a more nuanced approach for treating *S. aureus* SSTIs may be beneficial.

Acknowledgment of Funding: PHS Grant UL1TR002378 from the CTSA Program, NIH, Grant Number G12-RR03034 from NIMHD, NIH, and Grant Number HS024338–01, K-08 Mentored Clinical Scientist Award, Agency for Healthcare Research & Quality.

O-02 Title: ***Black Breast Cancer Survivors' Sociocultural Perspectives of Beauty, and Use of Personal Care Products Containing Endocrine Disrupting Chemicals***

Authors: **Jared Thomas Bailey** BS[1] , Marissa Ericson, PhD[2], Tiah Tomlin-Harris[3], Dorothy Galloway, BS[4], Lenna Dawkins-Moultin, PhD[5], Adana A.M. Llanos, PhD[6] , Lindsey S. Treviño, PhD[4], Susanne Montgomery, PhD[7], and Dede K. Teteh, DrPH[8]

[1] Morehouse School of Medicine, [2] University of Southern California, [3] My Style Matters, [4] City of Hope Comprehensive Cancer Center, [5] MD Anderson Cancer Center, [6] Columbia University, [7] Loma Linda University, [8] Chapman University

Mentor: Dede K. Teteh, DrPH, MPH, CHES, Chapman University

Background/Significance: The passage of the CROWN Act prohibits hair texture and style discrimination based on race or national origin, thus, theoretically reducing structural barriers to economic mobility. Regardless, hair is synonymous with Black women's identities. Possibly due to society's afro-political ideologies of beauty, Black women tend to use more hair products compared to other racial groups. These standards include social structures that affect self-mediated worth, as well as structural and interpersonal racism based on appearance and societal status. The use of personal care products containing endocrine disrupting chemicals (EDCs) has been shown to increase Black women's breast cancer risk. The *Black identity, hair product use, and breast cancer scale* (BHBS) was developed to measure the sociocultural constructs associated with Black women's hair product use and perceived breast cancer risk. The purpose of this study was to validate the BHBS and examine hair product use among Black breast cancer survivors.

Methods: Participants (N=162) completed a 27-item survey between 2020 and 2022 via a community-based participatory research project—*Bench to Community Initiative*. Principal component analyses (PCA) and confirmatory factor analysis (CFA) were used to establish the underlying component structures and determine the model fit. Chi-square tests were used to determine associations between BHBS subscales and product use, with a p-value <0.05 defined as statistically significant. Products evaluated included *washout and leave-in conditioners*, *salon, and do-it-yourself (DIY) relaxers*, and *salon and DIY hair dyes*. Response options were *used daily through several times a year*, *used but stopped*, and *never used*.

Results: Participants were African American (90%) and African or Caribbean (10%) Black breast cancer survivors. The mean age (standard deviation [SD]) and stage of diagnosis (SD) was 37.4 ± 8.8 and 1.9 ± 0.97, respectively. PCA yielded two components that accounted for 63% of the total variance in the model. Five items measuring *sociocultural perspectives about hair and identity* (subscale 1 [S1]) accounted for 28% of the total variance (α = 0.73, 95% CI 0.71, 0.82). Six items assessing *perceived breast cancer risk related to hair product use* (subscale 2 [S2]) accounted for 35% of the total variance (α=0.86, 95% CI= 0.81, 0.94). CFA confirmed the two-component structure (Root Mean Square Error of Approximation = 0.034; Comparative Fit Index = 0.93; Tucker Lewis Index = 0.89). On average, participants used hair products *daily–yearly,* including conditioners (64%), relaxers (32%), and hair dyes (33%). The use of *salon relaxers* was significantly associated with BHBS subscales (S1and S2). Similarly, *salon hair dye* was significantly associated with S2 of the BHBS.

Conclusions and Implications: The BHBS is a valid measure of sociocultural perspectives associated with hair product use and perceived risk for breast cancer. Given that hair remains an important cultural expression within the afro-political confines of identity, the health impacts of hair products containing EDCs used to craft these identities should be considered in intervention planning.

O-03 Title: ***A Systematic Literature Review: Society, Genomics, and Black Cancer-related Outcomes and Mortality; Intrinsic Reversibility for Cancer Prevention***

Authors: **Jamirah Chevrin, B.S.** and Brian M. Rivers, Ph.D., MPH

Cancer Health Equity Institute
Morehouse School of Medicine

Mentor: Brian Rivers, Ph.D., MPH
Morehouse School of Medicine

Background/Significance: Black persons residing in the United States have unique life experiences that may serve as an adverse trigger for toxic cellular processes, contributing to disparate cancer health outcomes. In previous studies, racism has been associated with severe chronic stress resulting in systematic chronic inflammation and allostatic load that may lead to cancer-causing modifications that serve as contributing factors to increased risk of developing cancer. Our research will address whether allostatic load is more prevalent in Blacks and if allostatic load affects cancer health outcomes. We will also identify factors associated with higher allostatic load, and if the effects of high allostatic load are reversible. This systematic review provides a comprehensive evaluation of these associations, interventions, and prevention strategies.

Methods: This is a scoping review design. The "PubMed" database was searched for articles published through July 2022. We only considered studies that addressed allostatic load, systematic chronic inflammation, or interventions among adult Black people at risk for or current cancer patients in the United States.

Results: Forty studies met inclusion criteria for this review. Higher rates of allostatic load and systematic chronic inflammation was found to be significantly present in Blacks. Higher allostatic load among Black women cancer patients was also found to be associated with increased odds of poorer tumor differentiation and larger tumor size. In a study of Black women at higher risk for cancer, effective interventions for reducing allostatic load were found to be increased exercise and consumption of lower sodium and balance diets. There have not been interventions that analyzed systematic chronic inflammation in Black cancer patients.

Conclusions and Implications: Findings from this systematic review indicates the contribution that structural, interpersonal, and personally mediated racism in the United States has on chronic stress and allostatic load leading to cancer health disparity gaps in Blacks. Lifestyle change interventions were found to be effective. Although intrapersonal interventions have been effective, further research must be conducted on structural interventions to address the multi-factorial contributors to chronic stress in Blacks. The findings of this review strongly indicate the necessity for developing multi-level intervention strategies that address negative social and structural determinants of health afflicting cancer outcome disparities in Blacks in the United States.

O-04 Title: ***Avoiding Incidence of Maternal Mortality by Normalizing Cultural Competency***

Authors: **Leah Arms**, PA-S[1], Olinese Augustin, PA-S[1], Jessica Joyner, PA-S[1], Christine Miller, PA-S[1], Cassandra Riley, PA-S[1], Kayla Sams, MPH, PA-S[1], and Shameka White, PA-S[1]

[1] Morehouse School of Medicine, Physician Assistant Studies Program

Mentor: Sharon Rachel, MA, MPH
Morehouse School of Medicine

Background/Significance: Culturally competent healthcare providers are critical to the quality of care and life among Georgia women. Racial disparities in maternal mortality are multifactorial, but it is necessary to identify the provider factors. The purpose of this research is to assess the association between cultural competency and maternal mortality incidence. Our research question is 'does having access to culturally competent providers reduce the risk of maternal mortality?'

Methods: Our goal is to create a health literacy app (MediCelp) to decrease incidence of maternal mortality in underserved communities. This app will allow users to define cultural competency and locate culturally competent providers. Using app data, usage, reviews, and pregnancy outcomes, we will measure contributing factors by comparing the incidence of maternal mortality after our intervention against the current rates. Finally, we will provide training sessions for providers to enhance their cultural competency.

Results: Individuals using our app will have a greater understanding of what culturally competent care looks like and how to access it without restrictions. We expect that use of our app will be associated with decreased maternal mortality in underserved communities by allowing users to research and locate culturally competent providers in their area. In-app training will also allow providers to enhance their cultural competency knowledge. We hypothesize that increased understanding of cultural competency by providers and patients will be associated with reduced incidence of maternal mortality.

Conclusions and Implications: High rates of maternal mortality are an important assessment of societal growth. Our goal is to reduce maternal mortality by avoiding preventable deaths amongst Georgia women. This goal is achievable by increasing health literacy in underserved communities using MediCelp app data to access culturally competent providers. Implications of this study include confidence in making informed healthcare decisions and bridging the gap between socioeconomic status and quality of care.

O-05 Title: ***Transgenic Transparent Zebrafish Strain Development as a Model to Delineate Microglia Function in the CNS***

Authors: **Karen Aikhionbare**[1] and Surendra Rajpurohit, PhD[2]

[1] Department: College of Science and Mathematics and Honors Program, Augusta University, Augusta, Georgia
[2] Department: Georgia Cancer Center, Augusta University

Mentor: Surendra Rajpurohit, PhD
Georgia Cancer Center, Augusta University, Augusta, Georgia

Background/Significance: The microglia, resident macrophages of the brain, have a high degree of plasticity with a multitude of functions including responding to damaged or infected tissues in the central nervous system (CNS). Zebrafish are an important model organism for studying microglia due to its advantages of *in-vivo* imaging and genetic manipulation in the laboratory setting. The Casper mutant zebrafish is a naturally occurring variant which expresses transparent skin pigmentation and allows for in-vivo imaging. A genetically modified zebrafish strain in which the microglia have been tagged by a red fluorescent dye known as mpeg1: mcherry FP allows for the functional role of the microglia in the central nervous system function to be observed. The aim of this study was to utilize the zebrafish as a model organism to observe the behaviors of the CNS microglia due to the advantages of in-vivo imaging and genetic manipulation in the laboratory setting.

Methods: The goal was to develop a transgenic transparent fish by crossbreeding zebrafish with the mpeg1: mcherry FP transgenic strain and the Casper mutant strain. The benefit of this strain is that the microglia would be tagged by a red fluorescent dye known as the mpeg1: mcherry FP and be able to be imaged *in-vivo*.

Results: After crossbreeding the zebrafish strains to the F03 generation 81% of the offspring were positive for the microglia tagged with mpeg1: mcherry and 11% of the offspring displayed the Casper phenotype. The offspring will be further crossbred to establish a homozygous line for the microglia tagged with mpeg1: mcherry and Casper phenotype.

Conclusions and Implications: The transgenic (red florescent tagged microglia) transparent zebrafish line developed in this study has the potential to be utilized as a model organism to investigate the roles of microglia associated oncogenic diseases that could aid in the development of cardio-oncology therapeutics.

Acknowledgment of Funding:
Extramural Grant Awarded: Investigator Initiated Grant 2022 and Foundation Grant: Kirk Kimmerling Foundation 2022 were awarded to Surendra Rajpurohit.

O-06 Title: ***Food Insecurity Among Medical Students: A pilot study of a Southern HBCU***

Authors: **Darlene Bio, MPH**, Fengxia Yan M.D., and Robina Josiah Willock, Ph.D., MPH

Department of Community Health and Preventive Medicine, Morehouse School of Medicine

Mentor: Robina Josiah Willock, Ph.D., MPH, Morehouse School of Medicine

Background/Significance:
Lack of food security is a persistent public health issue in the United States. Families who are food insecure lack predictable access to foods that are safe and nutritious and support an active and healthy lifestyle. Food insecurity among students particularly concerning due to its deleterious impact on their general health and academic outcomes. The primary objective of this pilot study was to assess food insecurity among all undergraduate medical students attending a Southern Historically Black College/University.

Methods:
A cohort of 427 undergraduate medical students were recruited to complete a survey of food security in June 2022. Food security status was assessed using the United States Drug Administration (USDA) U.S. Household Food Security Survey Module, and the Four Domain Food Insecurity Scale (4D-FIS). We also collected student demographic data and responses to new study-specific survey questions. The Chi square test of associations between key demographic variables and food security was conducted using IBM SPSS Statistics.

Results:
Of the 62 students (14.5%) who responded, 21 (33.9%) were identified as having either *Low* or *Very low food security* households by the USDA tool, and 61 (98.4%) as *Very Food Insecure* by the 4D-FIS tool; both notably higher than the 13% food insecurity reported for GA households. We also found that: 1) years since undergraduate graduation and 2) family household income were significantly associated with food insecurity. Students indicated that lack of time and availability of alternatives to current healthy campus options were barriers to food security.

Conclusion and Implications:
Food insecurity is notable among medical students surveyed and higher than the GA state levels. The USDA survey is not designed to capture the psychological aspects of insecurity, which may be more applicable to medical students. These findings signal the need for further research among a larger sample of students.

O-07 Title: ***Association of adverse childhood experiences and gendered racial stress among pregnant Black Women***

Authors: **Lasha Clarke, Ph.D., MPH** [1], Carol Hogue Ph.D., MPH [2], Tene Lewis Ph.D. [2], Anne Dunlop M.D., MPH [3], and Michael Kramer Ph.D., MPH [2]

[1] Morehouse School of Medicine, Center for Maternal Health Equity
[2] Emory University, Rollins School of Public Health
[3] Emory University, School of Medicine

Mentor: Natalie Hernandez, Ph.D., MPH
Morehouse School of Medicine

Background/Significance: Exposure to adverse childhood experiences (ACEs) is a risk factor for adverse birth outcomes, although the mechanisms by which they act have not been fully explored. As Black women and their infants are known to be at disproportionate perinatal health risk, it is imperative to understand the role of ACEs in proliferating types of stress specific to American Black women's sociohistorical context. One such type of stress is gendered racism, which suggests that, by virtue of being both Black *and* women, Black women uniquely experience psychosocial stressors that go beyond reports of racism. It is unknown whether ACEs confer risk for gendered racial stress among pregnant Black women. We examined associations between maternal ACEs, the intersectional construct of gendered racial stress, and coping responses among socioeconomically diverse Black women.

Methods: In this cross-sectional study of 405 pregnant Black women, we assessed gendered racial stress, ACEs, and response to unfair treatment via self-report. Women were enrolled if they were receiving prenatal care at one of two Atlanta-area hospitals.

Results: 82% experiences at least one ACE; 21% experienced 4 or more ACEs. Increased ACEs were associated with increased gendered racial stress scores (b=4.3, SE b=.49, CI b=3.3-5.3, p<.0001), particularly among those who reported passive versus active responses to unfair treatment (likelihood ratio test, p=.009). Model adjustment did not attenuate the magnitude of the associations of ACEs with gendered racial stress.

Conclusion and Implications: This study demonstrates novel associations between childhood adversity and gendered racial stress among pregnant Black women, which are moderated by learned coping responses. Thus, we enhance the call for inequities in Black maternal health to be targeted intersectionality, at multiple levels (inclusive of medical, political, social, and institutional factors), and at multiple points over the life course when coping tools are developed.

O-08 Title: ***Characterization of Novel Anti-HIV-1 Protein from Momordica balsamina Leaf Extract***

Authors: **Morgan Coleman[1]**, M. Powell[1], M. Khan[1], K. De Barros[1], V.C. Bond[1], E. Gbodossou[2], A. Diop[3], H. Duong[1], V. Floyd[1], and K. Kondwani[1]
[1]Dept Microbiology, Biochemistry and Immunology, Morehouse School of Medicine
[2]PROMETRA International, BP 6134 Dakar-Etoile, Senegal.
[3]Malango Traditional Healers Association, Fatick, Senegal.

Mentor: Michael Powell, Ph. D., Morehouse School of Medicine

Background/Significance: As the incidence of chronic illnesses and drug-resistant pathogens rises, researchers should focus on traditional folk medicine as a viable and sustainable treatment option. One such chronic illness is HIV/AIDS (human immunodeficiency virus / acquired immunodeficiency virus). To that end, our lab is exploring a traditional Senegalese remedy to treat HIV-1 infection through a partnership with an organization called PROMETRA International. The aim of this study was to determine if extracts of *Momordica balsamina* (*M. balsamina*) leaves have anti-viral activity and to determine the nature of the active ingredient(s) responsible for this activity.

Methods: *M. balsamina* leaves were crushed, boiled, and dried into a powder to be used for experiments. To determine if the herbal extract contained antiviral activity, we infected HeLa-CD4+-LTR-βgal cells with NL4-3 and treated them with varying doses of the extract. Antiviral activity was determined using multinuclear activation of a-galactosidase indicator (MAGI) infectivity assays. The data was used to generate a dose-response curve and to determine the therapeutic index. Cytotoxicity was determined using the 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyl-2H-tetrazolium bromide (MTT) assay as instructed by the manufacturer. Relative solubility and polarity of the active agent, we performed direct and fractionated extractions into water and various organic solvents. The anti-HIV-1 activity of each extraction was determined using MAGI infectivity assays. To determine the approximate size of the active agent, we passed the extract was passed through 3kD, 10kD, 30kD, and 100kD Amicon molecular weight cut-off filters. The retained liquids and filtrates for each were tested for anti-HIV-1 activity using MAGI infectivity assays. Based on the data, we ran a 4-20% SDS-PAGE gel and stained it with coomassie brilliant blue (CBB) to detect the presence of proteins.

Results: We found that M. balsamina leaf extract inhibits HIV-1 infection by >50% at concentrations of 0.02 mg/mL and above and is not toxic over its inhibitory range (0–0.5 mg/mL). We observed significantly more antiviral activity in direct water and acetonitrile extractions ($p \leq 0.05$). We also observed significantly more antiviral activity in the aqueous phases of ethyl acetate, chloroform, and diethyl ether extractions ($p \leq 0.05$). Though most of the antiviral activity partitioned into the aqueous layers, some antiviral activity was present in the organic layers. We show that the active agent in the plant extracts is at least 30 kD in size. Significantly more antiviral activity was retained in 3, 10, and 30 kD molecular weight cutoff filters ($p \leq 0.05$). In contrast, most of the antiviral activity passed through the 100 kD filter ($p \leq 0.05$).

Conclusions and Implications: Because the active anti-HIV-1 agent presented as a large, amphiphilic molecule we ran the purified extract on an SDS-page gel. We show that the anti-HIV-1 activity in the leaf extracts is attributed to a 30 kDa protein we call MoMo30.

O-09 Title: ***Adverse Childhood Experiences (ACEs) in adult patients at hospital-based primary care clinics***

Authors: **Hamdi Abdi**[1], Debby Song[2], and Marshall Fleurant[3]

[1] Morehouse School of Medicine
[2] Medical College of Georgia AU/UGA Medical Partnership
[3] Emory University School of Medicine, General Internal Medicine

Mentor: Marshall Fleurant, M.D., Emory University

Background/Significance: ACEs are events or circumstances that occur in childhood and negatively affect individuals' mental and physical wellbeing. ACEs are positively associated with higher disease morbidity and poor outcomes for chronic illness. Multiple studies indicate that ACEs are prevalent at a national level with 60% of patients reporting one or more ACEs; scores of ≥4 are correlated with multifold increases in disease risk. In our study, we looked at the associations between ACE scores in adult patients and their demographic, socioeconomic, and medical information.

Methods: We surveyed 360 adult patients at two hospital-based clinics during appointments with their PCP. ACEs were scored using a ten-item questionnaire developed by Felitti, et al. Each item was scored as the presence of absence of exposure; ACE scores ranged from 0 to 10. We obtained the patients' demographic information and medical history from the EMR.

Results: In an analysis of 200 patients, we found that ACEs were prevalent, with 80.3% of patients reporting an ACE score of one or more. 25% of patients scored ≥4. The mean ACE score was 2.36. In a stratified analysis, we found that there is significant association between employment and ACE score and between depression and ACE score. Questionnaire items with the highest percentage of affirmative responses were those in the household dysfunction category including parental separation, substance abuse and incarceration; items regarding verbal abuse and neglect also ranked highly.

Conclusions and Implications: Based on the results of the univariate testing stratified analysis, depression and employment may be important variables to consider when looking at factors associated with ACE score.

Acknowledgement of Funding: Morehouse School of Medicine, Grady Memorial Hospital Student Trauma and Resuscitation (STaR) Research Program

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POSTER ABSTRACTS

P-01 Title: ***Expression of FOSL1 is associated with worse survival in glioma patients***

Authors: **Hannah Akpobiyeri** and Mingli Liu, MD, PhD

Department of Microbiology, Biochemistry, & Immunology, Morehouse School of Medicine

Mentor: Mingli Liu, MD, PhD, Morehouse School of Medicine

Background/Significance: Glioblastoma (GBM) is considered one of the deadliest and most treatment-resistant cancers with a five-year survival rate of about 6.8 percent. (National Brain Tumor Society). FOSL1, encoding FRA-1, is an AP-1 transcription factor with prognostic value in human solid tumors such as breast, lung, pancreatic, and colon cancer, where its overexpression correlated with tumor progression or worse patient survival. FOSL1 controls cancer cell proliferation and survival, it acts as a master switch of epithelial-to-mesenchymal transition. In agreement with other studies, our results showed that FOSL1 is responsible for sustaining glioma cell growth and glioma cell invasion *in vitro* and is an unfavorable prognostic factor for GBM patients.

Methods: Analyses were conducted using Tissue Microarray. Immunohistochemistry staining was also performed on 5µm thick microarray slides. The immunohistochemical staining for FOSL1 was done using the rabbit monoclonal anti-FOSL1 antibody, which is specific for FOSL1, and a streptavidin-biotin unlabeled immunoperoxidase technique with diaminobenzidine as chromogen for FOSL1. Meyer's hematoxylin was used as a nuclear counterstain. The staining intensity of cells in tissue microarrays was evaluated as negative or positive in three different bright fields. Semi-quantitative HSCORE was calculated for FOSL1. Immunohistochemically stained slides were blindly reviewed and scored by two independent investigators.

Results: FOSL1 protein is expressed in grade II astrocytoma, grade III astrocytoma, and grade IV GBM, but is undetectable in normal brain tissues. Quantification of the IHC results shows that FOSL1's positive nuclear staining is significantly higher in grade IV GBM, grade III gliomas, and grade II gliomas compared to that of normal brain tissue. When FOSL1's expression in different grades of glioma was compared, we found that FOSL1's nuclear staining is significantly increased in grade IV GBM compared to that in grade II gliomas. The positive correlation between increased FOSL1 protein expression and glioma grades strongly indicates FOSL1's potential as a negative prognostic marker in glioma patients.

Conclusions and Implications: FOSL1 functioned as a tumorigenic gene in glioma pathogenesis, which was highly expressed in glioma tissues, and was shown to be an unfavorable prognostic factor for glioma patients.

P-02 Title: ***Disparities in lung cancer incidence and outcome***

Authors: **Briana A. Brock,** Hina Mir, PhD, and Shailesh Singh, PhD

Morehouse School of Medicine, PhD in Biomedical Sciences Program
Morehouse School of Medicine, Department of Microbiology, Biochemistry, and Immunology
Cancer Health Equity Institute

Mentor: Shailesh Singh, PhD, Morehouse School of Medicine

Background/Significance: Lung cancer is the second most common cancer and the leading cause of cancer-related deaths worldwide, irrespective of gender. African Americans (AAs) are disproportionally affected by lung cancer, compared with all other racial and ethnic groups in terms of incidence and survival. Disproportionate diagnosis of aggressive disease and poor survival among AAs suggest that racial differences in the biology of lung cancer and socioeconomic status may be the cause of the disparity.

Methods: We have used a PubMed search to ascertain the factors contributing to the disparity in lung cancer incidence and therapeutic outcome.

Results: There is a growing consensus that considerable variation in incidence and death rates among the different racial and ethnic groups is due to the interaction of genetic and environmental factors. African American men have the highest incidence and death rate in the United States, followed by Caucasian men. In women, the highest rates are in Caucasian women, followed by American Indians. Clinical trials suggest that first-line EGFR tyrosine kinase inhibitors (TKIs) respond differently due to the differences in EGFR and k-ras mutation. In addition, racial disparities in the tumor microenvironment also contribute to the therapeutic efficacy of immune-based therapeutics. Studies have shown higher suppressor cell infiltration in AA lung cancer compared to EA, which may contribute to aggressive disease and poor response to immune-based therapeutic in AA compared to EA.

Conclusions and Implications: To address the disparity in lung cancer, better understating racial differences in the biology of lung cancer and identification of socioeconomic factors contributing to early diagnosis, decision making, and participation in clinical trials are needed

Acknowledgment of Funding:
Number U54 CA118638 provided by NIH NIC

P-03 Title: ***Molecular mechanism of targeting transcriptional co-factors in ERG-positive Ewing Sarcoma and leukemias***

Authors: Shubhalaxmi Kayarthodi[1], **Joshua Byland**[1], Sepehr Serbian[1], Clopton Matthew[1], Yasuo Fujimura[1], Binu Tharakan[2], Veena N. Rao[1], and E. Shyam P. Reddy[1]

[1] Morehouse School of Medicine, Cancer Biology Program, Department of OB/GYN
[2] Morehouse School of Medicine, Department of Surgery

Mentor: E. Shyam P. Reddy, Ph.D., Morehouse School of Medicine

Background/Significance: Ewing sarcoma (ES) is an aggressive and metastatic malignancy that occurs in the bone or soft tissue. There is a clear health disparity seen among Blacks and Hispanic population compared to White population. The survival rates for black and white Hispanic patients have shown to be significantly worse compared to white patients. Drs. Reddy and Rao discovered ERG (ETS Related Gene) and have shown that ERG gene encodes for sequence specific transcriptional activators. Our preliminary results have shown that EWS-ERG inhibits transcriptional activation of RXR. To understand the molecular mechanism of action of how the fusion protein targets nuclear receptor function and to provide a clue for the cancer health disparity seen in ES, we hypothesized that the aberrant fusion protein EWS-ERG inhibits the binding of RXR with CBP and other members of the transcriptional machinery causing transcriptional repression of RXR activity.

Methods: We propose to study the interaction of EWS- ERG and CBP by co-immunoprecipitaion approach and also investigate whether this interaction has a role in the inhibition of RXR transcriptional activity by luciferase assays.

Results: 1. EWS-ERG inhibits RXR transcriptional activity. 2. EWS-ERG interacts with CBP in ES cells. 3. Overexpression of CBP relieves EWS-ERG inhibition. 4. Histone deacetylases (HDACs) inhibitors, Trichostatin A (TSA) and Valproic acid (VPA) relives the EWS- ERG inhibition of RXRα transcriptional activity.

Conclusions and Implications: The transcriptional co-factor (CBP) is sequestered by the aberrant fusion protein EWS-ERG which inhibits the binding of RXR with CBP and other members of the transcriptional machinery causing transcriptional repression of RXR activity. Anti-epileptic drug VPA targets Ewing Sarcoma. This study may provide a clue for the cancer health disparity seen in ES family of tumors.

Acknowledgement of Funding:
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P-04 Title: ***Cannabis use and depression: a systematic review and meta-analysis***

Authors: **Victoria Churchill, PhD, MPH**[1], Lucy Popova, PhD[2], Claire A. Spears, PhD[2], Terri Pigott, PhD[2,3]

[1] Cancer Health Equity Institute, Morehouse School of Medicine
[2] School of Public Health, Georgia State University
[3] College of Education and Human Development, Georgia State University

Mentor: Terri Pigott, PhD, Georgia State University

Background/Significance: Cannabis is the most widely used illicit drug, with approximately 2.5% of people worldwide currently using cannabis in some form. In 2020 there were 2.8 million new cannabis users in the US, of which about 1 million were adolescents aged 12-17, according to the National Survey on Drug Use and Health. However, there are gaps in our knowledge of the potential harms of cannabis, including understanding the relationship between cannabis use and mental health. This project updates a previously published review and meta-analysis looking at the development of depressive symptomology following cannabis use.

Methods: A systematic review of the literature concluded on April 22, 2022. The initial search started with articles that cited the original review by Lev-Ran et al, 2019. Searches were then conducted in PubMed, Ovid Medline, and Google Scholar. Gray literature was searched using ProQuest for unpublished theses and dissertations. Titles were reviewed, and those that mentioned cannabis and depression were then pulled for abstract/full-text screening. Data were extracted from the eligible studies, including information on how the study measured cannabis use and depression. A meta-analysis of the studies was conducted in R using a random effects model and REML estimation. Due to the presence of dependent effect sizes, robust variance estimation was used for the analysis.

Results: Overall, there were 22 studies included in the meta-analysis that met the eligibility criteria. There were multiple effects nested within studies, so a correlated effects model with small-sample correction resulted in an estimated mean log-odds of depression after cannabis use of 0.221 (SE= 0.0623, t=3.55, df=14.6). This corresponds to an OR of 1.25 (95% CI: 1.09, 1.42). The T^2 was 0.0372 and the I^2 was 56.09%.

Conclusions and Implications: Based on the results of the meta-analysis, cannabis users have an increased odds of developing depressive symptomology compared to non-user controls. However, further research needs to be conducted to investigate how different factors in cannabis use modality and strength may impact this association.

P-05 Title: *Secretion modification region-derived peptide and its effect on breast cancer cells*

Authors: **Miriame Gamra**[1], and Ming B. Huang, M.D., M.S.[1]

[1]Morehouse School of Medicine, Department of Microbiology, Biochemistry, and Immunology

Mentor: Ming B. Huang, M.D., M.S.

Background/Significance: According to the CDC report, breast cancer is listed as the number one new cancer cases in the United States and is second in terms of cancer deaths in 2019. Currently, there is no cure for metastatic breast cancer. Studies have found that the epithelial-to-mesenchymal transition (EMT) is a key player in the initiation and progression of cancer and metastasis by the process of releasing exosomes from infected cancer cells. The research aim of this study is to understand the mechanism of exosome release is modulated in secretion modification region (SMR) peptide treated cancer cells. We hypothesize a negative correlation between exosome release and SMRwt treated cancer cells.

Methods: Analyses were conducted using four SMRwt treated and non-treated breast cancer cell lines: ER/PR negative (MCF-7), HER2 positive (BT474), triple negative (MDA-MD-231) and normal human breast epithelium (MCF10A). The cells were prepared by cell culture and cell counting followed by exosome isolation. The concentration and size of the exosomes were then analyzed using NanoSight technology. Gene expression analysis was performed to detect reduced gene expression from isolated exosomes.

Results: The size and concentration of exosomes present in untreated breast cancer cells were MDA-MB-231: 46 nm and 62.87 x 10^8 particles/mL; MCF-7: 45 nm and 83.92 x 10^8 particles/mL; MCF-10A: 49 nm and 45.30 x 10^8 particles/mL. 122 out of 776 genes identified in breast cancer cells as important in breast cancer metastasis. SMRwt treated MDA-B-231 and BT474 breast cancer cell line show significant decrease in expressed Vimentin.

Conclusions and Implications: The findings suggest SMRwt peptide reduces Vimentin expression in exosomes of breast cancer cells and changes the composition of or reduces the release of exosomes in those cells. Therefore, SMRwt peptide may prevent breast cancer metastasis with the reduction of a key EMT biomarker, Vimentin. Further research is needed for informative analysis on other breast cancer related genes such as Mortalin, Hsc70 and Myosin10. In addition, further investigation is needed into the therapeutic value of SMR peptide in cancer metastasis.

Acknowledgement of Funding:
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P-06 Title: ***An Examination of Hookah Usage and Its Association with Lung Cancer Risk in the United States: A Literature Review***

Authors: Kamal Hughes [1] and Brian Rivers, PhD, MPH [2]

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[2] Morehouse School of Medicine, Cancer Health Equity Institute

Mentor: Brian Rivers, PhD, MPH, Morehouse School of Medicine

Background/Significance: Lung cancer continues to be one of the most prevalent forms of cancer in the United States. An estimated 236,740 new lung cancer cases and 130,180 lung cancer-related deaths will occur in 2022 alone. Although on the decline, cigarette smoking remains the most substantial risk factor for lung cancer. Meanwhile, waterpipe tobacco smoking (WTS), also known as hookah, is a widespread, alternative modality for tobacco use. As of 2020, WTS prevalence in the US was at an estimated 2.6 million adults. However, little research has thoroughly investigated hookah use and its association with lung cancer risk.

Methods: A literature search of articles was performed utilizing the biomedical database: PubMed. Inclusion search parameters included articles published between September 2012 and March 2022. Search keywords primarily consisted of a combination of "waterpipe tobacco smoking," "tobacco," "hookah," "lung cancer," and "United States or US.".

Results: The findings showed that WTS was associated with lung cancer risk. Research showed that WTS is most prevalent in adolescents and young adults, higher among men than women, and Blacks and Hispanics are at increased odds of smoking hookah than their white counterparts in the US. Moreover, waterpipe tobacco has similar carcinogenic substances present in cigarette smoke. WTS usage patterns typically extend between 45-60 minutes in which users can potentially inhale 100-200 times the amount of smoke compared to a single cigarette.

Conclusions and Implications: WTS is becoming a growing substitute for traditional cigarette smoking. As hookah stores and lounges continue to emerge, the increasing access and social popularity pose a significant risk to lung cancer incidence in America. Researchers should conduct more substantive research to quantify key measures of WTS prevalence and average frequency of use and produce more accurate demographic data on WTS use. Furthermore, there needs to be a more concerted effort to implement WTS interventions to stifle the growing use in adolescents and young adults.

Acknowledgment of Funding: Award Number: U54 CA118638 NIH, NCI

P-07 Title: ***HER2 Expression in African American Men with Prostate Cancer***

Authors: **Nicole Mavingire, PhD**[1], Mya Walker[2], Jabril R. Johnson, PhD[3], Isaiah J. Sailors[2], Greisha L. Ortiz-Hernandez, PhD[2], Rachel N. Martini, PhD[4], Melissa B. Davis, PhD[4], Justin Tran[2], Frank A. Myers, MD[5], Zhirong Yin, MD[2], Peter Kuhn, PhD[6], Tanya B. Dorff, MD[2], Rick A. Kittles, PhD[3], and Leanne Woods-Burnham, PhD[1]

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Mentor: Leanne Woods-Burnham, Ph.D., Morehouse School of Medicine

Background/Significance: African American (AA) men are more likely to be diagnosed with, and die from prostate cancer (PCa) than any other race/ethnicity in the United States. Metastatic PCa initially responds to standard-of-care androgen deprivation therapy, however, once resistance develops, treatment options are limited. Inhibiting androgen-independent signaling pathways that promote metastasis—like human epidermal growth factor receptor 2 (HER2) signaling—is promising. HER2 overexpression in PCa tumors correlates with worse prognosis and treatment-resistance, but HER2 overexpression has not been evaluated in AA men. We hypothesize that HER2 overexpression correlates with West African genetic ancestry (WAA) in AA PCa patients and worsens clinical features, treatment response, and survival outcomes.

Methods: RNA sequencing analysis was conducted on 36 AA PCa tissue samples from patients with WAA to determine a correlation between HER2 expression and WAA. Immunohistochemistry analysis of formalin-fixed paraffin-embedded (FFPE) tissue from the primary PCa tumors of AA patients was completed using anti-HER-2/neu antibody to detect HER2 protein. A score of 2+ or 3+ indicated a “positive” result. Quantitative polymerase chain reaction (qPCR) analysis of 2 AA and 3 European American (EA) cell lines measured differential HER2 mRNA expression, relative to control beta actin (ACTB). Lastly, cell viability was evaluated in PCa cells treated with anti-HER2 agent Trastuzumab for 72 hours using the CellTiter-Glo® assay. The treated group was normalized to untreated vehicle in each cell line.

Results: HER2 expression correlates with increasing West African ancestry (WAA) in prostate tissue (Corr=0.1593). Thirty percent (3/10) of the FFPE samples from primary prostate tumors from AA patients showed membrane HER2 staining scored as 2+. Basal HER2 expression was differentially expressed across five PCa cell lines with the greatest expression in the AA PCa cell line, RC-77T/E. Also, Trastuzumab significantly decreased cell viability in AA cell lines (RC-77T/E (p-value of 0.00081) and MDA-PCa-2b (p-value of 0.0036) but not in European American (EA) cell lines (PC3 (ns), DU145 (ns), 22Rv1 (ns).

Conclusions and Implications: The findings from this study will provide the foundation to pursue future HER2-focused clinical trials that have a major impact in reducing the higher risk of death in AA men by using a precision medicine approach.

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P-08 Title: ***Multivariate transcriptome analysis identifies networks and key drivers of metastatic castration-resistant prostate cancer***

Authors: Lawrence P. McKinney, MPH[1]; Rajesh Singh, PhD [1]; I. King Jordan, PhD[2], Sooryanarayana Varambally, PhD[3], Martin Sanda, MD[4], Eric Dammer[5] and James W. Lillard, PhD, MBA[1]

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[2] School of Biological Sciences, Georgia Institute of Technology
[3] Department of Pathology, University of Alabama at Birmingham
[4] Department of Urology, Emory University School of Medicine
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Mentor: James W. Lillard, PhD, Morehouse School of Medicine

Background/Significance: Prostate cancer (PCa) is the second most cancer-related cause of death in men. Most PCa are characterized as slow-growing or indolent, though some patients are at a high risk of for recurrent and eventually metastatic disease. The most lethal form of PCa is metastatic castration-resistant prostate cancer (mCRPC) that has progressed to the bone. The molecular mechanism underlying PCa progression to bone has not been fully elucidated. Here, we identify genes and structural variants associated with mCRPC.

Methods: Herein, we processed RNA sequencing data from patients with mCRPC (n=60) and identified 14 gene clusters (modules) highly correlated with mCRPC bone metastasis. We used a novel combination of weighted gene co-expression network analysis (WGCNA) and upstream regulator and gene ontology analyses of clinically annotated transcriptomes to identify genes.

Results: The cyan module (M14) had the strongest positive correlation (0.81, p=4E-15) with mCRPC bone metastasis. It was associated with two significant biological pathways through KEGG enrichment analysis (para-thyroid hormone synthesis, secretion, and action and protein digestion and absorption). In particular, we identified 10 hub genes (ALPL, PHEX, RUNX2, ENPP1, PHOSPHO1, PTH1R, COL11A1, COL24A1, COL22A1, and COL13A1) using cytoHubba of Cytoscape. We also found high gene expression for collagen formation, degradation, absorption, cell-signaling peptides, and bone regulation processes through Gene Ontology (GO) enrichment analysis.

Conclusions and Implications: Metastatic castration-resistant prostate cancer (mCRPC) is potentially lethal and often spreads to the bones through a biological mechanism we do not completely understand. A previous study sequenced RNA from patients with mCRPC, and in this study, we identified 10 genes associated with mCRPC that spread to the bones, and 2 of those genes are novel discoveries that could serve as new biomarkers for diagnosis or molecular targets for treatment. However, future studies are required to validate these genes' molecular role in mCRPC progression.

Acknowledgment of Funding: MSM/Tuskegee U/UAB Comp. Cancer Center Partnership NCI - U54CA118638

P-09 Title: ***Investigating Differences in Non-Small Cell Lung Cancer Treatments amongst White and Black Patients in the United States***

Authors: **Kirthi Rao**[1] and Dale Hardy, PhD, RD, LD, CDE, CHES[2]

[1] Morehouse School of Medicine, Master of Medical Sciences Program
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Mentor: Dale Hardy, PhD, RD, LD, CDE, CHES, Morehouse School of Medicine

Background/Significance: Lung cancer is the deadliest cancer for men and women in the United States. Prior studies have shown disparities in treatment of lung cancer between whites and blacks in the United States. However, there is a gap in knowledge in time to receiving chemotherapy treatment while considering covariates such as sex, tumor stage, grade, or histology. Our study investigated months from non-small cell lung cancer (NSCLC) diagnosis to chemotherapy treatment utilizing a different database to not only further elucidate the nature of racial disparities in lung cancer treatment, but also sex differences. It is imperative to conduct deeper research to better address deficiencies in the treatment of racial minorities and thereby promote health equity. Our hypothesis posits that there are differences in chemotherapy treatments between black and white patients.

Methods: Data was analyzed for 102,116 patients (50.4%: white males, 4.5%: black males, 41.4%: white females, 3.7%: black females). Statistical Package for Social Sciences (SPSS) software was used for statistical analyses. ANCOVA was performed with months from NSCLC diagnosis to chemotherapy treatment as the outcome, adjusted for covariates race, sex, and tumor stage (not including in situ). Further analysis will include more participant data and will adjust for other covariates including age, tumor grade, histology, and nodes.

Results: There was a significant difference between white males and black males in the time between diagnosis to receiving chemotherapy treatments. From the time of diagnosis to treatment, black males take on average 6.59 days longer than white males (<.001). There was a significant difference between white females and black females; from the time of diagnosis to treatment, black females take on average 7.38 days longer than white females (<.001). Preliminary results showed no differences between black males and females or between white males and females, indicating that race is a significant factor in time to receive chemotherapy treatment after diagnosis.

Conclusions and Implications: Our findings showed that black patients received chemotherapy treatment later than white patients. These findings were consistent with prior similar studies that studied surgery, external beam radiation, and stereotactic radiation. Our results highlight the nuances in health inequities between black and white patients.

P-10 Title: ***Mitochondrial-ATPase and COXIV genes are differentially expressed among colorectal adenocarcinoma patients' tissues***

Authors: **Mikayla Woodard**[1] and Felix O Aikhionbare, MS, PhD[2]

[1]Morehouse School of Medicine, MD program
[2]Morehouse School of Medicine, Department of Medicine/Cancer Health Equity Institute

Mentor: Felix O Aikhionbare, MS, PhD, Morehouse School of Medicine

Background/Significance: The clinical frequency of colorectal cancer (CRC) occurrence increases in adenoma differentiation to carcinoma and the histopathological of adenomas are tubular (T) (5%), tubulovillous (TV) (22%) and villous (V) (40%). Patients with adenomatous polyps have a threefold higher risk of colon cancer over the general population. The risk factor increases to sixfold if the polyps are multiple and with lower survival among African American population. Therefore, a study to identify mutations in early adenomas sequence among patients with an elevated risk of developing CRC is patho-pharmaceuticals important. Variants in mitochondrial (mt) protein expressions have been correlated with several clinico-pathological features of cancers as most of the energy for tumor transformation are of mitochondrial origin due to the important roles of COX and ATP in ROS productions. We hypothesize that there will be differential variants of ATPase and COX gene during the progressive stages of adenoma to carcinoma among patient's tissues.

Methods: Mitochondrial encoded subunits of complexes IV and V of the electron transport chain which include ATPase and COX subunits were analyzed with direct sequencing, high resolution restriction digestion, RT-qPCR and western blot techniques were used to assess differences in colorectal tumors. Tissue samples used include early adenomas classified as T, TV, V and cancer tissues (CA), and their normal surrounding tissues (NST).

Results: Most variants of complex IV were found in COX subunit III (9207-9990). Of these variants del9414C was found in 60% TA and 20% CA tissue samples. ATPase6 variant G9055A was abundantly high in TA and V samples and confirmed using high resolution restriction digestion. Expression levels of ATPase6 progressively increased from early adenomas to late-stage adenomas and COX subunits also varied within the adenoma -carcinoma sequence. COX subunit-4 isoform 1 protein expression decreased by 5-fold in cancer samples when compared to normal tissue and by 3-fold when compared to TA, TV.

Conclusions and Implications: The findings suggest a significant role of ATPase and COX IV gene variants as biomarkers during CRC tumor progressive stages, given that mitochondrial genes are involved in ROS productions and OXPHOS regulations. This study warrants further research to fully elucidate the pivotal role(s) of mitochondrial in designing of drugs for early stages of CRC.

Acknowledgment of Funding: NIH GM122669 and GM099663

P-11 Title*: Impact of Therapeutics on Cardiovascular Health and Breast Cancer Outcomes*

Authors: **Kaala S Berry**, Hina Mir, PhD and Shailesh Singh, PhD

Department of Microbiology, Biochemistry, and
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Morehouse School of Medicine, Atlanta GA

Mentor: Shailesh Singh, PhD
Morehouse School of Medicine

Background/Significance: Currently offered therapeutics to treat breast cancer have significantly declined the mortality rates over the past years and have increased life expectancy. However, these improvements in mortality rate and life expectancy come with the elevated risk for cardiovascular disease (CVD) due to the cardiotoxic effects of treatments. Breast cancer and cardiovascular disease have various overlapping risk factors, and treatment offered for breast cancer further impacts cardiovascular health (e.g., accelerated CVD, ventricular dysfunction). Patients with pre-existing CVD may alter their cancer treatment decisions, avoiding exacerbating the pre-existing heart condition. Furthermore, breast cancer treatment associated with cardiac dysfunctions often excludes patients from receiving aggressive treatment to treat recurrent disease. Hence, this work aims to ascertain the treatment-associated factors contributing to CVD and possible cardio protective options to reduce associated CVD.

Methods: We have used a PubMed search to ascertain the factors contributing to breast cancer treatment associated with CVD.

Results: Chemotherapy, radiation therapy, hormone ablation therapy, and immunotherapy are offered to treat breast cancer as a single agent or in combination as an adjuvant or neoadjuvant to treat breast cancer. However, these agents often negatively impact the cardiovascular and immune systems. Therapeutic regimens impact the immune system directly or indirectly by affecting heart function. Immune suppression often promotes disease faster and contributes to recurrence. Studies have shown that after receiving treatment, breast cancer survivors who developed CVD or events (i.e., heart attack, stroke, heart failure, coronary artery disease, or arrhythmia) had a 59% higher risk of breast cancer recurrence and 60% higher risk of dying from breast cancer. Studies on mice where heart attack was induced show accelerated tumor growth and lung metastasis compared to sham further suggest the impact of cardiac health on breast cancer progression and outcomes.

Conclusions and Implications: Cardiovascular health is key to disease progression, therapeutic outcome, and overall survival of breast cancer patients. Hence, cardioprotective strategies are needed while offering conventional therapies to treat breast cancer. Additionally, preexisting conditions such as diabetes, blood pressure, cholesterol, and lifestyle should be considered while developing treatment.

P-12 Title: ***Loss-of-STING function by nitro-fatty acids reduces endometriotic lesion formation in a syngeneic model of endometriosis***

Authors: **Mareena Pitts**[1], Wei Xu[1], Anne Louis Hansen[2], Christian K. Holm[2], Bruce A. Freeman[3], Minerva Garcia-Barrio[4], Francisco J. Schopfer[3], Winston E. Thompson[1,5], and Luis Villacorta[1]

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Mentor: Luis Villacorta, PhD, Morehouse School of Medicine

Background/Significance: Endometriosis is a painful, chronic inflammatory disease that impacts fertility and the overall well-being in 10% of reproductive-aged women. Current therapies include hormonal treatment to induce a hypoestrogenic state, causing complicating side effects. STING (Stimulator of Interferon Genes) plays an essential role in innate immunity but also exacerbates inflammation in chronic inflammatory diseases. Recent evidence suggests that STING is aberrantly upregulated in endometriosis patients. Nitro-fatty acids (NO_2-FAs) are bioactive lipids that exert anti-inflammatory and protective roles in preclinical models of inflammation and fibrosis, in part via inhibition of STING. We hypothesize that NO_2-FAs protect against STING-dependent endometriosis lesion formation and tested its therapeutic potential using a syngeneic transplant model of endometriosis.

Methods: Uterine fragments from Pregnant Mare Serum Gonadotrophin (PMSG)-treated enhanced green fluorescent protein (EGFP) mice were transplanted into wild-type (WT) female recipients randomized to receive NO_2-FA or vehicle. Endometriosis was allowed to progress for 28 days.

Results: In vivo imaging of EGFP donor-derived endometrial lesions demonstrates that NO_2-FA administration significantly reduces peritoneal endometriotic lesion formation in female recipients. Furthermore, loss-of-STING function, using STING KO female mice as recipients, demonstrates that STING plays a functional role in endometriosis development. Finally, our studies demonstrate that NO_2-FAs inhibit cyclic GMP-AMP Synthase (cGAS)-induced type I interferon signaling in bone-marrow-derived macrophages (BMDM) and in the peritoneum of WT female mice but not STING KO mice.

Conclusions and Implications: In summary, we show that STING is a novel, functional contributor to the pathogenesis of endometriosis and that NO_2-FA administration serves as a novel non-hormonal therapy against endometriosis.

Acknowledgement of Funding: This study is funded by the American Physiological Society William Townshend Predoctoral Fellowship (MP) and NIH R01-HL123333 (LV).

P-13 Title: ***Inverse Regulation of SGK1 and the Mitochondrial Calcium Uniporter Complex in Aortic Smooth Muscle Cells from Mice and Humans with Obesity and Type 2 Diabetes***

Authors: **Jade Avery**[1], Wei Zhong[2], and Sharon C. Francis, Ph.D.[1,2]
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Mentor: Sharon C. Francis, Ph.D., Morehouse School of Medicine

Background/Significance: The prevalence of obesity has become an epidemic in the US due to the increased consumption of foods high in fat. Individuals with a body mass index (BMI) of ≥30 have an increased risk of developing type 2 diabetes (T2D) which exacerbates vascular disease and cardiovascular adverse events such as strokes and heart attacks. However, the molecular mechanisms that underlie obesity- and T2D-induced vascular disease are not fully understood. We have shown that serum and glucocorticoid-inducible kinase 1 (SGK1) is up-regulated in vascular smooth muscle cells (VSMC) of mice fed a high fat diet to induce obesity and hyperglycemia, a main symptom of T2D. Moreover, knocking out SGK1 in VSMC protects against vascular damage by abolishing the mitochondrial dysfunction that typically accompanies obesity and T2D. Vascular protection correlated with elevated levels of the mitochondrial calcium uniporter complex suggesting that our newly discovered SGK1/MCU pathway plays a role in obesity-associated T2D vascular disease development in humans. MCU is a multimeric complex that regulates Ca^{2+} influx into the mitochondrial matrix and is tightly controlled by different regulatory subunits with varying functions but its regulation in VSMC exposed to obesity and T2D is unknown. Thus, we tested the hypothesis that obesity and T2D exacerbates VSMC dysfunction via a mechanism that involves SGK1-dependent negative regulation of the MCU complex.

Methods: We examined regulation of SGK1 and the MCU complex in aortic VSMC from lean and human subjects with obesity and lean and high fat diet-induced obese wildtype ($VSMC^{WT}$) and VSMC-SGK1 knockout ($VSMC^{KO}$) mice. To this end, western blot analysis was performed on VSMC total protein extracts from individuals with BMI = 23.5 ± 0.25 (Lean) and BMI = 37.6 ± 7.2 (Obese) with or without T2D and mice fed a 10 kcal% low fat or 45 kcal% high fat diet for eight weeks. Data was analyzed by 2-Way Analysis of Variance to determine significance.

Results: $VSMC^{WT}$ from obese mice showed reduced levels of MCU and its gatekeeping regulatory subunits, MICU1 and MICU2 compared to lean $VSMC^{WT}$ mice. Knocking out SGK1 in VSMC abolished obesity-related downregulation of MCU and its gatekeeper subunits. As observed in obese mice, SGK1 protein was enriched in VSMC from individuals with obesity. Interestingly, T2D potentiated up-regulation of SGK1 independent of BMI classification. In addition, VSMC from obese persons showed decreased levels of the MICU2 gatekeeping subunit and this decrease was exaggerated in VSMC from obese individuals with T2D.

Conclusions and Implications: These findings show that obesity enhances SGK1 abundance in VSMC which may promote negative regulation of the MCU complex and that T2D may exacerbate these effects in the vasculature of humans and rodents. Furthermore, the SGK1/MCU novel pathway may play a permissive role in obesity-related VSMC dysfunction and consequent vascular disease.

Acknowledgment of Funding: NIH RCMI grant 5G12MD007602; NSF Excellence in Research grant: 2100832

P-14 Title: ***The Prevalence of Cardiovascular Disease in the West End Population of Atlanta: A Cohort Study***

Authors: Janae' Bryant, Khayrassa Cantrell, Priscylla Carvalho, Alexis Gamble, **Ashley Johnson,** and Rachel Magistre–Thomas

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Mentor: Christopher Ervin, MD
Morehouse School of Medicine

Background/Significance: The West End area of Atlanta has experienced a rise in cases of cardiovascular disease (CVD) over the last few years. Research shows there have been no effective efforts to implement changes for the community that correct the risk for CVD. The purpose of this study is to assess whether a systematic approach that supplements physician-centered efforts will reduce risk of CVD when compared to the standard of care. We hypothesize that there will be a negative correlation between this supplementation and the risk of CVD.

Methods: Analysis will be conducted to use data and bring awareness of cardiovascular disease in adults with the median household income of approximately $19,000 living in the West End of Atlanta by hosting educational health fairs in the community. We will also assess the needs of the community by questionnaires and surveys. We will identify the outcomes of standard of care and compare against the prevalence of CVD. We plan to utilize continuous variables and techniques to assess risk of interest.

Results: When provided with a systematic physician lead nutrition and cardiovascular disease education, there will be a decrease in cardiovascular incidence rates for the West End population.

Conclusions and Implications: The findings of this cohort study suggest the necessity for continued surveillance of a systemic approach to minimizing the risk of CVD. Clinically cardiovascular interventions in the West End population of Atlanta should target at risk individuals, particularly among the adult female and male. Further research is warranted to explain the prevalence of CVD risk.

P-15 Title: ***Assessment of P311's influence on key genetic signatures within brown adipose tissue***

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Mentor: Kameswara Badri, Ph.D.
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Background/Significance: With the consistent increase in obesity levels the world has come to an infection point leading to rises in obesity associated disease states such as cardiovascular disease and diabetes. This development has created a need for a deeper understanding of the molecular underpinnings which drive obesity. P311 has been shown to be a factor which influences the characteristics of adipose tissue composition through the increase in adipocytes which display thermogenic gene markers such as brown and beige adipocytes; thereby leading to a more metabolically "healthier" adipose tissue phenotype. The objective of this project is to determine if P311 influences the characteristics of brown adipose tissue composition.

Methods: Using novel single cell RNA technology, we examined P311's impact on brown adipose tissue collected from wild type and P311 KO mice. Cells were differentiated based on their individual genetic makeup which was used to characterize distinct cell populations. Analysis using the Seurat package in R looked at a variety of markers for cell types found in brown adipose tissue such as white and beige adipocytes, stromal vascular fraction which includes, immune cells, endothelial cells, and precursor cells.

Results: Our findings showed a significant decrease ($p<0.01$) in key thermogenic genes such as UCP1 and other brown adipocyte markers in P311 KO brown adipose tissue compared to wildtype. Knockdown of P311 led to a significant increase in white adipocyte markers ($p<0.01$) in P311 KO brown adipose tissue.

Conclusions and Implications: Our findings suggest that absence of P311 leads to a decrease in thermogenetic adipocytes which could result in metabolically unhealthier adipose tissue.

P-16 Title: *Exploring Storing Conditions for Adipocyte Derived Exosomes (AdExos)*

Authors: **Ruta Panchal**[1], Kunheng Cai, MS[2], and Anthony Ferrante, MD, PhD[2]

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Mentors: Kungheng Cai, MS; Anthony Ferrante, MD, PhD
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Background: AdExos are lipid-filled exosome-sized particles released by adipocytes which have an important role in adipose tissue as they are a source of lipid for local macrophages. This was important to note as AdExos have an important function in immune modulation as well as having a role in an alternative pathway of local lipid release. The accumulation of released AdExos activates a lysosomal catabolism inhibiting TAG hydrolysis causing potential systematic metabolic complications such as insulin resistance and hepatic steatosis. The best temperature to store these particles has not been explored. This project focused on the best temperature over a period of time that AdExos were able to be stored.

Methods: A size exclusion chromatography column and gel content were first made. A washed gel mixture was then put through the column. After this was prepared, AdExos were extracted from perigonadal tissue from seven male black mice and stored in +4 °C and -80 °C for 16 days. At 6 time points (day 0, day 1, day 2, day 5, day 8, and day 16) the AdExos were measured using the Viewsizer 3000. An unpaired t-test was performed to see the significance between the two temperature points.

Results: At six time points the average of the 3 samples were taken. The samples stored in -80 C were steady through the 16 days, while the +4 C samples varied through the days. The results for 6 data points testing with two different conditions gave a p-value of 0.1675, showing no significance between the two conditions

Conclusions and Implications: The storage of AdExos helps in knowing how long the particles can survive. Future research should consider longer periods of time and more temperatures that the AdExos can be stored in. While the results were not statistically significant, particles stayed more stable at the lower temperatures than the higher temperatures. Future researchers should explore lower temperatures for better stabilization.

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P-17 Title: ***Effects of Brain-Derived Neurotrophic Factor on Heart Rate During Exercise***

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Mentor: Joe Nocera, MD
Atlanta Veterans Affairs Medical Center

Background/Significance: With the American aging population increasingly growing, there has been increased interest in establishing beneficial interventions that will enhance cardiovascular health and decrease cognitive decline. Recent findings suggest that brain-derived neurotrophic factor (BDNF) plays a role in heart rate regulation. Specifically, BDNF is conjectured as serving a beneficial role during energetic events such as vigorous exercise. The objective of this study is to assess changes in serum BDNF levels during a standardized exercise intervention.

Methods: Six participants voluntarily participated in this study and were randomly assigned into two exercise groups: aerobic and circuit. Over the course of twelve weeks, participants exercised three times a week at the Atlanta Veterans Affair Medical Center (VAMC). Exercise time progressed from an initial 20 minutes per session to a maximum of 60 minutes by increasing 5 minutes each week. BDNF levels were measured before each exercise session, at ten-minute intervals during the exercise sessions, and ten minutes after each exercise session. Microsoft Excel and SPSS software version 21 were used for data management and analyses.

Results: Six participants have participated in this intervention. Preliminary findings indicate that there is no established correlation between BDNF and heart rate. However, our results suggest that resting BDNF levels have increased after twelve weeks of the exercise program in some participants.

Conclusions and Implications: Our findings, thus far, suggest that there is no established correlation between BDNF levels and heart rate during exercise. After the exercise program concluded, resting BDNF levels were increased in several participants. Because the mechanisms that explain these changes are not easily understood, it would be beneficial for future research to include other biological and physiological markers. Moreover, studies with larger sample sizes could be more illuminating in identifying the relevance between changes in heart rate and its relationship to exercise and BDNF.

P-18 Title: ***Heme-induced expressions of IL-6R, TLR4 and NFκB in human brain endothelial cells (HBEC-5i) and macrophages (THP-1) are modulated by miR-451a and let-7i-5p-loaded extracellular vesicles (EVs)***

Authors: Alaijah Bashi[1]; Justin Thomas M.H.S.[1]; Keri Oxendine Harp Ph.D.[1]; Joshua L. Hood M.D., Ph.D.[2]; Jonathan K. Stiles Ph.D.[3]; and Adel Driss Ph.D.[1]

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Mentor: Adel Driss, PhD, MSc, Morehouse School of Medicine

Background/Significance: *Plasmodium falciparum,* a species of parasite involved in malaria pathogenesis, damages erythrocytes, releasing cytotoxic heme into the circulation. Increased circulatory heme correlates with proinflammatory cytokines that exacerbate malaria morbidity and mortality. Among the mechanisms mediating heme-induced inflammation are microRNAs (miRNAs) including miR-451a targeting the interleukin-6 receptor (IL-6R) and let-7i-5p targeting Toll-Like Receptor 4 (TLR4). Both targets are associated with nuclear factor kappa B (NFκB) signaling via the JAK/STAT3 and PI3K/AKT pathways. Circulating miRNAs can be transported in extracellular vesicles (EVs) such as exosomes. We hypothesized that EV-loaded-miRNAs could modulate heme-induced inflammation in vascular endothelial cells (HBEC-5i) and macrophages (THP-1).

Methods: We incubated each cell line with liposomes (artificial EV)-loaded miR-451a or let-7i-5p mimic oligonucleotides simultaneously with and without 30 μM heme or with DMSO controls. The expression of IL-6R, TLR4, p65/NFκB and GAPDH (internal control) genes was assessed using RT-qPCR after 24 hours. ANOVA test was used to determine significance ($p < 0.05$).

Results: Results showed that heme induced inflammation in HBEC-5i and THP-1 cells. MiR-451a and let-7i-5p-loaded liposome treatment significantly decreased the expression of IL-6R, TLR4, and NFκB genes. This suggests a reduction in M1/pro-inflammatory phenotypes.

Conclusions and Implications: Both vascular endothelial cells and macrophages can be attenuated by miR-451a and let-7i-5p-loaded liposomes in response to excess heme-induced cellular stress and pro-inflammatory immunity. MiRNA-loaded EVs could be used as a therapeutic tool to decrease cellular stress and toxicity caused by heme. In hemolytic diseases like malaria and sickle cell an approach like this can reduce the vasoocclusion crisis and inflammation induced by heme.

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P-19 Title: ***The Impact of Human Cytomegalovirus Infection on Innate Immune Signaling at the Placental Interface***

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Mentor: Erica L. Johnson, Ph.D.
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Background/Significance: The placenta is a target for many maternal viruses, including human cytomegalovirus (HCMV). HCMV is the most common viral infection transmitted from mother-to-child and establishes lifelong latency in immunocompetent hosts; however viral reactivation often occurs with impaired host immunity. Recent studies have demonstrated that HCMV can reactivate during pregnancy, with 30-50% of HCMV-seropositive women showing evidence of viral shedding during pregnancy. HCMV replication during pregnancy has been shown to induce immune activation and upregulate inflammation by direct upregulation of pro-inflammatory cytokines and mediators in host cells. In addition, placental HCMV infection triggers an inflammatory response that alters the trophoblast, inducing placental dysfunction. Very little is known about the molecular triggers and mechanisms underlying activation of immune pathways associated with HCMV infection. In this study we will develop a mechanistic understanding of how placental cells respond to HCMV.

Methods: We will culture trophoblast cell lines (JEG3) and isolate primary maternal decidua cells from placental tissue. Cells infected with HCMV and/or exposed to specific agonist for TLR-9 (ODN-2216) and STING (2'3'-cGAMP). ELISA, Western Blot, and qPCR will determine HCMV infection and the expression of type-1 IFNs, inflammatory cytokines, cGAS-STING, TLR-9, MyD88, IRF-3, IRF-7, and NFκB. Data will be analyzed using T-test.

Results: The data collected throughout this experiment will determine how HCMV is detected at the placenta by the intracellular DNA sensors, TLR-9 and cGAS-STING. We will also determine how placental cells respond to infection directly or indirectly via the TLR-9 and cGAS-STING innate immune pathways. Here we demonstrate that the cGAS-STING pathway is critical for HCMV detection and inhibition of HCMV infection at the placenta.

Conclusions and Implications: More than 50% of women are affected and endure complications during pregnancy because of HCMV infection. It is important to understand how cGAS-STING pathways and toll-like receptors (TLRs) are important in the response to HCMV infection during pregnancy.

P-20 Title: ***The Use of Momo30 in Developing a Novel, Affordable Drug Against SARS-CoV-2***

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Mentor: Michael D. Powell, Ph.D., MA
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Background/Significance: The COVID-19 pandemic has caused millions of deaths throughout the world especially in damaging areas such as Africa that lack the necessary healthcare infrastructure to provide its citizens with vaccines. Due to this, alternative treatment methods must be created so that millions of more African people do not die from SARS-CoV-2. The objective of this study is to determine if MoMo30, a protein derived from Momordica balsamina (Senegalese Bitter Melon) that has demonstrated antiviral potential in preliminary studies conducted by our lab, can be used as a potential drug for COVID-19. As Momordica balsamina is native to the tropical regions of Africa the creation of a drug based on Momo30 would be cheaper than drugs manufactured in other countries. This study is innovative in that it uses computer models and simulations to model the effect this purposed drug has on the concentration of SARS-CoV-2 in a human body.

Methods: In preliminary experiments, our lab has isolated MoMo30 from extracts of M. balsimina and tested its antiviral activity in vitro against HIV, Ebola, Influenza, HSV, and HCoV-229E. Through doing literature reviews upon how Momo30 binds to SARS-CoV-2 and inhibits it, we have seen that Momo30 inhibits SARS-CoV-2 the same way that it does for HIV, Ebola, Influenza, HSV, and HCoV-229E. We then use computer models and simulations to test the purposed drug in different concentrations against COVID-19 in a human body and track the decrease in concentration over time.

Results: We have found that MoMo30 through our computer models and simulations that Momo30 is able to effectively decrease significant concentrations of SARS-CoV-2 when used in particular concentrations.

Conclusions and Implications: So far, these findings suggest that MoMo30 has the potential as a drug against SARS-CoV-2 infection. Additional data collected via testing in vivo would lead to the next step of testing MoMo30s drug potential in animal models and unlocking Momo30's ability to affordably treat the citizens of Africa from COVID-19.

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P-21 Title: ***Immunization with rVCG-MECA vaccine assessed for cellular and humoral immune responses***

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Mentors: Francis O. Eko, PhD, and Yusuf Omosun, PhD, Morehouse School of Medicine

Background/Significance: *C. trachomatis* is the most reported sexually transmitted infection in the United States. It is a gram-negative obligate intracellular bacterium which affects, the genital tract, eyes, and throat. In 2019, a total of 1.8M cases of *Chlamydia trachomatis* were reported to CDC. However, most cases are not reported because of the asymptomatic nature of the bacterial infection. If untreated, this can result in severe complications such as pelvic inflammatory disease (PID), ectopic pregnancy, and infertility. We have generated a self-adjuvating rVCG-based subunit vaccine candidate containing a multiepitope *C. trachomatis* antigen rVCG-MECA composed of Chlamydia immunogenic proteins composed of T and B cell epitopes of polymorphic membrane protein (PmpD), porin B protein (PorB) and outer membrane complex protein B (OmcB). Previous studies have established VCG as an effective delivery system capable of delivering antigens to the immune system to generate substantial immune responses and protection. We hypothesize that rVCG-MECA will protect against chlamydia by eliciting strong and effective cell-mediated and humoral responses capable of providing long-term cross-protective immunity.

Methods: Female Mice C57BL/6J mice (N=8, per treatment) were immunized intramuscularly (IM) and intranasally (IN) and boosted twice, two weeks apart, with rVCG-MECA, once with live Chlamydia (*C. trachomatis* serovar D elementary bodies) and PBS. Specific mucosal and systemic immune responses were characterized. Vaccine efficacy was determined from chlamydia shedding following the transcervical challenge. Additionally, Chlamydia-specific cytokine (IFN-γ and IL-4) production by splenic and ILN T cells was assessed after 16-weeks.

Results: IM and IN immunization elicited IFN-gamma producing CD4 T cells, with IM eliciting higher IFN-gamma response compared to IN. IM also elicited higher humoral response than IN in mucosal and systemic tissues. These results highlight the potential of rVCG-MECA immunization as potential vaccine for inducing protective immunity in female genital tract *Chlamydia* infection.

Conclusions and Implications: This study suggests that IM and IN immunization with rVCG-MECA induces immune effectors such as IFN-gamma and IgG2c that mediate chlamydial clearance in the genetical tract.

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P-22 Title: ***Immunization with the chlamydial multi-epitope antigen MECA elicits Chlamydia-specific systemic and mucosal immune responses***

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Mentor: Francis O. Eko, PhD
Morehouse School of Medicine

Background/Significance: Chlamydia is a significant public health concern being the most common bacterial sexually transmitted disease (STD) worldwide. Most cases of Chlamydia infection are asymptomatic, however, if left untreated, infection could lead to the development of pelvic inflammatory disease (PID), ectopic pregnancy, and tubal infertility. An efficacious vaccine is necessary to control the incidence of Chlamydia infections. In this study, we investigated the immunogenicity of a novel, multi-epitope chlamydial vaccine, MECA, composed of T- and B-cell epitopes derived from the *Chlamydia trachomatis* polymorphic membrane protein D (PmpD), porin B (PorB), and outer membrane complex B (OmcB) proteins. We hypothesize that immunization with the vaccine candidate will induce robust systemic and mucosal immune responses.

Methods: Female C57BL/6J mice were immunized intramuscularly (IM) with MECA or PBS and boosted twice at two-week intervals or received a single transcervical inoculation with live *C. trachomatis* serovar D elementary bodies (EBs). Twelve weeks after the last immunization, serum and vaginal wash samples were obtained to quantify *Chlamydia*-specific antibodies (IgG2c and IgA). Additionally, *Chlamydia*-specific cytokine (IFN-γ and IL-4) production by splenic and iliac lymph node T cells was assessed.

Results: The results showed that immunization with MECA elicited *Chlamydia*-specific antibodies in both mucosal (vaginal wash) and systemic (serum) compartments. Also, the induction of IFN-γ relative to IL-4 indicates a Th-1 type cellular response.

Conclusions and Implications: Previous models have identified protective immunity against chlamydial infection, including humoral and cell-mediated responses. The study demonstrates the potential of MECA to provide protective immunity through the induction of *Chlamydia*-specific antibodies and cytokines. These results therefore encourage further research into its efficacy.

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P-23 Title: ***Epidemiology of meningitis and antibiotic resistance patterns in neonatal intensive care units***

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Mentor: Lance Okeke, MD
Duke University School of Medicine

Background: Meningitis is more common in infants than any other age group and is associated with significant mortality. The epidemiology of meningitis is changing due to use of intrapartum antibiotics and changing bacterial resistance patterns. The objective of this study is to evaluate the epidemiology and outcomes of bacterial meningitis, and antibiotic resistance patterns in neonatal intensive care units (NICUs).

Methods: We identified all infants < 90 days of age, with a positive cerebrospinal fluid (CSF) culture for a bacterial pathogen, between 2013-2017 at Pediatrix Medical Group NICUs. We excluded infants with a viral CSF isolate. We divided infants into 3 groups: early onset (0-6 days of age), late onset (7-29 days of age) and extremely late onset (30- 90 days of age). We assessed the epidemiology and hospital outcomes of meningitis, distribution of causative organisms, and their resistance patterns. Outcomes of interest were a failed hearing screen, and death before discharge.

Results: 325/452,258 infants from 332 NICUs were diagnosed with meningitis; nearly half of the infants were in the late onset group (n=151; Table 1). The median (25th-75th percentile) gestational age of the cohort was 35 (26 – 39) weeks. 32% (n=97) of infants with meningitis had a positive blood culture within 7 days of positive CSF culture. Group B Streptococcus (GBS) (n=38) and Escherichia coli (E. coli) (n=38) were the most common infecting organisms overall. For the 38 infants with E. coli, 18 (47%) were ampicillin-resistant (Table 2). Infants in the very late onset group had the highest prevalence of adverse outcomes (Table 3).

Conclusions and Implications: In this cohort of infants diagnosed with meningitis in the first 90 days of age, GBS and E. coli were the most prevalent causative organisms, with substantial proportion of ampicillin resistant E. coli. Infants with very late onset meningitis were more likely to have an adverse outcome prior to discharge.

P-24 Title: ***Comparison of Patient Demographics and Risks of Readmissions Following Open Reduction and Internal Fixation for Bimalleolar Ankle Fractures***

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Mentor: Dr. Afshin E. Razi, Maimonides Medical Center

Background/Significance: Open reduction and internal fixation (ORIF) is the standard treatment for numerous types of fractures however readmissions rates can be more prevalent within certain populations. Therefore, the aims of this study were to 1) compare patient demographics and 2) identify risk factors regarding 90-day readmissions following ORIF for bimalleolar ankle fractures.

Methods: The study conducted was a retrospective analysis that used hospital information from January 1st, 2010, to October 31st, 2020. Cohorts were identified in the dataset by using syntax-based language with respect to International Classification of Disease, Ninth Revision (ICD-9), ICD-10, Current Procedural Terminology (CPT) and various other diagnostic and procedural codes. Cohorts consisted of patients who underwent ORIF for bimalleolar ankle fractures. The three research domains utilized in this investigation were baseline demographic profiles of patients who were and were not readmitted within 90-days following ORIF of bimalleolar ankle fractures, assessing annual trends of readmissions, and identifying patient-related risk factors associated with hospital readmissions. The demographics of age, sex, and prevalence of comorbid conditions were assessed using Pearson's Chi-Square Analyses or Fischer's Exact Test. Linear regression analysis were used to determine the annual trends of readmissions and an alpha value less than 0.001 was considered to be statistically significant. A multivariate regression model was constructed to calculate the odds-ratios (OR) of readmissions following ORIF for these ankle fractures.

Results: The two patient demographics that yielded the highest readmission rates were the 70–74-year-old age group and women. Women were readmitted 68.47% of the time which is double the rate when compared to men at 31.52%. The 70–74-year-old population were readmitted 17.35% of the time. There were numerous comorbidities that were significantly prevalent in those who were readmitted. According to the Odds Ratio (OR) and p-values ($p<0.0001$), the top three conditions seen in readmitted patients were Coagulopathy (OR 1.46%, p-value<0.0001), Pathologic Weight Loss (OR 1.43%, p-value<0.0001), and Congestive Heart Failure (OR 1.32%, p-value<0.0001).

Conclusions and Implications: This study is important because it outlines comorbidities significantly associated with increased readmission rates following ORIF for bimalleolar ankle fractures.

Acknowledgment of Funding: Maimonides Medical Center, Department of Orthopaedic Surgery, Brooklyn, NY

P-25 Title: *Outcomes of Ballistic Tibial Shaft Fractures*

Authors: **Leslie Roman**[1], MS, Roberto Hernandez-Irizarry, MD[2], and Jesse Seilern, MD[2]

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Mentor: Roberto Hernandez-Irizarry, MD
Emory University School of Medicine, Grady Memorial Hospital

Background/Significance: We are comparing blunt tibial shaft fractures to ballistic tibial shaft fractures. The tibia is the second most common fractured long bone caused by civilian firearm. This mechanism of injury is on the rise and learning more about these outcomes can better help treat this patient population. Grady treats over 1,500 cases of firearm related injury each year and we are looking at a 5-year span of fracture care related to blunt and ballistic tibial shaft fractures.

Methods: We conducted a retrospective review from 2016 to 2020 on patients that sustained tibial shaft factures requiring surgical intervention of intramedullary nailing (IMN). Patient charts were reviewed for demographics, fracture characteristics, time from admission to first antibiotic dose and surgical irrigation and debridement (I&D), type of fixation, and type of soft tissue coverage. Clinical outcomes included compartment syndrome, need for soft tissue reconstruction, unplanned returns to the operating room, nonunion, and fracture related infection. Distributions and correlations were analyzed using chi-squared and logistic regression analyses.

Results: We currently only have results for the patients in the ballistic tibial fracture group. Of 113 patients, 10 required initial external fixation, while all others received an IMN with I&D. 16 (14.2%) patients developed a deep infection, 9 (8%) a nonunion, and 11 (9.7%) underwent fasciotomy for compartment syndrome. 19 (16.8%) required soft tissue reconstruction/coverage with split-thickness skin grafting and/or rotational muscle flap. Those requiring soft tissue reconstruction had 8x higher odds of developing a deep infection or nonunion. Patients returned to the operating room 1.06 (range: 0-8) times for tibia-related procedures. Time to first antibiotic dose and to I&D was not significantly longer in patients with deep infection or nonunion. Number, location, and diameter of ballistic wounds showed no significant relationship with outcome measures. Of 113, 100 of these patients identified as Black, had an average age of 29, and 89% were male.

Conclusions and Implications: This is the largest sample of ballistic tibial shaft fractures so far, showcasing the risk profile including compartment syndrome, deep infections, and soft tissue reconstruction requirements. The more we learn and understand the outcomes of ballistic tibial shaft fractures, the closer we can get to providing physicians with a better understanding of how to treat these fractures more effectively to increase positive patient outcome.

Acknowledgement of Funding: Morehouse School of Medicine & $4,000Student Trauma and Resuscitation Research Program (Grady Memorial and Emory University School of Medicine)

P-26 Title: ***Impact of Rib Fracture Repair on Patient Outcomes in Severe Thoracic Trauma: A Retrospective Chart Review***

Authors: **Janelle Tanghal** [1] and Jonathan Nguyen DO [2]

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Mentor: Jonathan Nguyen DO, Morehouse School of Medicine

Background/Significance: Rib fractures are amongst the most common injuries associated with thoracic trauma; however, only a small percentage of rib fracture patients receive surgical stabilization of rib fractures (SSRF). Although most rib fracture treatments are nonoperative, rib fracture repair may be performed in cases with severe displacement, flail chest, or other persistent problems. In order to determine the benefits of performing SSRF, we look at patient outcomes of chest trauma patients. Since SSRF patients receive more treatment following their injuries compared to those who receive supportive treatment, we hypothesize that patients who received SSRF have better patient outcomes compared to those who did not receive SSRF.

Methods: A retrospective chart review was conducted on trauma patients at Grady Memorial Hospital located in Atlanta, Georgia over an 8-month period due to the high volume of trauma incidences within the Atlanta metropolitan area. Data was gathered from 1030 patient charts, of whom 138 had a Thorax AIS ≥ 4, which is classified as severe thoracic trauma. Variables collected include Injury Severity Score (ISS), max Thorax AIS, ICU length of stay, total length of stay, vent days, and mortality rates. Patient outcomes based on these variables were compared between patients who received SSRF and those who did not. Data analysis was performed using two-sample t-test assuming unequal variances.

Results: Of the 138 patients who had a Thorax AIS ≥ 4, 14 patients received SSRF, and 124 patients did not receive SSRF. Average maximum Thorax AIS and ISS between the two groups were not statistically significant. Average vent days, ICU length of stay, and total length of stay were not statistically significant. Patients who received SSRF were shown to have a lower mortality rate than patients who did not receive SSRF.

Conclusions and Implications: Although patient outcomes did not show statistical significance between hospital stay and injury severity, we observed better patient outcomes in terms of lower mortality rates in patients who received SSRF compared to those who did not. Many variables impact patient outcomes beyond body region AIS. Moving forward with this study, we would case match patients based on specific injuries to determine if SSRF is the major determining factor on the lowered mortality rates in these patients.

Acknowledgment of Funding:

- Summer Student Research Experience, Morehouse School of Medicine

P-27 Title: ***Does Wrist Denervation Improve Satisfaction and Functional Scores in the Acute Treatment of Perilunate Injuries?***

Authors: Adam Whitsett, BS[1], Andrew M. Gabig, MD[2], Samuel S. Payne, MD, and Paul Ghareeb, MD[2]

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Mentor: Paul Ghareeb, MD
Emory University School of Medicine

Background/Significance: Perilunate injuries (PLIs) are devastating with no consensus on optimal treatment. The current standard of care is generally considered to be open reduction and internal fixation; however, a significant number of patients develop post-traumatic wrist arthritis requiring additional intervention. Patients with significant pain after PLI may be offered wrist denervation with anterior and posterior interosseous nerve (AIN/PIN) neurectomy. The benefit of AIN/PIN neurectomy in acute treatment of PLIs is not well understood. The purpose of this study is to analyze post-operative patient-reported outcomes in patients who underwent acute AIN/PIN neurectomy in addition to standard treatment of PLI.

Methods: A retrospective review of all perilunate injuries that were treated surgically at two institutions from 2016-2020 was conducted. Demographic, surgical and outcome data was reviewed. Patients were excluded with less than 12-months of follow-up. Patient Reported Wrist Evaluation scores (PRWE), satisfaction scores, and additional surgical interventions were the primary outcome measures.

Results: Thirty-seven patients met inclusion criteria for the study. The average age was 37 (SD 15) years, and 84% of the patients were male. Seven patients (19%) underwent surgical fixation of PLI with an additional AIN/PIN Neurectomy. No significant differences in functional scores were observed in patients treated with AIN/PIN neurectomy compared to those without: PRWE Pain 20.7 (SD 17.1) vs 22.8 (SD 11.9, p=.767), PRWE Function 11.9 (SD 10.1) vs 14.9 (SD 11.6, p=.532), PRWE Total 32.8 (SD 26.7) vs 38.0 (SD 21.7, p=.595). Satisfaction at final follow-up was 7.8 (SD 2.3) vs 6.2 (SD 3.3) for ORIF with AIN/PIN neurectomy compared to ORIF alone, respectively (p=.142). No significant difference was found in the rate of reoperation between groups (p=.107). Patients had an average of 35 months of follow-up (12-64 months).

Conclusions and Implications: Denervation with AIN/PIN neurectomy is typically reserved for the treatment of pain developed after the initial treatment of PLIs. The role of AIN/PIN neurectomy as part of initial operative treatment is not well understood. Our results trended towards better outcomes with the addition of AIN/PIN neurectomy; however, none of the differences met statistical significance in this analysis. Additional research is needed to draw conclusions, but our data indicates that wrist denervation may be considered along with standard operative treatment of PLIs as a means of improving patient outcomes and satisfaction.

Acknowledgement of Funding:

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P-28 Title: ***The Protective Role of Sleep in Regulating Cortical Response to Social Stress***

Authors: **Eva-Jeneé Andrews,** Zhimei Qiao, Brittany Bush, Hamadi Brewer, Hadiya Johnson, and J. Christopher Ehlen, Ph.D.

Morehouse School of Medicine, Department of Neurobiology

Mentor: J. Christopher Ehlen, Ph.D.
Morehouse School of Medicine

Background/Significance: Environment and sleep-wake history influence the quality and quantity of sleep. Specifically, negative social encounters can alter individual sleep and behavior, leading to resilience or susceptibility. The mechanisms underlying resilience are largely unknown. Recent evidence from our lab demonstrates that non-rapid eye movement (NREM) sleep contributes to resilience to social-defeat stress. The medial prefrontal cortex (mPFC) suppresses limbic circuits responsible for negative behavioral responses to stress and predicts resilience. *Based on this evidence, we hypothesize that NREM sleep within the mPFC determines resilience.* These studies characterize the mPFC single-unit activity in socially defeated mice to assess the role of sleep.

Methods: Mice, 8-10 weeks old, expressing (CaMKIIα-Cre X Ai32) channelrhodopsin-2/EYFP exclusively in CaMKIIα neurons were used in this study. After identifying CaMKIIα-expressing neurons using blue-light stimulation, we recorded multi-unit activity (MUA) and local field potential changes in mPFC neurons. This was before, during, and after social-defeat stress administered using a resident intruder paradigm.

Results: Preliminary findings suggest that firing patterns in mPFC CaMKIIα neurons differ in mice resilient to the effects of social defeat stress. Furthermore, these results suggest that resilience is predicted by sleep-related local firing patterns in the mPFC. These findings suggest that sleep may serve to enhance the ability of mPFC cells to promote resilience to social-defeat stress.

Conclusions and Implications: Sleep has a protective role in the resilient behavioral response based on the relationship between the firing patterns of mPFC neurons in response to social-defeat stress and prior sleep-wake history.

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P-29 Title: ***Acid-sensing ion channel 1a in long-term stroke recovery***

Authors: **Ariel Armstrong** [1], Tao Yang [2], Tiandong Leng MD, PhD [2], and Zhigang Xiong MD, PhD [2]

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Mentor: Zhigang Xiong MD, PhD
Morehouse School of Medicine

Background/Significance: Stroke is a leading cause of death and long-term disabilities in the United States. Tissue plasminogen activator (tPA) is currently the only FDA approved therapeutic for ischemic stroke but must be administered within 4.5 hours of stroke symptom onset and it can be dangerous for many patients because of severe side effects. Searching for new targets and mechanisms involved in stroke recovery may lead to alternative and more effective therapeutic strategies.

Acid sensing ion channel 1a (ASIC1a) is a proton-gated cation channel in the central nervous system that has well-established roles in several neurological disorders. Our lab was the first to demonstrate a critical role for ASIC1a activation in ischemic brain injury. The application of ASIC1a antagonist reduces neuronal death in stroke conditions and ASIC1a knockout mice have reduced infarct size after middle cerebral artery occlusion (MCAO). However, most studies done to establish the role of ASIC1a in stroke are short-term and only demonstrate their acute impact. Also, it is unknown if the reduction in infarct size translates to improved behavioral recovery. The objective of this study is to determine if ASIC1a contributes to long-term stroke survival and behavioral recovery in mice and to explore the underlying mechanisms.

Methods: 30 min MCAO was performed in wild-type and ASIC1a$^{-/-}$ mice. 24 hours after MCAO, mice were assessed for neurological deficits, and then underwent behavioral analysis weekly following MCAO for 4 weeks. Neurological deficit and behavioral analysis scores were analyzed for relative improvement over the 4 weeks and the differences between ASIC1a$^{-/-}$ and wild-type mice were compared.

Results: ASIC1a$^{-/-}$ mice showed no difference from wild-type mice in neurological focal deficit scoring. However, ASIC1a$^{-/-}$ mice had a decreased general sickness score by day 7 post-MCAO while wild-type mice did have a decreased general sickness score until day 14. After MCAO ASIC1a$^{-/-}$ mice had decreased mobility in the open field test for 28 days, while wild-type mice only showed decreased mobility until day 5 post-MCAO. Wild-type and ASIC1a$^{-/-}$ mice did not show differences in sidedness bias in the corner test nor motor strength and coordination in the vertical pole test.

Conclusions and Implications: These findings suggest that ASIC1a plays a multifactorial role in stroke injury/recovery - mediating injury in the acute phase of stroke and recovery in the sub-acute phase of stroke. Determining how ASIC1a affects stroke injury and long-term behavioral outcomes will aid in the development of new stroke therapies.

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P-30 Title: ***The role of cimetidine on lead-induced neurotoxicity***

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Mentor: Kennie Shepherd, PhD, Morehouse School of Medicine

Background/Significance: Lead (Pb) a naturally occurring element, has been demonstrated to be an occupational toxin as well as a ubiquitous environmental toxicant. Despite various preventative measures to reduce Pb accumulation and release in the environment, Pb continues to remain a public health problem. Although extensive research has addressed Pb induced toxicity, therapies to alleviate Pb induced toxicity are limited and have very serious side effects. Oxidative stress plays an essential role in Pb induced neurotoxicity. Specifically, production of reactive oxygen species and mitochondria dysfunction proceeds cell death induced by Pb. Additionally, intracellular modulation of antioxidants has also been demonstrated after exposure to Pb. Organic Cation Transporters (OCT's) have been demonstrated to be a target of toxicants, including metals. Previously, our lab has demonstrated an inhibitor of the Organic Cation Transporter 1 (OCT1), attenuates some of the markers of Pb induced neurotoxicity. However, the OCT1 inhibitor had several limitations, such as it had a very narrow therapeutic window of effectiveness, and it only prevented a few markers of Pb induced neurotoxicity. Thus, its plausible that an inhibitor of multiple types of the OCT may have a better therapeutic window and offer better protection against Pb induced neurotoxicity. The goal of this project was to determine the role of cimetidine (a pan- inhibitor of OCT's) in Pb induced neurotoxicity. The central hypothesis is that cimetidine will prevent Pb induced neurotoxicity.

Methods: To test our hypothesis, we first characterized the effects of cimetidine on mitochondria function, and cell viability of differentiated human neuroblastoma (SH-SY5Y) cells, and then we assessed the role of cimetidine on Pb induced neurotoxicity. Markers used to assess neurotoxicity included measuring the production of reactive oxygen species (ROS), glutathione levels, assessing mitochondrial dysfunction, and cell viability.

Results: Treatment of cell cultures with 100 μM Pb acetate caused an increase in ROS, mitochondrial dysfunction, and a subsequent reduction in cell viability. Interestingly, Pb caused an increase in ROS and mitochondrial dysfunction at low concentrations (1 μM) that does not reduce cell viability. Pretreatment with cimetidine in concentrations ranging from 0.1 to 100 μM prevented Pb induced ROS production, mitochondrial dysfunction, and reductions in cell viability. Additionally, cimetidine significantly attenuated Pb acetate uptake in differentiated human neuroblastoma (SH-SY5Y) cells.

Conclusions and Implications: In summary, the current project establishes that cimetidine prevents Pb induced neurotoxicity, and possibly exerts its neuroprotective action by reducing Pb uptake into differentiated human neuroblastoma (SH-SY5Y) cells. Therefore, continued research of cimetidine on in vivo models of Pb exposure may offer potential insight into additional therapeutic strategies to mitigate the toxic effects associated with Pb exposure.

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P-31 Title: ***Sex differences in sleep exist prior to exposure to social defeat stress***

Authors: **Brittany Bush**[1], Affra Mohamed[1], Caroline Donnay[1], Eva Andrews[1], Hadiya Johnson[1], Gabrielle Cain[1], Ashton Arocho[1], Chioma Okafor[1], and Christopher Ehlen[1]

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Mentor: J Christopher Ehlen, PhD, Morehouse School of Medicine

Background/Significance: Poor sleep quality is linked to neuropsychiatric disorders in men and women. Social defeat stress is frequently used to model features of neuropsychiatric conditions. Evidence from this model shows that sleep differences predict stress-induced maladaptive behavioral outcomes and that NREM sleep plays a causal role in such behaviors. Despite this, the lack of an effective female model prevented similar investigations in females. An animal model of social stress in female rodents may provide the opportunity to conduct these investigations. In the present study, we test the hypothesis that sleep differences predict behavioral responses to social defeat stress in female mice.

Methods: We recorded electroencephalographic (EEG) data in a cohort of female and male mice at baseline and after exposure to a six-hour sleep restriction. This was done both before and after exposure to social defeat stress. The defeat model used a resident-intruder paradigm where pairs of one female and one male mouse were maintained in the same cage, separated by a perforated barrier, throughout the duration of the study. Pairs were then exposed to social defeat stress simultaneously. Twenty-four hours after 10 days of social defeat stress, social avoidance was tested against a caged novel mouse. Sleep was score in ten second epochs and comparisons were made between sexes and animals susceptible and resilient to social defeat stress.

Results: We observed significant sex differences between mice that displayed resilience or susceptibility to social defeat stress, prior to social stress exposure. In baseline sleep, resilient female mice displayed increased NREM sleep during the active period compared to males. Conversely, susceptible males displayed increased NREM sleep, and REM sleep-time when compared to females. During recovery from sleep deprivation, both resilient and susceptible female mice displayed increased recovery of NREM and REM sleep immediately following sleep restriction. This difference was not observed in the males. However, resilient and susceptible males had increased REM sleep-time during the active period, when compared to females. No differences in slow wave activity (SWA) were observed in susceptible males or females; however, there were significant differences in NREM SWA in both resilient male and female mice following sleep restriction. Resilient males displayed an increase in NREM SWA following sleep recovery during the inactive period, while resilient females displayed a decrease in NREM SWA during the active period.

Conclusions and Implications: Our results show that pre-existing differences in sleep regulation predicts the behavioral responses to social defeat stress in both males and females. Furthermore, there are sex differences in sleep patterns that predict these outcomes. Our findings suggest that sex plays a significant role in the interaction of sleep, stress and behavior, and that sex differences in behavioral responses to stress may be partially due to differences in sleep and sleep regulation.

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P-32 Title: ***The Effect of Chronic Sleep Deprivation on ΔFosB expression in the Limbic System***

Authors: Gabrielle Cain, Hadiyah Johnson, and J. Christopher Ehlen**,** PhD

BS/MS in Neuroscience Program, Morehouse School of Medicine

Mentor: J. Christopher Ehlen, PhD
Morehouse School of Medicine

Background/Significance: The prefrontal cortex (PFC) is part of the limbic circuit regulating maladaptive responses in neuropsychiatric disorders. The PFC is also sensitive to the effects of sleep deprivation. Importantly, the expression of ΔFosB in the PFC is necessary and sufficient for susceptibility to maladaptive behaviors in a mouse model of stress-induced neuropsychiatric disorders. This implicates ΔFosB in the PFC as a potential interaction point of sleep and stress. Notably, the expression of ΔFosB in the prelimbic cortex increased after social defeat only in mice that were susceptible to the stressor. Studies have also shown region-specific increases in ΔFosB, after chronic sleep deprivation. Despite this evidence, to our knowledge the effect of chronic sleep deprivation on ΔFosB has not been measured in the PFC. Knowledge of this response will provide a better understanding of the interactions of stress and sleep potentially through ΔFosB and how this relationship contributes to executive function and emotional processing.

Methods: We will be sleep depriving 8 male CD57/BL6J mice. At the beginning of the study, these mice will be 8-10 weeks old. Sleep deprivation will be accomplished by an automated rotating bar and will last for 10 days, 8 hours each day. On the 10th day, an hour after the end of the sleep deprivation, the mice will be sacrificed through cardiac perfusion and their brain sections will be observed and analyzed for the presence of ΔFosB using immunohistochemical analysis.

Results: Preliminary findings indicate increased ΔFosB in the PFC when compared to non-stressed controls. These changes were observed in two areas of the limbic cortex: the infralimbic and prelimbic cortex.

Conclusions and Implications: Recent evidence indicates chronic sleep changes play a direct role in resilience to social-defeat stress. Furthermore, there is a well-established role of ΔFosB in resilience. Determining the expression of ΔFosB after chronic is an important first step in determining the molecular pathways that may be involved in this interaction of social stress and sleep loss. The alteration of ΔFosB after sleep deprivation would implicate this molecule as a key mediator between sleep and stress interactions in the prefrontal cortex

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P-33 Title: ***The Role of Sleep Homeostasis in Susceptibility to Social Defeat Stress***

Authors: **Caroline Donnay, MS**[1] and J. Christopher Ehlen, PhD[2]

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Mentor: J. Christopher Ehlen, PhD
Morehouse School of Medicine

Background/Significance: Stress and sleep abnormalities are associated with increased risk of various neuropsychiatric disorders such as PTSD, schizophrenia, anxiety, and depression. Exposure to acute and chronic stressors can result in numerous behavioral and physiological changes including the alteration of the homeostatic process, a key process regulating sleep. Disturbed sleep is a core feature of many neuropsychiatric disorders, but the relationship between the regulation of sleep, stress and the development of these disorders is relatively unknown. In order to understand this relationship between the regulation of sleep and stress, we have employed a mouse model wherein sustained changes in behavior occur following social defeat stress. Only some animals are susceptible to the effects of social defeat stress, others are resilient; this provides the opportunity to explore differences in sleep and sleep regulation between the two populations. Preliminary data from this model is the first to demonstrate that the development of one of these behaviors, social avoidance, can be predicted by differences in the regulation of sleep before and after exposure to social stress. Based on these findings, we hypothesize that the regulation of sleep before and after exposure to social stress underlie the behavioral responses. Furthermore, we predict that social stress can alter sleep-regulatory mechanisms.

Methods: In this study C57BL/J6 mice were sleep deprived for the first 6 hours of the dark period, given 5 days to recover and then experienced 10 days of social defeat stress. The mice were tested after social defeat stress to assess susceptibility to social avoidance.

Results: Animals susceptible to social avoidance, show differences in homeostatic regulation of sleep, as measured by wave incidence and NREM SWA, prior to social defeat stress, compared to animals susceptible to social avoidance.

Conclusions and Implications: Social defeat stress changes the homeostatic regulation of sleep, measured by NREM sleep after sleep deprivation, in resilient animals. This study adds to further understanding the role of sleep in vulnerability to maladaptive behavior.

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P-34 Title: ***The Effects of Cannabinoids on Sleep: An exploratory report***

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Mentor: Angelita P. Howard Ed.D, MBA-PM, MA, MS
Morehouse School of Medicine

Background/Significance: Sleep deprivation adversely impacts various domains of health-related quality of life among many individuals in the United States. The purpose of this analysis is to assess the effects of cannabinoids with terpenes on the sleep cycle. The CDC states that 70 million Americans suffer from a sleep disorder with insomnia being the most common. The sleep cycle progresses in 4 stages. Stage I is drowsy sleep, Stage II is light sleep, and Stage III is moderate to deep sleep. The deepest sleep occurs during Stage IV which is characterized by high amplitude oscillations on EEG indicative of restorative sleep. The sleep cycle typically takes approximately 1 hour, and the beginning of sleep is initiated by the release of GABA which inhibit other NTs such as histamine, serotonin, NE & other excitatory NTs. These NTs are responsible for the level of arousal & motor activities. Recently Cannabinoids, specifically CBD (cannabidiol), have been scientifically demonstrated to be medically useful as an analgesic and demonstrated significant medical efficacy in various diseases. Additionally, terpenes like myrcene have been associated with a reduction in pain, anxiety, and insomnia. We hypothesize that there will be a positive correlation between the use of CBD alone or with terpenes for an increase in restorative sleep. Based on the neuropathological pathways activated during REM sleep, we hypothesized that CB1 receptors will be found in the same areas interfacing with the sleep pathways. These pathways decrease wakefulness and initiate restorative sleep. There is compelling evidence that suggests calming effects of CBD on the central nervous system however, no clinical studies currently exist in any Psychiatric literature as evidence. This case study seeks to elucidate the mechanism of action associated with the pathophysiology of sleep disorders and the effects of the Endocannabinoid system on sleep.

Methods: Analyses were conducted using data from peer reviewed journals, PubMed databases, and Google Scholar databases. The data revealed studies that improve the knowledge of the neurobiological mechanisms controlling the regulation of sleep homeostasis. The studies also investigated circadian rhythms, physiological hyperarousal, genetics, stress, and cognition. These factors are needed to adequately evaluate the causes and mechanisms of sleep disorders.

Results: Studies suggested that cannabinoids could improve sleep quality, decrease sleep disturbances, and decrease sleep onset latency. Intended results will show that CBD, MCT, and terpenes drastically improve sleep quality by interfacing with the ECS and increasing restorative sleep while decreasing pain, anxiety, and sympathetic tone.

Conclusions and Implications: Sleep is a vital function of the nervous system that contributes to brain and body homeostasis, energy levels, cognitive ability, and other key functions. Dysfunctional sleep induces neural problems and is a key part of almost all human psychiatric disorders including substance abuse disorders. Cannabidiol and specific terpenes like myrcene may hold benefits for sleep-related disorders. There is need for larger, controlled trials to further investigate the molecular mechanisms of cannabinoids and their safety and efficacy for treating sleep disorders.

P-35 Title: *Transcriptomic Responses to HIV-1 in Astrocytes*

Authors: Je'Niesa Manning[1] and Walter Royal, III, MD[2, 3]

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Mentor: Walter Royal, III, M.D., Morehouse School of Medicine

Background/Significance: Many people who are infected with Human Immunodeficiency Virus-1 (HIV-1) experience HIV-associated neurocognitive disorders (HAND). Over the past 40 years since discovering HIV was the cause behind AIDS there has been much progress in managing the more severe effects of the disease. Despite the development of antiretroviral therapy, many still experience neurocognitive impairment (NCI). The causes of NCI in HIV infected people are still poorly understood, but reports indicate inflammation, oxidative stress, and apoptosis, as a few of the causes of NCI. Additionally, it is reported the percentage of people infected with HIV smoke cigarettes an average of 2-3x more than the national average. This affects the whole body's immune response further exacerbating the inflammatory response. The objective of this study is to identify molecular pathways that are activated and associated with proinflammatory responses in HIV-1 rat astrocytes transfected with HIV genes and exposed to components of cigarette smoke extract (CSE).

Methods: Primary rat astrocytes were isolated from wild-type rat brain and cultured in DMEM-F12 medium containing 5% FBS and penicillin-streptomycin. Astrocytes were transfected with pEVd1443 plasmid or control plasmid (pUC18) using lipofectamine. pEVd1443 contains a HIV-1 provirus rendered non-infectious by deletions present in the reverse transcriptase and integrase genes. Optimization of transfection time was performed using a time course experiment at time points 24, 48, 72, and 96 hours to determine optimal transfection time for astrocytes. RNA was isolated and expression of the HIV protein *Nef* was measured utilizing RT-PCR. Differences in expression levels were compared using GraphPad software.

Results: Astrocytes were transfected with the plasmid pEVd1443. Preliminary results indicate successful ability to transfect astrocytes and maximum RNA expression of *Nef* occurs at 72 hours. Fold change compared to wild type astrocytes are 27.3 at 24 hours, 39.2 at 48 hours, 47.9 at 72 hours, and 6.7 at 96 hours. Statistical analysis showed significance with a p-value < 0.0001 for each time point except 96 hours, which showed no significant change.

Conclusion and Implications: Results indicate successful transfection of cells, and 72 hours is the timepoint for optimal RNA expression. In future studies, transfection efficiency will be measured using fluorescent expressing plasmids, and imaging. Total RNA sequencing will be used to study differential gene expression. There will be four experimental groups that will be sequenced: pEVd1443-CSE$^+$, pEVd1443-CSE$^-$, Puc18-CSE$^+$, and Puc18-CSE$^-$. Investigating the roles astrocytes hold in the CNS during HIV infection and how CSE affects those roles will provide a specific insight that will direct future studies. Targets for new therapy might be pursued using information from this project. It has the potential to improve the quality of life in individuals with HIV-1.

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P-36 Title: ***Siah2 Contributes to Female Specific Metabolic Effects that Protect Estrus Cycle and Circadian Clock Function***

Authors: **Kiandra Smith** [1, 2] and Jason DeBruyne, PhD

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Mentor: Jason DeBruyne, PhD
Morehouse School of Medicine

Background/Significance: The circadian clock system tailors an organism's behavior and physiology to constant day-night environmental cycle. While the circadian biology field has been vastly growing, the regulatory effects of ubiquitin E3 ligases on circadian clock function remain a gap in our understanding of circadian function. We have previously found that the E3 ubiquitin ligase SIAH2 is a female specific regulator of circadian rhythms and metabolism (a first of its kind) in the liver. Here wanted to extend these studies to examine the role of Siah2 in regulating rhythms in other tissues, as well as to consider the estrus cycle present in adult females (which also has a broad effect on circadian rhythmicity in the liver).

Methods: We collected liver, white adipose and kidney tissues at 3-hour intervals around the daily clock, across both the proestrus and estrus days of the estrus cycle (when the most genes cycle) from both wild type and Siah2 KO females. RNAs were extracted and subjected it to RNAseq to quantify all mRNA transcripts present in each tissue, from each genotype and at each time of day (96 samples harvested from 32 mice). Data analyzed using Nitecap (website) which provides statistical approaches to assess circadian parameters in transcriptomic data.

Result: In contrast to our previous results, we found that Siah2 loss drastically impacted the circadian clockwork in all three tissues- but that this effect was limited to the Proestrus phase of the estrus cycle.

Conclusions and Implications: This result suggests that Siah2 may be a 'protecting' the circadian clock work from the cycling estrus/hormones in females – providing a "estrus-cycle compensation" mechanism. This was unanticipated but is consistent with the circadian clock being robust against other cycling 'environmental' factors. In addition, it suggests that Siah2 mice undergo transient environmentally induced 'circadian disruption' caused by the estrus cycle. Moreover, combined with our previous data, it suggests the circadian system sits at the top of a cascading mechanism that fully regulates circadian rhythmicity – and that clock perturbations on one day may only have affects several days later due to a 'cascading' type of transcriptional output network.

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P-37 Title: A Multicenter International Study on Incidence and Predictors of Poor Functional Outcomes despite Successful Endovascular Recanalization in Acute Stroke

Authors: Vanesha Waiters, BS[1], Jonathan Grossberg, MD[2], Laurie Dimisko, BSN[2], Brian Howard, MD[2], C. Michael Cawley, MD[2], and Ali Alawieh, MD, Ph.D.[2]

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Mentor: Jonathan Grossberg, MD

Background/Significance: Despite the high efficacy of endovascular thrombectomy in improving patient outcomes following acute ischemic stroke, less than 50% of patients with successful recanalization achieve functional independence at 90 days. The objective of this study was to explore demographic, radiographic and procedural variables that predicted mismatch between successful recanalization and functional outcomes after stroke thrombectomy using the STAR multicenter registry.

Methods: Patients undergoing endovascular thrombectomy for ischemic stroke were reviewed from the Stroke Thrombectomy and Aneurysm Registry, a prospectively maintained international multicenter registry of patient outcomes. A total of 32 centers contributed to this study from the US and globally. Successful recanalization was defined as Thrombectomy in Cerebral Ischemia (TICI) score 2B, 2C or 3 corresponding to more than 50% reperfusion of the target territory. Functional outcome was measured using the modified Rankin Score for disability at 90 days. An mRS score of 0-2 was considered good outcome. Only patients with successful recanalization (TICI≥2B) were included and dichotomized based on functional outcome. TICI-Rankin mismatch was defined as poor outcome despite successful recanalization. Univariate and multivariate analyses for predictors of functional outcome were included.

Results: A total of 6693 patients were included in the study. Good outcome was achieved in 42%, TICI-Rankin mismatch rate was 58%, and mortality rate was 18% by 90 days. On multivariate analyses, independent predictors of TICI-Rankin mismatch included female gender (aOR=1.6, p<0.01), black race (aOR=1.4, p<0.01), age (aOR=1.3, p<0.01), baseline disability (aOR=1.6, p<0.05), admission stroke scale (aOR=1.1, p<0.01), and favorable ASPECT score (aOR=0.5, p<0.01). Procedural variables that independently predicted higher rates of TICI-Rankin mismatch included: procedure time > 60 min (aOR=1.6, p<0.01), more than 2 attempts (aOR=1.9, p<0.01), post-operative hemorrhage (aOR=4.7, p<0.01), and intra-operative complications (aOR=1.6, p<0.01). The mismatch rate had significant linear correlation with age (6% increase per 10 years), attempts (6% increase with each attempt), and procedure time (2% increase with each 30 min). The mismatch rate did not correlate with the TICI score (2B vs 2C vs 3) in recanalized patients.

Conclusions and Implications: TICI-Rankin mismatch after ischemic stroke thrombectomy is dependent on baseline functional status and rapid intra-operative recanalization. Post-operative hemorrhage is a major contributor to the TICI-Rankin mismatch.

P-38 Title: ***The Role of Cellular Prion Protein in Cognitive Impairment Following Repetitive Head Injury***

Authors: **Vanesha Waiters**[1], Seema Yousuf[3], Christian Mustroph[2], Sepehr Saberian[1], and David Gimbel[2]

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Mentor: David Gimbel, MD
Emory University

Background/Significance: Chronic Traumatic Encephalopathy (CTE) is associated with short-term memory loss, lack of spatial awareness, and mood changes in individuals exposed to repetitive head injury. Currently there exists no therapy for head trauma besides supportive care. Our preliminary data shows that Cellular prion protein (PrPc) is a protein critical for the generation of CTE after head injury. Using PrPc knockout (PrPc -/-) mice, we have seen a protection against the characteristic memory dysfunction and tauopathy associated with CTE.

Methods: Morris Water Maze (MWM) testing consisted of a multi-day learning trial of wild-type and PrPc -/- mice. For all experimental groups, mice were subjected to a bilateral closed head injury (CHI) with reproducible pneumatic impactions for 10 days. A 6-week recovery period followed to best mimic the delayed nature of CTE initiation. MWM reversal trials was then performed followed by a probe trial. We then performed immunohistochemistry and western blot analysis of an array of proteins to note differences in expression between the PrPc -/- and control mice regarding CTE and head trauma.

Results: Prior to impaction, the time taken to reach the hidden platform is similar between the wild-type and PrPc -/- mice. Following impaction, the wild-type mice display increased latencies in reaching the hidden platform, whereas latencies remain unchanged in the PrPc -/- mice. The impacted PrPc -/- mice exhibited identical memory to unimpacted controls. Furthermore, protein analysis demonstrated critical differences between the PrPc -/- and control mice following impaction. The implication of these differences is that the PrPc -/- mice are resistant to CTE after head trauma.

Conclusions and Implications: Our mouse model of CTE shows profound memory deficit and characteristic histopathological changes. Using the same model, impacted PrPc -/- mice are behaviorally and histologically indistinguishable from uninjured control mice. This observation identifies a novel role for PrPc in the generation of CTE after head injury. PrPc, or its putative intracellular signaling mechanism, provide promising targets for therapeutic intervention.

P-39 Title: ***Epigenetic reprograming of GABAergic neurons in brain ischemia***

Authors: **Nora Jean-Baptiste**, MS[1] and An Zhou, PhD[2]

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[2] Morehouse School of Medicine, Department of Neurobiology

Mentor: An Zhou, PhD
Morehouse School of Medicine

Background/Significance: A stroke is an acute neurologic condition caused by a disruption in cerebral perfusion due to ischemia or hemorrhage, leading to focal deficits. A new focus of research is to explore endogenous, protective mechanisms against ischemia since there are hibernating animals where ischemic tolerance is achieved. Analyses of the proteomic signature of ischemic tolerant brains revealed upregulation of polycomb group (PcG) proteins that suppress gene expression, thereby attenuating the harmful effects of hypoxia and glutamate-induced apoptosis.

PcG protein EZH2 is neuroprotective and high in ischemic-tolerant brains. However, it represses GAD expression and alters the balance between glutamate and GABA neurons. There is a general understanding that increased inhibitory GABA activity attenuates glutamate excitotoxicity. However, recent literature has shown that EZH2 reduces GABA production by repressing GA expression. Therefore, we hypothesize that an increase in EZH2 in an ischemic-tolerant brain is most prominent in subtypes of GABAergic neurons that control other GABA neurons, the latter controlling excitatory neurons. Therefore, an increase in EZH2 may cause disinhibition of the latter.

Methods: Fluorescent immunohistochemistry analyses were performed and analyzed under fluorescence microscopy. The viability of frozen brain sections from mice was first assessed using DAPI to identify the nuclei of neurons. Single staining of EZH2 (PcG protein) and GAD (GABA synthesis enzyme) was performed to assess their relative abundance with various amounts of antibodies in ischemic-tolerant brains. Double staining of EZH2 and GAD was performed to examine the overlap between both proteins and their relative spatial relationship. Primary antibody dilutions were optimized at 1:250 and 1:150. Secondary antibody dilutions were optimized at 1:500.

Results: A higher number of GAD positive cells were detected in comparison to EZH2 positive cells. DAPI staining revealed increased immunoreactivity of cell nuclei, indicating cell viability.

Conclusions and Implications: Immunohistochemistry analyses of ischemic-tolerant brains double stained with GAD and EZH2 revealed intense positive signals, with a high number of GAD-positive cells than EZH2-positive cells. These results suggest a spatial relationship between EZH2 and GABAergic neurons. Future directions include further optimizing analytic protocols of methodology and tissue sample preparation. Exploring relative immunoreactivity of GAD and EZH2 in predominant GABAergic cortical interneuron subtypes may further elucidate their spatial distribution.

P-40 Title: ***Sleep and Social Stress Interactions in the Ventral Medial Prefrontal Cortex***

Authors: **Grace Olofintuyi**[1] and Christopher Ehlen, PhD[2]

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Mentor: Christopher Ehlen, PhD
Morehouse School of Medicine

Background/Significance: Sleep disorders are a known attribute of various neuropsychiatric disorders; these include post-traumatic stress disorder (PTSD) and major depressive disorder (MDD). In these neuropsychiatric disorders, the mechanisms underlying sleep, and behavioral responses to stress, are mostly unknown. Social defeat stress leads to social avoidance; however, some individuals do not show this negative response—they are resilient. Recently, we found that sleep deprivation eliminates resilience to social defeat stress. Furthermore, our evidence shows that changes in the ventral medial prefrontal cortex (vmPFC) may mediate this effect. In this study, we hypothesize changes in mRNA expression occur in the sleep-deprived vmPFC of socially defeated mice. In addition, we hope to identify epigenetic markers in the vmPFC that are associated with susceptibility and resilience.

Methods: Prior to social defeat stress, major differences in slow-wave activity (SWA) existed in the prelimbic and infralimbic regions of the ventral medial prefrontal cortex (vmPFC); thus, we are analyzing the vmPFC to reveal the sleep-stress mechanism and determine if epigenetic machinery is involved. Mice were subjected to 10 days of social defeat stress with or without daily sleep deprivation for 8h. RNA sequencing will be performed on tissue obtained from the vmPFC using the forceps minor corpus callosum (fmi) and lateral ventricle (LV) as landmarks. From the anterior to the posterior, 1 mm coronal sections will be taken from the mice brain.
NEB Ultra II RNA Library Preparation Kits will be used to produce the RNA sequencing data library; in addition, short/long read RNA sequencing will produce a total of 100 million sequencing reads. When comparing the changes in mRNA levels, we will use a fold change > 1.3 to determine if mRNA changes are statistically significant.

Results: We predict there will be mRNA expression differences in the vmPFC of resilient and susceptible mice. Furthermore, we predict that sleep deprivation will reproduce mRNA changes similar to those observed in susceptible mice.

Conclusions and Implications: This study will provide candidate cortex mechanisms responsible for the interaction of sleep deprivation and social defeat stress. In addition, it can delineate a specific role for sleep in the cortex. Overall, these studies will enhance our understanding of how sleep is involved in the development of stress-induced neuropsychological disorders. This knowledge could improve treatment by providing new biomarkers, targets, and potential therapies.

Acknowledgement of Funding:
NIGMS SC1 GM127260 (Ehlen)

P-41 Title: ***Addressing the Social Determinants of Health through Medical-Legal Partnerships***

Authors: **Kosimasichi Orizu**, Amy Zeidan, MD, and Randi Smith, MD

[1] Morehouse School of Medicine, MD Program
[2] Emory University School of Medicine

Mentors: Amy Zeidan, MD
Dr. Randi Smith, MD
Emory School of Medicine

Background/Significance: The purpose of this study was to assess the health harming legal needs (HHLN) of trauma patients at Grady memorial Hospital. We hypothesize that by embedding legal professionals in clinical care settings and partnering with a host of community-based providers and advocates (e.g., housing advocates, community action programs, public health departments), medical-legal partnerships (MLPs) help improve cross-sector communication and problem solving in healthcare.

Methods: A survey screening for health-harming legal needs (HHLN) was created with assistance from Atlanta Legal Aid and HeLP clinic at Georgia State University. Surveys were conducted on trauma patients at Grady Memorial Hospital which included 32 participants. Health-harming legal needs were self-reported by the patients. The data was collected between June 2022 and July 2022.

Results: Among 32 survey participants, 31 (96.9%) reported at least one health-harming legal need. The most common need pertained to financial issues (bankruptcy, collections, paying for medications and medical bills) endorsed by 19 (59.3%) patients. Concerns about access to benefits (SSI, SSDI, WIC, social security) was the second most common need reported by 14 (43.8.%) participants. When asked if respondents were concerned about access to government benefits (specifically Medicare, Medicaid and other government funded health benefits) almost 30% expressed deep concern.

Conclusions/Implications: The findings of this study suggest that health harming legal needs (HHLN) are widespread among trauma patients at Grady. It suggests that there is potentially very high demand for the services that a medical-legal partnership could provide to Grady trauma patients. Further research is needed to collect more data on patient needs and just how effective an MLP would be for Grady Memorial Hospital.

Acknowledgement of Funding:
Funding was provided by the Student Trauma and Resuscitation Research (STaR) Program

P-42 Title: ***Evaluation of HIV/AIDS Policies and their Implications on People in Vulnerable Communities Living with HIV/AIDS in the United States***

Authors: Farah Abaza[1], Maisha Standifer, PhD, MPH[2], and Jareese Stroud[2]

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Mentor: Maisha Standifer, PhD, MPH,
Morehouse School of Medicine

Background/Significance: Black and African American individuals are disproportionately affected by HIV/AIDS accounting for 42% of total new HIV diagnoses in the US in 2019. This project aims to characterize the causes of such stark disparities in HIV diagnoses and care in vulnerable communities through the lens of the Political Determinants of Health (PDOH). The PDOH describe how policy can inform the social determinants of health and can cause systemic inequities in the healthcare continuum. We hypothesize that the disparities in HIV/AIDS prevalence and healthcare for people living with HIV (PLWHIV) is directly or indirectly affected by the PDOH.

Methods: A literature review was conducted focusing on peer reviewed articles published in 2017-2022 based in the United States. Key terms used were “HIV and “Policy” or “Politics” in addition to terms such as “stigma” or “cost”. Additionally, a key informant interview was conducted with Dr. Kathleen Kennedy, Dean of Xavier University’s College of Pharmacy and founding director for the center for minority health and health disparities research and education. Questions included in the interview were open-ended and focused on health care disparities and barriers to care for PLWHIV. Qualitative analyses were performed for both.

Results: Analyses from the literature review and key informant interview show common themes as to why certain communities are disproportionately affected by HIV/AIDS. These themes can be classified into the following categories: stigma, criminalization of HIV, lack of Education (both for the public and in medical education programs), lack of access, and cost.

Conclusions and Implications: The findings of this project show that current policies are directly or indirectly exacerbating healthcare disparities for PLHIV especially in vulnerable communities by creating stigmas that deter patients from receiving care as well as decreasing the accessibility of care. Future recommendations include increasing education for healthcare providers focused on the PDOH and policy changes where applicable.

P-43 Title: ***It Starts in Childhood: Eradicating Comorbid Disease Development Through Early Nutrition Education Intervention***

Authors: **Arlisia Ables-Jones**[1], Vanessa Castro[1], Jennifer Farman[1], Raynesha Franklin[1], Jessica Lyster[1], Timoya McKay[1], and Shavanna Tappin[1]

[1] Morehouse School of Medicine, Physician Assistant Studies Program

Mentor: Sharon Rachel, MA, MPH
Morehouse School of Medicine

Background/Significance: Between 2016-2020 heart disease and hypertension were ranked as the top causes of death in Fulton County. Research revealed substantial comorbid conditions (diabetes, heart disease, hypertension) related to poor nutrition. Studies support the idea that childhood obesity is likely to contribute to chronic diseases. Interviews with NPU-V stakeholders implicated lack of nutrition education within the community. We believe a nutrition education workshop will positively impact the health of children and families, preventing future adulthood diseases. Research suggests adherence to healthier lifestyles in children requires family participation.

Methods: A 3-month series of nutritional workshops will implement health literacy, promote healthier eating, and eliminate comorbidities. 30 participants from NPU-V, aged 8 -13 will be recruited through community schools. We will provide education on nutrition with presentations, hands-on demonstrations, and 'mock supermarket' experiences. A scoring system, via statistical analysis, will quantify data from participant submissions. Submission tools include: MyPlate, Smiley Face Assessment scales, TikTok, and Parent surveys. Points will be assigned for 'healthy foods' per MyPlate build, and 'healthy food' knowledge via TikTok submissions while participating in the nutrition education program. We will compare participant assessments to those within NPU-V that have not received intervention.

Results: We anticipate an increase in nutritional literacy and health outcomes for children and families within the NPU-V. We seek to decrease the incidence of obesity and rates of comorbidities by providing healthy food resources and nutrition education. Incentives will be provided to participants. We anticipate a significant change in food choices, activity levels, and health status.

Conclusions and Implications: Our intervention will promote healthier lifestyle choices within the NPU-V. Increasing health literacy can lead to healthier food choices, increased activity, and regular health maintenance. Intervention potential includes clinician awareness about rates of obesity and malnutrition within the community, fresh food resources and more green spaces. Further research is needed to follow changes in the community's health literacy.

P-44 Title: ***The Relationship Between Anxiety and Self-Management in Black Adults Living with Epilepsy***

Author: Omojolaade Akintade, MSCR [1], Demetrius Geiger, MPH [2], and Rakale Quarells, PhD [2,3]

[1] Morehouse School of Medicine, Doctor of Medicine Program
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Mentor: Rakale Quarells, PhD
Morehouse School of Medicine

Background/Significance: Epilepsy is a brain condition characterized by 2 or more recurrent unprovoked seizures. Studies have shown that people who have epilepsy are more likely to have co-morbid psychological conditions, with one-third experiencing depression and anxiety. However, epilepsy research has focused more on depression and other psychological conditions as the most common comorbidities despite evidence of anxiety being a stronger predictor of poor quality of life than depression. Self-management education was shown to improve behavior and quality of life of individuals with several chronic conditions. The purpose of this study is to investigate the relationship between anxiety and self-management among black adults with epilepsy.

Methods: A total of 82 Black/African American adults with epilepsy participated in a baseline survey completed as part of the Project UPLIFT study. Anxiety symptoms were measured using Generalized Anxiety Disorder (GAD) scale. Self-management was measured using the 38-item Epilepsy Self-Management Scale (ESMS). SPSS was used to conduct descriptive and bivariate analyses to examine the relationship between anxiety on self-management.

Results: The study population included 69.5% women and 30.5% men. Majority of participants completed some college and beyond (63.4%); however, nearly 60% of the participants were earning less than $25,000 annually. Anxiety levels were classified into minimal, mild, moderate, and severe based on the GAD scores with 75.8% of the participants reporting minimal to mild anxiety. The ANOVA performed to compare the mean epilepsy self-management score among the four anxiety levels found no statistically significant differences [$F(3, 78) = 0.833$, $p = 0.480$].

Conclusions and Implications: Although self-management did not differ by anxiety levels, it may be helpful to consider other factors that can confound our variables such as seizure severity and seizure frequency.

Acknowledgement of Funding: This work was funded in part by the CDC funding to Dr. Quarells (#U48DP005042), Summer Scholars in the Community and Health Careers Opportunity Program Academy at Morehouse School of Medicine (#D18HP32123-04-00).

P-45 Title: ***Addressing Health Disparities in the Deaf and Hard of Hearing Community***

Authors: **Germyah Heart**[1] and Sarah Greene, Ph.D.[2]

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[2] Morehouse School of Medicine, Department of Pathology and Anatomy

Mentor: Sarah Greene, Ph.D.
Morehouse School of Medicine

Background/Significance: It is known that there are health disparities in the Deaf and Hard of Hearing (DHH) Communities. Due to these health disparities, DHH patients are at an increased risk for adverse health outcomes. Communication barriers are significant contributing factors to health inequities and the overall quality of care. Healthcare professionals are responsible for ensuring applicable communication access for DHH patients. The purpose of this study was to explore students' responses to an eight-week seminar series focused on DHH patient care.

Methods: Students in all programs at Morehouse School of Medicine were invited to voluntarily participate in the eight-week seminar series during the spring of 2021 and 2022. At the end of the seminar series, the students (n=43) were given a survey to assess the seminar's effectiveness. The survey highlighted how the students' perspectives changed after the completion of the seminars and their key takeaways. The anonymized qualitative data were analyzed utilizing the software, Dedoose (version 9.0.62), as a method for coding and identifying themes. The authors independently and collaboratively reviewed the data to finalize themes.

Results: Six themes emerged from the data analyses. The six themes were understanding healthcare disparities (n=30, 7%), having a better understanding of deaf culture (n=30, 70%), respecting patient autonomy (n=27, 63%), feeling more prepared to work with deaf patients (n=26, 61%), understanding how to effectively work with interpreters (n=23, 54%), and understanding effective communication (n=20, 47%).

Conclusions and Implications: The eight-week seminar series had a positive impact on the students and their future DHH patient care. Students gained more insight into how to effectively work with DHH communities. This study suggests that student participants will ensure proper communication access is readily available and appropriate for DHH patients in the future.

Acknowledgment of Funding: Amy Cohen Efron, Seminar Series Co-Coordinator and the U.S. Department of Education (P03-1B141018)

P-46 Title: ***Targeted and Tailored Communications to the African American Community to Prevent COVID-19 Disease and Premature Death***

Author: **Eden Obomeghie** and Sonja Hutchins, MD, MPH, DrPH

Department of Community Health and Preventive Medicine
Morehouse School of Medicine

Mentor: Sonja Hutchins, MD, MPH, DrPH
Morehouse School of Medicine

Background/Significance: After US reporting of COVID-19 disease began in January 2020, African Americans (AA) led in reported risk for cases, hospitalizations, and premature death. National targeted and tailored communications for AAs began in March 2020 by national AA organizations and coalitions about the AA health risk and CDC community mitigation recommendations to prevent disease. A reduction in health risk for AAs followed, suggesting a link with specific communications. This study describes the communications and their relationship to health risk for COVID-19 and improvements in vaccination among AAs.

Methods: An ecological study was conducted that compared the timing of national targeted and tailored communications to AAs with COVID-19 risks as health outcomes and vaccinations as a process measure by race/ethnicity. The type and timing of communications from key AA organizations were obtained from their websites.

Results: Communications targeted to and tailored for AAs by the National Medical Association and the Black Coalition Against COVID-19 began as early as March 2020 and were consistent throughout the pandemic. Reductions in risk of COVID-19 cases, hospitalizations and deaths followed.

Conclusion and Implications: This relationship suggests that targeted and tailored communications to AAs precede reductions in AA health risks (compared with some racial and ethnic populations) and improvement in vaccination.

P-47 Title: ***COVID-19 and the Mental Health Concerns at Paul L. Dunbar Elementary School***

Authors: Miranda Perry, MPH [1], Lakesha Tables, MD, MPH [1], Anita Bouie [2], and Martina Jackson Brown, EdS [2]

[1] Morehouse School of Medicine
[2] Paul L. Dunbar Elementary School

Mentor: Lakesha Tables, MD MPH
Morehouse School of Medicine

Background/ Significance: Paul L. Dunbar Elementary School is a Title I school located in Mechanicsville, Atlanta where the majority of the students are Black/ African American and qualify for free or reduced lunch. The COVID-19 pandemic has led to the declaration of a national state of emergency regarding child and adolescent mental health and racism has been identified as a major factor of mental health inequities of this age group. The purpose of this assessment was to determine the mental health needs at Dunbar Elementary School.

Methods: An MSM medical student conducted key informant interviews with staff. Themes and prioritization of the most pressing mental health needs from key informant interviews along with the theory of intersectionality were used to design and implement culturally relevant mental health interventions at Paul L. Dunbar Elementary School.

Results: The six themes identified included home environments, gender differences in mental health concerns, current resources in place, the major impact of COVID-19, resource need, and mentorship need. The identified priorities were hygiene, mental health, and self-esteem. The interventions included meditation videos, affirmation posters, and collection of toiletries found to be more expensive in neighboring stores through a hygiene drive.

Conclusions and Recommendations: Our findings suggest the need for short-term and long-term mental health resources at Dunbar Elementary School. Schools must build community partnerships to provide basic needs support, mental health support, and academic support all in one place.

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- Community Site: Paul L. Dunbar Elementary School
- Community Site Leaders: Ms. Anita Bouie and Mrs. Martina Jackson Brown

P-48 Title*: Factors Associated with Genetic Counseling and Testing for Black Women Diagnosed with Triple Negative Breast Cancer in the United States*

Authors: **Carolina Mahaffey** [1] and Brian M. Rivers, Ph.D., MPH [2,3]

[1] Morehouse School of Medicine, Master of Public Health Program
[2] Department of Community Health and Preventive
[3] Cancer Health Equity Institute

Mentor: Brian M. Rivers, Ph.D., MPH
Morehouse School of Medicine

Background/Significance: Black women have a higher occurrence of early-age onset breast cancer before 50 years old and are two times more likely to be diagnosed with Triple Negative Breast Cancer (TNBC). There are disparities in awareness and utilization of genetic testing for inherited breast cancer with lower rates being seen among Blacks compared to non-Hispanic whites. The purpose of this literature review is to better understand the factors associated with Black women's receptivity to genetic counseling and testing. The research question that guided this research review is as follows: What multi-level factors contribute to black women's willingness to engage in genetic testing/counseling?

Methods: A literature search was performed using the online database: PubMed. This systematic review consisted of search parameters from January 2012 to June 2022. Keywords guiding this search included: "TNBC", "genetic counseling", "genetic testing", "genetic mutations", "black women", and "cancer health disparities".

Results: The findings indicated that there were lower genetic testing rates in Blacks compared to non-Hispanic whites. Factors associated with low testing rates among Black women included lack of awareness of genetic counseling and testing, low referral rates, geographical location, cost of testing, insurance coverage, distrust in medicine, and limited recall of family history. Additionally, women were more receptive when they perceived the benefits of genetic counseling, sharing information with family members, being informed about their health, and feeling worthy of contributing to society and science through research.

Conclusions and Implications: Genetic counseling and genetic testing are underutilized resources among black women with TNBC who is most affected. The most common factor associated with low testing rates was the lack of awareness of the services. There should be an increase in education on the importance of these resources is warranted in the Black community to cause receptivity and use of these vital services. Continued research is warranted for the benefits of genetic counseling and testing in Black women diagnosed with breast cancer.

Acknowledgment of Funding:
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THREE MINUTE THESIS® Participants

NAME	PRESENTATION TITLE
Kiam Preston, Jr.	Thankful for Night Vision
Kaylin Carey	Ovarian Cancer Cells, Oh, the Places You'll Go!
Xiting Lin	Rising Antibiotic Resistance: Tracking the Storm
Melayshia McFadden	Weeding out Ovarian Cancer
Aliyah Anderson	The Influence of Age & Sex on Traumatic Brain Injury Outcomes
Mikaili Abdullah	The Efficacy of Dexamethasone as a Ciliogenic Agent in Prostate Cancer Cells

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