GMS Presents:

SUMMER TRAINING AS RESEARCH SCHOLARS (STaRS) PROGRAM

Research Symposium

Thursday, August 6, 2020

Boston University Graduate Medical Sciences
Summer Training as Research Scholars

STaRS Research Symposium

Boston University School of Medicine

Thursday, August 6, 2020
12:00 AM – 1:30 PM

Welcome to the Annual Summer Training as Research Scholars (STaRS) Research Symposium, hosted by the Division of Graduate Medical Sciences.

The STaRS program would like to acknowledge the achievements of our students and the faculty and researchers across BU Medical Campus whose mentorship has enhanced both the quality of our students' work and their overall summer experience.

The students have spent this summer conducting research in labs across the BU Medical Campus and we hope you enjoy learning more about their projects.

Dr. Isabel Dominguez, STaRS Director

2020 Scholars

The summer 2020 cohort of STaRS scholars included 15 scholars selected of almost 400 applicants. The STaRS program hosted scholars from the AHA- SURE program that are also participating in the symposium.

STaRS Scholars
Princess Maryam Abdul-Akbar
Erika Beyer
Rachel Choate
Cassie Deshong
Cisco Espinosa
Lazaro Fernandez
Cynthia Flores
Reyna Gariepy
Ziko McLean
Asel Mustafa
Andrea Navarrete Vargas
Elizabeth Nelson
Joseph Waiguru
Madeline West
Carolyn Wilson

AHA-SURE Scholars
Destinee Bledsoe
Germya Bradley
Ayana Gray

In this booklet, abstracts are organized in alphabetic order by poster session

GerMya Bradley
Tougaloo College, Class of 2021

The Association between a Polysocial Risk Score and Incident Atrial Fibrillation in the Jackson Heart Study Cohort

GerMya Bradley, Ludovic Trinquart, PhD

Department of Biostatistics
Boston University School of Public Health, Boston, MA, USA

BACKGROUND and SIGNIFICANCE: About 5.1 million people in the United States are affected by atrial fibrillation (AF). AF is associated with a considerable increase in risk of stroke and premature death. The lifetime risk of AF is 1 out of 3 in whites, but only 1 out of 5 in Blacks. Paradoxically, African Americans in the United States have a higher prevalence of AF risk factors and the stroke incidence rate after AF is nearly double the rate for Whites. Social determinants of health (SDoH) influence cardiovascular risk. Previous studies, based on Jackson Heart Study data, have found that perceived discrimination, psychosocial factors, and lifecourse socioeconomic position are associated with CVD risk in African Americans. Moreover, the accuracy of the Pooled Cohort Equations Risk Model to predict major atherosclerotic cardiovascular disease-related events is influenced by neighborhood socioeconomic position.
Accurate risk prediction is important for primary and secondary prevention of AF. The CHARGE-AF risk prediction model for AF is based on clinical risk factors. Incorporating SDoH may improve risk prediction of AF. We propose to develop a measure of the aggregate burden of socially determined vulnerabilities. The development of a polysocial risk score to modify existing risk models could help us analyze the relationship between SDoH and incident AF.

METHODS: We selected participants ages 45 to 95 years without prevalent AF at Jackson Heart Study's Exam 1. We further selected participants with available data for the CHARGE-AF clinical risk scores. To measure the burden of SDOH, we extracted socioeconomic factors (9 variables), psychosocial factors (14 variables), and neighborhood factors (4 variables) based on a review of literature. Socioeconomic factors consisted of childhood amenities, home ownership, and education. Participants' psychosocial factors included a score for major life events, a stress score, and discrimination factors. The last domain consisted of neighborhood factors including problems, cohesion, violence, and median income.

CONCLUSIONS: Over 40% of participants were excluded because of missing data on social factors. In the next steps, we will use a Principal Component Analysis approach to reduce dimension and Confirmatory Factor Analysis to build a SDoH summary index. Lastly, we will assess the association between the SDoH and incident AF by using Cox proportional hazards model.

Erika Beyer
St Francis College, Class of 2021

Defects in the mitochondrial protein quality control and tumorigenesis

Erika Beyer; Hui Feng, M.D., Ph.D.

Departments of Pharmacology and Medicine, Section of Hematology and Medical Oncology, University School of Medicine, Boston, MA, USA

BACKGROUND and SIGNIFICANCE: Mitochondria are the foundation of energy in the cell. The mitochondria employ various quality control pathways in order to sustain an appropriate amount and confirmation of mtDNA. When defects occur in these pathways, various human diseases can arise as a result. In this review paper, the role of defects in mitochondrial protein quality control in connection to cancer is explored since little data has been gathered on this topic previously. Its significance is great, as the review paper serves as a comprehensive overview of the research till present day and explores areas where there is still lack of knowledge, thus serving as a foundation for future research.

METHODS: First an audience and a topic were chosen to start the preparation of the paper. Cancer and those interested in creating anti-cancer therapies were chosen. In order to increase in specificity, a large reading of many research articles was conducted, and it was noted that there were many articles lacking on the mitochondrial quality control check specifically. Many research articles were collected, and the paper was split into sections in order to begin writing. Figures were also created to act as a visual aid for the review paper. Many versions of editing have been done in order to control for scientific and grammatical inaccuracies.

RESULTS: It was found that when translation and or translocation of mitochondrial proteins is disrupted, it can lead to an accumulation of mitoproteins in the cytosol of the cell. This is parallels to what happens to oncoproteins that are also mislocalized in tumor cells as compared to normal cells. For the mitoCPR quality control check pathway, many of the machinery that transports mitoproteins are upregulated in cancer. A decrease in these protein translocases has been found to impair tumor growth and sensitized cancer cells to chemotherapy. For the UPRmt quality control check pathway, its inhibition has been found to kill cancer cells.

CONCLUSIONS: By exploring the role of defects of the mitochondrial quality control check and cancer, certain tumor suppressors and drivers can be identified and subsequently target by anti-cancer medication. Therapy that includes a combination of medication would be needed to keep the mitochondrial quality control check pathways working efficiently, suppress/express tumor drivers/suppressors and safeguard neighboring healthy cells from possible toxic side effects.

Rachel Choate

California State University, Stanislaus, Class of 2020

Identifying key mutations in CK2α in human cancers using cancer genomics databases

Rachel Choate; Elizabeth Nelson; Isabel Dominguez, PhD

Department of Medicine, Section of Hematology and Medical Oncology, Boston University School of Medicine, Boston, MA, USA

BACKGROUND and SIGNIFICANCE: Cancer is a genetic disease caused by mutations. Identifying and characterizing key mutations is important to understand cancer and tumor progression. Mutations in kinases are implicated in cancer, and kinases are major therapeutic targets. Our study focused on CK2α, a highly conserved serine/threonine protein kinase that is considered a proto-oncogene. That is, its transcript, protein, or activity levels are elevated in various human tumors and cancer cells, it promotes tumors when overexpressed in mice, and it is involved in cell survival and proliferation. However, no mutations in CK2α in cancer have been described in the literature. This is a hypothesis-generating study to identify and assess point mutations and mutational hotspots in CK2α transcript.

METHODS: CK2α variants in the coding region were downloaded from cBioPortal (46,959 samples) and Catalog of Somatic Mutations in Cancer (COSMIC, 38,705 samples), which both provide simplified mutation and detailed clinical data. Information from the databases were combined, duplicate samples were removed, and a database of unique mutation data (n=337) was sorted for analysis. We identified cross-tumor mutation distribution and hotspots often mutated. We then used mutation3D to visualize hotspots within functional regions of the CK2α crystal structure. We also analyzed the potential functional effect of the mutations using Provean, Polyphen2, and SIFT.

RESULTS: CK2α point mutations were found in all cancer types except eye, small intestine, adrenal gland, soft tissues, thymus, vulva/vagina, and penile. CK2α mutations were most prevalent in large intestine (13%), skin (11.9%) cancers and lymphoid (6.8%) cancers. The prevalence of CK2α mutations in lung (5.9%), breast (4.7%), and prostate (4.7%), the top 3 cancers with the highest incidence in the US is comparatively lower. We identified nine mutational hotspots (n>5), and made some predictions about two hotspots. Provean and Polyphen2 analysis showed that deleterious and benign mutations were largely found in the first 200 amino acids and last 191 amino acids, respectively. A limited number of mutations fell in kinase domains where cancer driver mutations typically occur, and most of these mutations were predicted to be deleterious.

CONCLUSIONS: Here, we describe for the first time that mutations in CK2α are found in the majority of human tumors. The number of hotspots and potential driver mutations was limited, and they may have functional significance. The effect CK2a mutations in cancer may lay in the first 200 amino acids, where the majority of deleterious mutations are found. Our next steps will investigate the significance of the higher prevalence of CK2α mutations in large intestine, skin, and lymphoid cancers. We will also investigate the structural, functional and cellular consequences of hotspot and potential driver mutations in CK2α. This is a significant step towards our ultimate goal to uncover the mechanism of CK2 in cancer and tumor progression to identify the means to target CK2 therapeutically.

Andrea Navarrete Vargas
University of California, Davis; Class of 2021

WHSC1 knockdown affects gene expression in *Xenopus laevis* embryonic development

Andrea Navarrete Vargas, Parth Patel, Laura Lowery, PhD

Hematology and Oncology Section – Dept. of Medicine, Boston University School of Medicine, Boston, MA, USA

BACKGROUND and SIGNIFICANCE: Wolf-Hirschhorn Syndrome (WHS) is an extremely rare chromosomal disorder estimated in 1 in 50,000 live births. 90% of WHS cases are not linked to inheritance but rather caused by a new deletion that occurs for the first time in the person with WHS. This deletion is located on the short arm of chromosome four and encompasses WHSC1 among several other genes. In addition to pronounced intellectual disability, seizures and delayed growth, WHS presents facial dysmorphisms such as a wide flattened nasal bridge, a high forehead, prominent eyebrow arches and pronounced brow bones, widely spaced eyes, undersized jaw, and abnormally positioned ears with underdeveloped cartilage. These affected craniofacial tissues all derive from a shared embryonic precursor known as the cranial facial neural crest. In previous work done in the lab, when they knocked down WHSC1, several craniofacial defects were observed including increased facial width, reduced neural crest cell migration, as well as reduced brain size. Therefore, due to the reduced levels of WHSC1 in this disorder and its implication in transcription, our objective was to take an unbiased approach to globally assess and determine which genes are affected by WHSC1 knockdown and how these WHS phenotypes can be explained by gene interactions that can be further investigated.

METHODS: To assess gene expression in WHSC1 knockdown embryos, embryos were injected at the two-cell stage with antisense oligonucleotide known as a morpholino, which blocks proper splicing of this protein preventing its translation. As a control, some embryos were injected with control morpholino or water injection to mimic the stress of injection. The embryo then grows until it reaches stage 22 and is processed for RNA extraction and sent to de Novo where the RNA is sequenced. After being sequenced, the data is sent back to be analyzed. We are interested in seeing which genes are downregulated or upregulated as a result of the knockdown. This would give us greater insight into which genes would be interesting to look at further to connect these genes to irregular craniofacial development.

RESULTS: Numerous genes seem to be affected including Vwa5a, Has1, and Casq1. However, we were most interested in those which could be linked to craniofacial development. Vwa5a was found to be significantly downregulated in WHSC1 knockdown versus the control. Vwa5a is a tumor suppressor gene known to play a critical role in breast cancer metastasis by triggering the switch of melanoma cells from a proliferative to a migratory. Hyaluronan synthase 1 (Has1) was also seen to be significantly downregulated. Has1 is essential to hyaluronan synthesis, a major component of most extracellular matrices that has a structural role in tissues architectures and regulates cell adhesion, migration and differentiation. Finally, Casq1 was found to be significantly upregulated. This protein, also known as calmitine, functions as a calcium regulator in the mitochondria of skeletal muscle.

CONCLUSION: We successfully determined candidate genes to include in a grant proposal to further investigate the mechanism by which WHSC1 knockdown affects these genes and in turn affect craniofacial development.

Elizabeth Nelson
Boston University, Class of 2022

Investigating the prevalence and significance of *ATRX*, *DAXX*, *SMARCAL1,* and *SLX4IP* mutations in potentially ALT-positive tumors

Elizabeth Nelson; Rachel Choate; Christopher Heaphy, PhD

Department of Medicine, Section of Hematology and Medical Oncology, Boston University School of Medicine, Boston, MA, USA

BACKGROUND and SIGNIFICANCE: Telomeres are hexameric repeats ($TTAGGG_n$) at the ends of linear chromosomes that protect chromosome integrity. Telomeres shorten after each cell division cycle until a critically shortened length is reached. The cell then enters a state of cellular senescence which limits the number of cell divisions. Cancer cells must overcome this replicative barrier by either upregulating telomerase, or using the Alternative Lengthening of Telomeres (ALT) pathway which uses homology directed repair to maintain telomere length. ALT is present in ~10% of cancers, yet the exact underlying mechanisms are still being discovered. Two known drivers of ALT are loss of function mutations in either *ATRX* or *DAXX*, genes that encode chromatin remodeling proteins. Recent discoveries have shown that mutations in *SMARCAL1* and *SLX4IP* are also associated with ALT-positive cancers. The goal of this hypothesis-generating study is to investigate the prevalence and significance of *ATRX, DAXX, SMARCAL1*, and *SLX4IP* mutations in potentially ALT-positive cancers, along with establishing co-mutation patterns and mutational impact on percent copy number alterations (CNA), in order to identify potential prognostic biomarkers of ALT-positive tumors.

METHODS: We gathered mutation data from 46,959 tumors across a multitude of cancer types from cBioPortal. All curated, non-redundant studies were surveyed for tumor samples with mutations in each of the four genes of interest, as well as associations with co-mutations and percent CNA. A 2x2 chi square test was performed for the co-mutation analysis.

RESULTS: Based on the prevalence data, *ATRX* is most commonly mutated out of the four studied genes. Although, little data is available on *SMARCAL1* and *SLX4IP*. There are statistically significant co-mutations, such as *ATRX* with *TP53* and *IDH1* in several CNS cancers; *DAXX* with *MEN1* in pancreatic neuroendocrine tumors and *DAXX* with *LRP1B* in cutaneous melanoma, skin cancer non-melanoma, and lung adenocarcinoma. Interestingly, CNA analysis revealed uterine endometrioid carcinomas with *ATRX* mutations presented with low percent CNA (mean=2.6%) while having thousands of mutations, likely due to the presence of a *POLE* mutation. Finally, colon adenocarcinomas and CNS tumors also presented with low percent CNA (mean=13.1% and 15.6%, respectively), whereas adrenocortical carcinomas presented with noticeably higher percent CNA (mean=44.6%).

CONCLUSIONS: These results indicate that ALT is associated with mutations in various genes, and the prevalence varies widely by tumor type. The novel co-mutations and percent CNA are also tumor-type dependent. The next steps are to analyze the identified tumor types of interest to determine if the co-mutation patterns are also present in tissue samples and associated with ALT. Further investigation into these genes' roles may unveil potential biomarkers for ALT-positive tumors.

Carolyn Wilson
Boston University School of Medicine, Class of 2023

Inhaled nitric oxide treatment in spontaneously breathing COVID-19 patients

Raj Parikh, **Carolyn Wilson**, and Christine C. Reardon

Department of Pulmonary, Allergy, Sleep & Critical Care Medicine, Boston University School of Medicine, Boston, MA, USA

Background and significance: Inhaled nitric oxide (iNO) is a pulmonary vasodilator that has been utilized as a rescue therapy in patients with severe hypoxemia by improving ventilation-perfusion matching and decreasing pulmonary vascular pressure. iNO was an effective therapy during the severe acute respiratory syndrome (SARS) outbreak in 2003 by improving oxygenation in patients with significant hypoxemia. Given the similarities between the SARS outbreak and the Coronavirus disease-2019 (Covid-19) pandemic, iNO therapy may have a role in Covid-19 patients as well. Here, we describe our experience with utilizing iNO therapy in spontaneously breathing Covid-19 patients.

Methods: We identified non-intubated Covid-19 patients who received iNO therapy. The study was approved by the Institutional Review Board of Boston Medical Center and the requirement for informed consent was waived. The decision to administer iNO was at the discretion of the treating physician and based on institutional clinical guidelines. The starting dose of iNO was 30 parts per million (ppm) for all patients, and the mean duration of therapy was 2.1 days. Estimated SpO_2/FiO_2 (SF) ratio, a surrogate for PaO_2/FiO_2 ratio, was utilized to assess the patient's oxygenation status. Types of respiratory support administered included nasal cannula, nasal pendant with oxymizer, and non-rebreather mask. Descriptive statistics were used to summarize clinical data; categorical variables were reported as counts and percentages. Statistical analysis was performed using SAS v9.4, with $p < 0.05$ considered statistically significant.

Results: There were 39 patients with laboratory-confirmed Covid-19 infection who were treated with iNO therapy while spontaneously breathing. Mean age of the patients was 61 years with an average body mass index (BMI) of 33. A total of 22 patients (56.4%) were male, 18 patients (46.2%) identified as Hispanic, and 24 patients (61.5%) had a pre-existing cardiac condition. Of the 39 patients, 29 (74.4%) were initially admitted to the general medical floor, although 24 of these patients later required transfer to the intensive care unit (ICU). There were 20 hospital discharges, 9 deaths, and the remainder of patients remained hospitalized at the time of analysis. Management of the Covid-19 patients included immunomodulator therapy with an IL6-receptor antagonist (34 patients; 87.2%), hydroxychloroquine (24 patients; 61.5%), azithromycin (21 patients; 53.9%), and self prone (23 patients; 59%). A total of 21 patients (53.9%) did not require invasive mechanical ventilation after treatment with iNO. Of the 21 patients, 20 were successfully discharged and there was 1 death. Median SF ratio prior to iNO initiation were similar between the 21 non-intubated patients (SF ratio: 108) and the 18 patients that eventually required mechanical ventilation (SF ratio: 113). Median Ferritin (intubated: 1002 ng/ml, non-intubated: 625 ng/ml; $p = 0.38$) and D-dimer (intubated: 566 ng/ml, non-intubated: 596 ng/ml; $p = 0.38$) levels were also comparable between both groups, whereas C-reactive protein (CRP) levels assessed prior to iNO therapy were significantly higher in the intubated patients (intubated: 122.9 mg/l, non-intubated: 48.3 mg/l; $p = 0.0108$). Following iNO therapy, the SF ratio improved in the 21 non-intubated patients with a median of 54.9 ($p = 0.0078$). CRP and ferritin did not significantly change after iNO treatment though D-dimer levels increased in 25 patients (64.1%) with a median change of 115 ng/ml ($p = 0.0052$).

Conclusion: From the 39 spontaneously breathing patients with Covid-19 who underwent therapy with iNO, more than half did not require mechanical ventilation after treatment. These findings suggest that iNO therapy may have a role in preventing progression of hypoxic respiratory failure in Covid-19 patients. During the SARS outbreak, researchers hypothesized that iNO may not simply improve oxygenation, but also potentially have an antiviral mechanism of action. The similarities between Covid-19 and SARS are well-documented and our analysis emphasizes the need to further investigate iNO therapy in future Covid-19 studies. Randomized controlled trials are already underway, and findings from such large-scale investigations can ideally reflect upon the role of this therapy in potentially helping avoid mechanical ventilation and improve patient outcomes.

Destinee Bledsoe
Tougaloo College, Class of 2021

Smoking Behaviors, Perception of Risk of Young Tobacco Products Users During the COVID-19 Pandemic

Destinee Bledsoe; Sana Majid; Naomi M. Hamburg, MD

Whitaker cardiovascular Institute, Boston University School of Medicine, Boston, MA, USA

Background: COVID-19, the respiratory illness caused by SARS-CoV-2, has generated a pandemic in the year 2020 that has led to a national shut down. People with certain underlying conditions, such as pulmonary and heart diseases, are at increased risk of worse outcomes from COVID-19. Smoking is a known risk factor for such comorbidities, and current data suggests that tobacco product users might be at increased risk for severe illness from COVID-19.

Objective: To assess the impact of the COVID-19 pandemic on access to, perception towards and usage of tobacco products.

Study Design and Research Methods: With collaborative efforts between Boston University and the University of Louisville, we designed a COVID-19 related questionnaire to implement among 122 people from 18-45-year-old participants in a longitudinal study without pre-existing cardiovascular disease or risk factors who are either tobacco nonusers, combustible cigarette users or electronic cigarette users. The questionnaire asks a range of questions varying from exposure to COVID, frequency of tobacco use, perception related to COVID, and other drug usage. This questionnaire drew from open-source question banks from the Phenx Toolkit and the NIH Public Health Emergency and Disaster Research Response among others and was programmed into the electronic data capture software REDCap. The questionnaire will be delivered to study participants via email. Once data is collected and cleaned, we will use the statistical software SPSS to carry out the analyses. The analysis will cover out a comparison of baseline usage and the changes related to COVID.

Results and Conclusion: The questionnaire is in the process of being delivered to study participants. Upon completion, our study will help gain insight into how tobacco product users were affected by the present pandemic with respect to product access, perception towards COVID-19 outcomes associated with tobacco product use, and any behavioral modifications regarding tobacco use.

Cassie Deshong
Williams College, Class of 2021

The Effect of UFD1 on Tumor Progression

Cassie Deshong, Andrew Lam, Xiaodan Qin, PhD, Hui Feng MD., PhD

Departments of Pharmacology and Medicine, Section of Hematology and Medical Oncology Section, Center for Cancer Research. Boston University School of Medicine, Boston, MA, USA

BACKGROUND and SIGNIFICANCE: In 2018, there were 17 million new cancer cases and 9.5 million cancer deaths worldwide. Moreover, 90% of cancer-associated deaths are because of tumor metastasis. It is crucial that innovative and efficient tactics be discovered to halt tumor progression. MYC, a proto-oncogene, is activated in almost all cancers and upregulated in tumorigenesis. More specifically, there is a correlation with enhanced MYC activity and rapidly spreading aggressive cancer cells. Increased gene transcription and protein synthesis are the two main cellular changes caused by increased MYC activity that aid in cancer cell survival. A consequence of these changes is endoplasmic reticulum (ER) stress. To restore equilibrium, two major pathways are activated: unfolded protein response (UPR) and ER-associated degradation (ERAD). Moreover, recently it has been found that complete inactivation of ubiquitin fusion degradation 1 (ufd1) gene is a key regulator of the ER stress response by impairing ERAD, exacerbating ER stress, and inducing apoptosis. Since the discovery of the importance of ufd1 is relatively novel, more research is needed to fully understand the capacity of the control of the gene.

METHODS: To further assess the control of ufd1, the same MYCN tumor cells were injected into a ufd1 wild type (wt) and ufd1 heterozygous (het) fish. Tumor burden was monitored over a course of 17 days. The tumor cells came from MYCN fish bred with dβh promoter on MYCN and EGFP.

RESULTS: The area and intensity of the tumor were calculated and analyzed for a significant difference. In both the ufd1 wt and het fish, there was an initial increase in tumor burden. However, around day 10, the tumor burden in the het fish decreased and tumor burden in the wt increased. On day 17, most het fish had no tumor present and most wt fish had a significantly larger tumor burden than their initial tumor. There is statistical significance on days 3 to 7 and on the final day, day 17 in tumor burden between wt and het fish. There was no significant difference in tumor intensity between ufd1 wt and het fish for any of the 17 days.

CONCLUSIONS: Our results confirm that partial inactivation of ufd1 continues to play a significant role in halting the progression of tumor burden. Moreover, partially inactivated ufd1 led to the disappearance of the tumor. This insight hints at ufd1 having a larger capability than stopping tumor metastasis.

Cisco G. Espinosa
Cornell University, Class of 2022

Reevaluating Risk Factors of Lung Cancer Screening in a Diverse Safety Net Population

Cisco G. Espinosa; Kei Suzuki, MD

Department of Surgery, Boston Medical Center;
Boston University School of Medicine, Boston, MA, USA

BACKGROUND & SIGNIFICANCE: Lung cancer (LC) screening using low-dose computed tomography (LDCT) is recommended with eligibility criteria of pack-year (PY) ≥ 30, and age ≥ 55 years. Screening yields remain low with published cancer detection rate of 1-2%. Additional clinical risk factors have been proposed; however, these have not been validated in a diverse population.

HYPOTHESIS/ OBJECTIVE: This study sought to review the association of additional clinical factors with LC in Boston Medical Center (BMC), safety-net, diverse populations.

STUDY DESIGN & RESEARCH METHODS: This retrospective study evaluated BMC LC-screening population (BMCP) that met National Lung Screening Trial (NLST) criteria (age ≥ 55 & PY ≥ 30) from 3/2015- 2/2019. For the study population the following variables were collected: sex, smoking status, race, education, body mass index (BMI), history of: chronic obstructive pulmonary disease (COPD), emphysema, pneumonia, personal history of any cancer (PHxCa), family history of lung cancer (FHxLCa), and LC status. The BMC population was compared to the NLST population. Then, associations between collected variables and LC status were analyzed via relative odds ratios (OR) with 95% confidence intervals (95% CI).

RESULTS: The study population consisted of 2,847, of which, 72 patients had LC. Comparing BMCP and NLSTP established significant differences. Median age in BMCP was 62 compared to 60 in NLSTP ($p<0.0001$) and there is a 61.6:38.4% male:female ratio in the BMCP compared to 58:42% ratio in the NLSTP ($p=0.0006$) Regarding race BMCP composed of 39.9% White, 38.2% Black/African American, and 5.9% Other race compared to 90.8%,4.4%, and 4%, respectively, in NLSTP ($p<0.0001$). In education level, BMCP had 36.9% people with <high-school education and 35.6% having graduated high-school (or GED) in comparison to 6.1% and 23.7%, respectively, in NLSTP ($p<0.0001$). BMCP had: 45.9%, 64.9%, 15.0%, and 11.5% incidence of COPD, emphysema, PHxCa, and FHxLCa respectively, compared to 5.0%, 7.7%, 4.2%, and 25.0% in NLSTP ($p<0.0001$). Analysis of BMCP indicates a median age of 65 in the LC+ group compared to 62 in the LC- group ($p=0.009$) as well as a median PY of 49.5 compared to 40 in LC- group ($p<0.0001$). No significant differences were found between LC status and sex, race, education, pneumonia, or PHxCa. The LC+ group had 75%, 93.1%, and 27.8% incidence of COPD, emphysema, and FHxLCa compared to 45.2%, 64.1%, and 11.1% in LC- group ($p<0.0001$). These also yielded OR's (95% CI) of: 3.5 (2.1-6.2), 7.3 (3.0-18.6), and 2.96 (1.8-5.2), respectively, indicating exceptional predictive capacity of these variables with respect to lung cancer status.

CONCLUSIONS: Ultimately, the BMCP was more diverse in smoking history, sociodemographics, and medical history. Following comparison of proportions of the LC+ and LC- populations, smoking history in PY, age, and incidence of COPD, emphysema, and FHxLCa were found to be significant risk factors associated with LC status.

Reyna Gariepy

Bryn Mawr College, Class of 2021

The Association Between Experiences of Discrimination and Hippocampal Subfields in a Diverse Sample of Older Adults

Reyna Gariepy, Michael Rosario, Karin Schon

Department of Anatomy and Neurobiology, Boston University School of Medicine, Brain Plasticity and Neuroimaging Lab

Ayana Gray
Harvard University, Class of 2022

Cardiac Phenotyping in the Framingham Heart Study

Ayana Gray; Chunyu Liu, PhD, Jessica Fetterman, PhD, Yi Li, MS, Deepa Gopal, MD

Cardiovascular Medicine at Boston University School of Medicine

Background and Significance: Heart failure, a clinical entity where the heart is unable to meet the metabolic needs of the body, is a heterogeneous process that has been difficult to capture in cohort studies. While there is a universal definition of prevalent (stage C) HF, researchers use varying definitions of preclinical (stage B) HF. Both stages B and C can be further broken down into two subcategories - HF with preserved ejection fraction (HFpEF) and HF with reduced ejection fraction (HFrEF). The purpose of my work was to analyze echocardiographic phenotypes used to define stage B and C HF in a variety of cohort studies and create a definition for stage B in both HFpEF and HFrEF for eventual application with metabolomic and genomic correlation in the Framingham Heart Study (FHS).

Hypothesis: Metabolomic and genomic signatures will predict heart failure phenotypes in the Framingham Heart Study cohorts. The objective of this work was to define the 4 echocardiographic phenotypes within FHS.

Methods: As abnormal cardiac function and morphology are often our only evidence of HFpEF presence, the American Society of Echocardiography (ASE) guidelines on diastolic dysfunction and chamber quantification were analyzed for key variables of interest. A comprehensive literature search was then performed to evaluate echocardiographic phenotypes in stage B HF definitions used by various large cohort studies. The search terms used to find these studies were “cohort studies”, “stage B heart failure”, “longitudinal study”, and “echo”. Papers published by FHS researchers were subsequently reviewed.

Results: A total of 15 studies were reviewed in conjunction with detailed review of the ASE guidelines. For stage B HF, common phenotypical parameters included the presence of diastolic dysfunction and functional abnormalities including left atrial (LA) dimension, mitral e' velocity, and E/e' ratio, and left ventricular (LV) chamber abnormalities such as LV mass indexed to height$^{2.7}$, global longitudinal strain, and end diastolic volume indexed to body surface area.

Conclusions: We ultimately defined stage B HFpEF using the phenotypes for ejection fraction, LV mass indexed to height$^{2.7}$, average e’, LA volume index, and tricuspid regurgitation velocity. Stage B HFrEF was identified only by reduced ejection fraction. Stage C HFpEF and HFrEF were defined using the Framingham classification of HF using an LV ejection fraction greater and equal to 50% and less than equal to 40%, respectively. Based on these phenotypic classifications for these four HF phenotypes, we plan to apply these categorizations to determine whether these echocardiographic phenotypes relate to HF, overall CV risk outcomes, and metabolomic and genomic data available in the FHS.

Madeline West
Macalester College, Class of 2021

Kinetics of Amyloid Formation by Antibody Light Chains

Madeline West, Gareth Morgan, PhD

Hematology & Medical Oncology, Boston University School of Medicine, Boston, MA, USA

Background and Significance: AL amyloidosis is a rare disease caused by aggregation of antibody light chains as amyloid fibrils. Light chains are proteins secreted by cancerous plasma cells in the bone marrow. The amyloid fibrils build up in the heart, kidney and peripheral nervous system and cause progressive organ damage. To quantify amyloid formation, one can measure how quickly recombinant proteins aggregate and ask which factors influence this process. The kinetics of aggregation tell us about the molecular changes involved in the process. Every patient has a unique light chain protein sequence, but not everyone who has the underlying cancer (multiple myeloma and related conditions) has amyloidosis. Knowing how and why different sequences aggregate could help gain a better understanding of the pathology of the disease, ultimately helping to identify processes that could be a target for therapy.

Hypothesis and Objective: Our research question was why do only some light chains cause amyloidosis? To address this question, we looked at the behavior of how light chains with different sequences aggregate in vitro. Our hypothesis was that all light chains aggregate with the same kinetic mechanism. To test this hypothesis, we wanted to compare the concentration dependent behavior of aggregation and ask whether the light chains all behave in a similar way or whether there are differences between them. The goal of this project was to determine the relationship between the properties of the proteins, its concentration and its aggregation rate. To examine the mechanism of aggregation, we investigated how aggregation rate varies with light chain concentration, which tells us about the differences in initiation and elongation of the fibrils. We aim to use high-level analytical tools provided by the Amylofit software suite to understand the mechanisms of amyloid formation.

Methods: We studied the aggregation of seven related light chain proteins: Two natural proteins from AL patients, two natural proteins from individuals without AL, and three designed mutations. Aggregation of the different light chain proteins was measured at multiple concentrations using thioflavin T, an amyloid-binding dye that is fluorescent when bound to amyloid but not when free in solution. The change in fluorescence was measured by spectroscopy. Raw data from the plate reader was imported into RStudio, which was used to convert the raw data into a format Amylofit could read. We then performed mechanistic analysis using Amylofit, an online platform for the processing and analysis of protein aggregation kinetic data. Processed data were further analyzed in RStudio.

Results and Conclusions: The different light chain variants had diverse relationships between concentration and aggregation rate. The two disease-associated light chains had strong negative slopes (a rapid decline in half time), which means that the more protein added, the faster the aggregation. However, the relationship between concentration and rate was curved: At high concentrations the rate of change was reduced. We interpret this to mean that as the concentrations became higher, dimerization occurred, which inhibited aggregation because the native state was being stabilized. The natural, non-disease-associated light chains and the three designed mutants had a smaller dependence of aggregation rate on concentration than the two disease associated light chains. This suggests that the disease associated proteins have more of a driving force for aggregation, and suggests there are perhaps other factors at play other than simply stability and concentration. Further studies are required to clarify what these additional mechanisms may be.

Princess Maryam Abdul-Akbar
Florida Atlantic University, Class of 2021

The Effect of Chronic Exposure to Excess Nutrients on Lipid Accumulation and Glucose-Dependent Calcium Oscillations in Clonal Pancreatic ß-cells (INS-1); ImageJ/Fiji Analysis

Princess Maryam Abdul-Akbar, Jude T. Deeney, PhD
Department of Medicine, Endocrinology, Diabetes, Nutrition and Weight Management, Boston University School of Medicine, Boston, MA

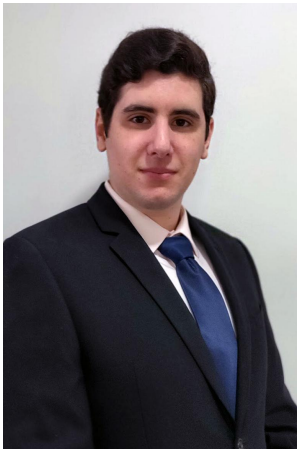
BACKGROUND/SIGNIFICANCE: Type 2 diabetes (T2D) is diagnosed when blood sugar levels remain persistently elevated due to insulin resistance or the inability to produce enough insulin. Most patients with T2D are overweight/obese. The global epidemic of obesity largely explains the dramatic increase in the incidence and prevalence of type 2 diabetes over the past 25 years. Hyperinsulinemia (HI) is elevated plasma insulin at basal glucose. The generally accepted view is that HI results from insulin resistance, although chronic excess nutrients have been shown to stimulate basal insulin secretion. Therefore, an alternative hypothesis is HI may be the cause rather than the consequence of the progression of insulin resistance and T2D. Chronic excess nutrient exposure results in lipid droplet accumulation and a left-shift in glucose-induced calcium oscillatory behavior leading to insulin secretion from the pancreatic ß-cell. Quantifying these changes will help to identify mechanisms regulating insulin secretion under conditions of normal and excess nutrients.

OBJECTIVE: Understanding the mechanisms by which excess nutrients stimulate HI and contribute to insulin resistance will help to identify potential targets that may prevent/delay the development of T2D.

METHODS: INS-1 cells were cultured in RPMI medium with 10% serum containing low and high glucose concentration (4 or 11 mM). Intracellular triglycerides containing lipid droplets are measured by Nile Red. Ca^{2+} was measured in INS-1 cells loaded with fura-2 AM (Invitrogen) (2 µm) for 30 min in the KREBS buffer. Fluorescence images were captured every 20s with wavelengths of 340/380 nm dual excitation and 510 nm emission. NIH ImageJ/Fiji was used to analyze the images by quantifying the fluorescence of the total intracellular lipid accumulation and calcium oscillation. Excel was used to further analyze the data.

RESULTS: ImageJ/Fiji analysis showed that lipid accumulation linearly correlates to glucose concentration in pancreatic ß-cells. It was also determined that the size of the lipid droplet was influenced by the glucose concentration as there was a trend for droplets from cells cultured in 8 mM glucose to be larger diameter compared to cells cultured in 6 mM glucose. An ImageJ/Fiji protocol for single cell calcium analysis was developed and needs further testing of its merits.

CONCLUSIONS: ImageJ/Fiji are important tools to identify and quantitate lipid droplets in pancreatic ß-cells exposed to excess nutrients. The use of ImageJ/Fiji will allow for analysis of all cells eliminating bias inherent in choosing cell zones required for use of other software packages. Evidence of the profound relationship between lipid stores, calcium handling and hypersecretion of insulin at submaximal glucose warrants further investigation and may provide insight into mechanisms leading to T2D.



Lazaro Fernandez
Florida International University, Class of 2021

The Fine Structure of Excitatory Synapses in the Aging Macaque: Effects of Dietary Curcumin

Lazaro Fernandez[1]; Carmen Freire, PhD[2]; Jennifer Luebke, PhD[1]; Maya Medalla, PhD[1];

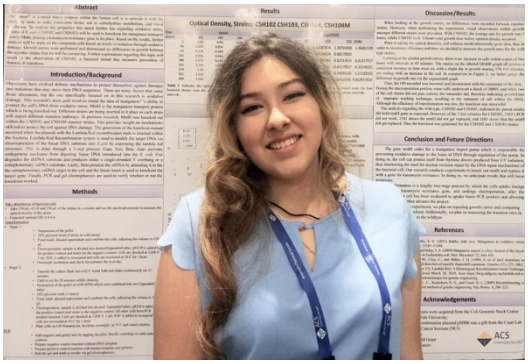
[1]Department of Anatomy & Neurobiology, Boston University School of Medicine, [2]Department of Neuroscience, Icahn School of Medicine at Mount Sinai

Background/ Objective: There is evidence that the cognitive effects of aging are related to the diminishing numerical density of spines and synapses in the lateral prefrontal cortex (LPFC) and that oxidative stress that occurs during normal aging may play a role in changing the morphology or number of synapses. We hypothesize oxidative stress in aging results in the loss or dystrophy of synapses and that by reducing oxidative stress during the aging process, synapses may be rescued. If this hypothesis is proven, anti-oxidant therapy may present the possibility of reducing the cognitive decline associated with aging. Curcumin is a well-documented anti-oxidant and anti-inflammatory compound that is known to protect against oxidative stress. There is evidence that curcumin is capable of relieving oxidative stress in other body systems, though there exists sparse literature documenting its effects in the aging brain.

Research Methods: To evaluate ultrastructural properties such as synaptic structures, a high resolution imaging technique must be used. Therefore, this study employed the use of serial block face electron microscopy to image samples of layers 1 and 3 of LPFC from middle-aged control and curcumin treated monkeys. Blocks of cortex from 4 behaviorally characterized monkeys of both sexes were processed for serial electron microscopy. This study focused primarily on the characterization of the 3D structure of excitatory “asymmetric” synapses (bouton, post synaptic densities [PSD], and spine) in layers 1 and 3 of the LPFC. Using Reconstruct™ software, asymmetric synapses on spines were identified and reconstructed via blind processing. This study has thus far performed this process on a total of 6 different samples. Differences between groups of samples were assessed statistically through a Student’s T-test.

Results: Samples from curcumin treated middle aged macaques when compared to samples without dietary curcumin were found to contain larger post-synaptic dendritic spines, PSD, and pre-synaptic mitochondria. When analyzed via a Student’s T-test, the surface areas and volumes of the dendritic spines and PSD areas were determined to be statistically significant between the control and the dietary curcumin. For pre-synaptic structures, boutons were not different in size, but their mitochondria showed significant difference in volume and surface area.

Conclusions: While there were significant effects of curcumin on several synaptic features, a larger sample size including a comparison with young monkeys is needed to determine if it alleviates age related changes.



Cynthia Flores
Mount Saint Mary's University, Los Angeles Class of 2021

Quantifications of Collagen Accumulation in the liver of Endothelial-Cell-specific Glutaredoxin Transgenic mice

Cynthia Flores, Yuko Tsukahara PhD, Reiko Matsui MD

Department of Vascular Biology (X-720), Boston University School of Medicine, Boston, MA

Background: Non-alcoholic fatty liver disease (NAFLD) is the most common liver disease found in the US, and may progress to more serious pathology, non-alcoholic steatohepatitis (NASH) with fibrosis. In a healthy liver, liver sinusoidal endothelial cells (LSECs) function to control fibrotic processes. In NASH liver however, LSECs may be activated and increase vascularity (angiogenesis), stimulating fibrosis. The lab found that overexpression of the enzyme redox regulator Glutaredoxin-1 (Glrx) in EC inhibited angiogenesis. Glrx is a ubiquitous cytosolic enzyme that regulates redox reactions by removing S-glutathionylation, or glutathione adducts at cysteine residues of proteins, in turn, modulating protein functions. Following studies where Glrx up-regulation prevented endothelial cell migration, we hypothesize EC-specific Glrx up-regulation inhibits angiogenesis and suppress liver fibrosis in diet-induced NASH model in mice.

Methods: EC-specific Glrx over-expressing mice (EC-GlrxTG) were created by introducing the VEcad-tTA; tetO-Glrx system (tet off system). EC-GlrxTG and control mice were fed a NASH diet consisting of high-fructose, high cholesterol, high fat diet for 15-20 weeks. Liver tissue samples were collected and fixed and a Picrosirius collagen Staining was performed. Quantitation of collagen area was done using Image J.

Results: We analyzed collagen areas in the liver of 15 EC-Glrx TC mice and 14 control mice for both females and male. Collagen area was lower in EC-GlrxTG female ($p<0.01$) mice as predicted, but higher in male ($p<0.05$) EC-GlrxTG compared to control mice.

Conclusion: The data suggests a successful induction of fibrosis by NASH diet. EC-Glrx TG inhibits liver fibrosis in females but the opposite in male mice, indicating sexual dimorphism EC Glrx function.

Ziko McLean
Boston University School of Medicine, Class of 2023

Assessing the relationship between increases in BMI percentile and the severity of Obstructive Sleep Apnea in children from 2015-2019

Ziko McLean; Jessica Levi MD

[1]Department of Otolaryngology, Boston University School of Medicine

Background/ Objective: Obstructive Sleep Apnea (OSA) is a disorder primarily of the upper airway, and is characterized by its narrowing and the consequent lack of oxygen during sleep. OSA has been linked with the development of Type 2 Diabetes Mellitus, hypertension, and Cardiovascular Disease, and its incidence has been rising, in part to the rise of obesity. A higher Body Mass Index (BMI) in adults has been found to increase the risk of OSA, as increased fat deposits in the upper respiratory tract cause the airways to narrow. This is supported by many studies that show that adults with the highest weight gain have the most severe scores on the Apnea-Hypopnea Index (AHI), a scale used to define the respiratory stress of OSA. Since 1980, obesity prevalence among children and adolescents has almost tripled, and childhood obesity is occurring at progressively younger ages, with OSA being documented to occur in about 60% of obese children. Furthermore, BMI is not as accurate with children. This research assesses the relationship between different BMI measures and OSA severity in children, especially as past research has showed different conclusions. The objective of this project is to ascertain the degree of association between different weight measures and OSA severity in children, along with how certain comorbidities impact this relationship.

Research Methods: For this retrospective chart review study, Children aged 2-18 years old who had OSA (diagnosed by polysomnogram) during 2015 to 2019 will be the focus of this research. The severity of OSA in the children across this time period will be recorded, with their BMI percentile to see if there is a linear correlation between the two. The expectation would be that those with a higher BMI percentile will have more severe OSA symptoms. Other factors such as asthma, race/ethnicity, gender and more will also be collected to see if the relationship between weight and severity holds true more for some populations than others. In order to evaluate which measure for weight would be the most appropriate for children, different measures will also be used. Body Mass Index Percentile, which has been deemed a more accurate way of examining weight in children, will be compared with BMI and % Weight for Height. Another measure, Tri-ponderal Mass Index (TMI), has recently been seen as a better measure for teenagers specifically. It will be compared to BMI Percentile, BMI, and % Weight for Height by solely examining their teenage populations.

Results: While data collection and analysis are still ongoing, the degree of association for each weight measure and OSA will be processed and compared. In addition, how closely each weight measure adheres to the weight of the children will be compared and determined. The statistical analysis will be completed with SPSS, the chi-squared technique, and regression, in order to see the degrees of association between these factors.

Conclusions: This research will shed more light on the relationship between weight and OSA severity in children, as well as help us to determine the best measure to use for assessing children's weight in studies.

Asel Mustafa
University of the Virgin Islands, Class of 2020
Boston University Graduate Medicine Sciences, Class of 2021

Patients With External Ear Abnormalities Should Have an Audiogram to Assess For Any Hearing loss

Asel Mustafa, Jennifer Shehan, MD, Jessica Levi, MD

[1]Department of Otolaryngology, Boston University and Boston Medical Center, Boston, MA, USA

BACKGROUND & SIGNIFICANCE: The external ear is an important component of sound conduction, and any abnormality in external ear development can also lead to middle and inner ear malformations. External ear abnormalities (EEA) are reported at 1: 6,000 newborns to 1: 6,830 newborns, and thus are prevalent in a variety of clinical settings. The American Academy of Audiology has guidelines on how physicians should manage cases with EEA, including referring those patients for audiological testing. This study aims to determine the prevalence and demographic features of pediatric patients with external ear abnormalities who were referred for audiological testing, their hearing status, treatment, and follow-up. This insight allows medical professionals to determine whether targeted reforms may be introduced to alleviate losing patients to follow up. Because this study also notes the amount of referred patients with EEA who were diagnosed with hearing loss, it also determines whether the current guidelines allow for the most efficient use of resources.

METHODS: This retrospective study identified 1,358 pediatric patients (≤18 years old) from Boston Medical Center, a tertiary safety-net hospital, who were diagnosed with external ear abnormalities between 2012 and 2020. Patients were identified using Internal Classification of Diseases (ICD) codes for EEAs. Once identified, their electronic medical records were then reviewed for demographic factors (age, sex, race, language) and clinical findings (diagnoses, relevant exam, newborn hearing test results, hearing abnormalities, audiology results, ENT procedures). Of the 598 cases analyzed so far, 379 cases were excluded for lack of relevant external ear abnormality. The results were then analyzed through univariate analysis with a chi-square test.

RESULTS: Of 219 cases (M=113, F= 106), a majority of the patients identified their race as black (black = 42%, declined = 30.6%, white = 10.5%, Hispanic = 9.6%, other = 7.3%). English was the primary language for a majority of the cases (English = 55.3%, Spanish = 25.6%, Haitian Creole = 8.8%, other = 10.3%). Audiology evaluated patients 42.4% of the time, and otolaryngology evaluated patients 64.8% of the time. Patients underwent a procedure for their diagnosis 32% of the time. There were 17.4% of patients who had some degree and type of hearing loss recorded. There were 23 patients or 10.5% who were considered lost to follow-up for not presenting as recommended additional otolaryngology evaluation. There were 50 patients or 22.8% who were considered partially lost to follow-up for presenting for evaluation once, but then not returning for additional evaluation. When patients did follow-up, on average they were seen in the otolaryngology clinic 4.12 ± 6.39 times.

CONCLUSIONS: On chi-square univariate analysis, there was no association between either sex or race and audiology, otolaryngology evaluation, or lost to follow-up status. There was a significant association between primary language and these measures. Reviewing the remaining cases and further evaluating these factors will allow researchers to understand how targeted reforms may benefit these patients and prevent loss to follow up.

Joseph Waiguru
Georgia State University, Class of 2021'

Exploring the lack of immune response to tumor cells in metastatic lymph nodes by looking at genetic expression

Joseph Waiguru, Dennis Jones, PhD

Department of PATHOLOGY & LABORATORY MEDICINE, Boston University School of medicine, Boston, MA

Background and significance: The lymphatic system is composed of a network of lymph vessels that transport fluid called lymph throughout the network and into lymph nodes (LNs). Cancer cells can also enter the lymphatic system and then travel to other organs in the body. In the lymph nodes, immune cells such as regulatory T cells (Tregs), natural killer cells (NK), T cells, B cells and dendritic cells (DCs) are tasked with the job of eliminating tumor cells. Tregs and DCs prime and activate T cells to eliminate the tumors in the LNs. An unanswered question is why these immune cells, specifically T cells, do not destroy tumor cells in LNs. We hypothesize that the tumor cells change the genetic expression of T cells and in doing so alter the functionality of T cells to promote tumor growth and metastasis. Most cancer deaths are due to the spread of cancer. Understanding why the immune cells do not attack the tumor cells can be crucial in treating cancer metastasis and in developing therapeutic treatments for cancer patients.

Methods: To test the hypothesis, Single cell RNA sequencing was performed from single cells suspensions of naïve and metastatic lymph nodes containing 4T1 mice breast cancer cells. Cells from naïve and metastatic lymph nodes were filtered and integrated using the Seurat package (version 3.1.5) in Rstudio. The naïve node had several low-quality cells and because of this it was combined with the metastatic node in Rstudio to compare the populations of cells. Clusters were also identified according to expression of marker genes, as referenced from the GeneCards database. Next, a literature review of the different cell types was performed to understand their functions in LNs.

Results: Graphs like Heatmaps and Umaps were produced and used to cluster the genes. In total, 18 different genetic clusters were present. 14 clusters were identified in the single cell sequencing data of naïve LN and 9 clusters in the metastatic LN. We identified dendritic cells (DCs), T cells, Natural killer cells (NKs), Regulatory T cells, and B cells. There were subsets of the different cell types in multiple clusters as there were a few different types for example there are CD4, CD8 T cells, cytotoxic T cells, and regulatory T cells.

Conclusion: In addition to identifying the genetic clusters, the clusters containing the T cells were also identified. This is important because in the long run it would allow for the comparison of the T cell cluster between naïve and metastatic LN. To support our hypothesis that the tumor cells are changing the genetic expression of the T cells we would expect to see a difference in the genetic expression between naïve and metastatic T cells. Moreover, understanding why the immune cells do not attack the tumor cells can be crucial in treating cancer metastasis and in developing therapeutic treatments for cancer patients. This is an important step towards more research on metastasis of cancer through the lymphatic system. Further and more in-depth research can provide insight into how we can control cancer metastasis.

STaRS PROGRAM ACKNOWLEDGEMENTS

We would like to acknowledge the outstanding contributions of the following professionals to the career development of our scholars:

FACULTY PRECEPTORS
Jude Deeney
Isabel Dominguez (2 scholars)
Hui Feng (2 scholars)
Christopher Heaphy (2 scholars)
Dennis Jones
Jessica Levi (3 scholars)
Laura Lowery
Jennifer Luebke
Reiko Matsui
Gareth Morgan
Karin Schon
Kei Suzuki
Deepa Gopal
Naomi Hamburg
Ludovic Trinquart

PROGRAM ADMINISTRATORS
Farrah Belizaire, STaRS program
Tinuola Ajayi, AHA-SURE program

BUSM STUDENT MENTORS
Carolyn Wilson, M.D. Class of 2023
Zijad McLean, M.D. Class of 2023
Devin Kenney, PhD student, Microbiology, Program in Biomedical Sciences (PiBS)
Jhon Henao, PhD student, Molecular and Translational Medicine, PiBS

STUDENT PANELISTS

Ph.D. students
Jhon Henao
Devin Kenney
Bukola Adeoye
Christine Nowa
Jean Royce
Stacy Jankowski
Elim Na
Renee DeVivo
Anna Smith
Katie Babcock
Sema Quadir
Karen Bottenfield
Katie Babcock
Margaret Downs
Kim Barker

M.D. students
Yoel Benarroch
Sojourna Ferguson
Danielle Lafond
Dhanesh Binda
Julio Mejias
Chioma Anyikwa
Roya Edalatpour
Victoria Rosales
Anthony Spinella
Anthony Yeung
Carly Merritt
Omar Mohtar

STaRS alumn
Luis Perez Valencia
Victor Bacelar
Annalyn Brown
Chelsey Skeete
Sojourna Ferguson

LECTURERS
David Flynn, MSc
Joe Farmer Ed.M
Gareth Morgan PhD

Suzanne Sarfaty, M.D.
Anna Bortnik
Matt Layne, PhD
Hui Feng, MD, PhD
Christopher Heaphy, PhD
Gwyneth Offner, PhD
Faetitia Desamours
Jamie McNight PhD
Gary Balady, MD
Lauren Celano, BS MBA
Mark Braun
Emelia J. Benjamin, MD ScM
Anita Distefano
Simone Gill, PhD
Jessica Fetterman, PhD
Lisa M. Sullivan, PhD
Katia Bulekova
Kefei Wu, BU SPH
Jennifer Luebke, PhD
Jonathan Wisco, PhD
Kirsten Goodell, MD
Sabreea Parnell, BUSM M3

PANELISTS

Rafael E. Luna, PhD	Boston College
Dennis Dean, PhD,	Principal Investigator, Seven Bridges
Natalie Cain, Ph.D	Scientific Editor, Cell Press
Jacobi Cunningham, PhD	Principal Scientist, Alkermes
Andrew Henderson PhD	BUSM

ADMINISTRATIVE STAFF

Millie Agosto	Registrar
Dennis Murphy	Associate Director, Student Financial Services
Yolanda Barros	Research Occupational Health Program
Solaris Bujor	Grants Administration
Victoria Kacprzak	Grants Administration
Israel De La Cruz	Financial Manager
Shannon Kelly	Ed Media
Liz Clark	Ed Media
David King	Ed Media
Maureen Demmert	EPIC training
Richard McGuire	EPIC training

THANK YOU ALL !