Poster Number: 1

Abstract Title: *Trpc6 Promotes Doxorubicin-Induced Cardiomyopathy in Male Mice With Pleiotropic Differences Between Males and Females*

Presenter: Katelyn Bruno (Research Faculty in the College of Medicine, Department/Division of Cardiovascular Medicine)

Additional Authors: Nadine Norton, Damian N Di Florio, Emily R Whelan, Anneliese R Hill, Andrea Carolina Morales-Lara, Anna A Mease, John M Sousou, Jose A Malavet, Lauren E Dorn, Gary R Salomon, Logan P Macomb, Sami Khatib, Zacharias P Anastasiadis, Brian M Necela, Molly M McGuire, Presley G Giresi, Archana Kotha, Danielle J Beetler, Raegan M Weil, Carolyn K Landolfo, DeLisa Fairweather

Abstract: Background: Doxorubicin is a widely used and effective chemotherapy, but the major limiting side effect is cardiomyopathy which in some patients leads to congestive heart failure. Genetic variants in TRPC6 have been associated with the development of doxorubicin-induced cardiotoxicity, suggesting that TRPC6 may be a therapeutic target for cardioprotection in cancer patients.

Methods: Assessment of Trpc6 deficiency to prevent doxorubicin-induced cardiac damage and function was conducted in male and female B6.129 and Trpc6 knock-out mice. Mice were treated with doxorubicin intraperitoneally every other day for a total of 6 injections (4 mg/kg/dose, cumulative dose 24 mg/kg). Cardiac damage was measured in heart sections by quantification of vacuolation and fibrosis, and in heart tissue by gene expression of Tnni3 and Myh7. Cardiac function was determined by echocardiography.

Results: When treated with doxorubicin, male Trpc6-deficient mice showed improvement in markers of cardiac damage with significantly reduced vacuolation, fibrosis and Myh7 expression and increased Tnni3 expression in the heart compared to wild-type controls. Similarly, male Trpc6-deficient mice treated with doxorubicin had improved LVEF, fractional shortening, cardiac output and stroke volume. Female mice were less susceptible to doxorubicin-induced cardiac damage and functional changes than males, but Trpc6-deficient females had improved vacuolation with doxorubicin treatment. Sex differences were observed in wild-type and Trpc6-deficient mice in body-weight and expression of Trpc1, Trpc3 and Rcan1 in response to doxorubicin.

Conclusions: Trpc6 promotes cardiac damage following treatment with doxorubicin resulting in cardiomyopathy in male mice. Female mice are less susceptible to cardiotoxicity with more robust ability to modulate other Trpc channels and Rcan1 expression.

Poster Number: 2

Abstract Title: *Integrative control of cardiac tissue biomechanical and biomolecular changes due to doxorubicin*

Presenter: Camara Casson (Graduate Student in the College of Engineering, Department/Division of Biomedical Engineering)

Additional Authors: Meghan C Ferrall-Fairbanks

Abstract: As chemotherapy becomes more effective, patients live longer allowing more long-term effects to emerge such as cardiac dysfunction. However, there exist knowledge gaps in how long-term effects result in changes to the tissues' biomechanical and biomolecular properties which can be assessed through an integrative approach. Therefore, an in vitro model system to determine changes in these properties in response to anthracycline exposure is needed. Towards this goal, tissue-engineered cardiac constructs will be used to create a baseline of cardiac tissue properties compared with known values in physiology and to assess the changes in biochemical and mechanical properties due to doxorubicin. This project will explore two parts: (1) development of a cardiac construct of the left ventricle and its response to anthracycline drugs and (2) determine the synergistic effects of the host environment on tissue degeneration of the cardiac tissue. This research will evaluate mechanical properties such as extracellular matrix density and tensile strength, and molecular properties like fibrinolytic and proteolytic activity. Data collected from proteolytic assays, RNA sequencing, and mechanical testing will be input into a data-driven model to determine if any of these biomolecular properties correlated with mechanical property changes due to doxorubicin. Further complexity to the system will be added with the use of human-induced pluripotent stem cells cardiomyocytes in an alginate matrix to maintain contractile properties and confirm the biomechanical and biomolecular trends observed in the first tissue construct. When these are successful, an established protocol for small-scale reconstructions of the left ventricle will be leveraged evaluating anthracycline effects on pumping efficiency. The proposed project provides a pathway to better understanding the physiological effects of anthracyclines on cardiac tissue.

Poster Number: 3

Abstract Title: *Racial and Ethnic Differences in Cardiac Surveillance Evaluation of Patients Treated with Anthracycline-Based Chemotherapy*

Presenter: David DeRemer (Clinical Faculty in the College of Pharmacy, Department/Division of Pharmacotherapy and Translational Research)

Additional Authors: Nam K Nguyen, Avirup Guha, Faraz S. Ahmad, Rhonda M. Cooper-DeHoff, Carl J Pepine, Michael G Fradley, Yan Gong

Abstract: Introduction: Anthracyclines remain a key treatment for many malignancies but can increase the risk of heart failure or cardiomyopathy. Specific guidelines recommend echocardiography (ECHO) and serum cardiac biomarkers such as B-type natriuretic peptide (BNP) or N-terminal pro-BNP (NT-proBNP) evaluation before and 6-12 months after treatment. Our objective was to evaluate associations between racial and ethnic groups in cardiac surveillance of cancer survivors after exposure to anthracyclines.

Methods: Adult patients in the OneFlorida Consortium without prior cardiovascular disease who received at least two cycles of anthracyclines were included in the analysis. Multivariable logistic regression was performed to estimate the odds ratios (OR) and 95% confidence intervals (CI) for receiving cardiac surveillance at baseline prior to anthracycline therapy, six months after (6M), and 12 months after (12M) anthracycline exposure among different racial and ethnic groups.

Results: Among the entire cohort of 5430 patients, 63.4% had a baseline ECHO, with 22.3% receiving an ECHO at 6M and 25% at 12M. Non-Hispanic Blacks (NHB) had a lower likelihood of receiving a baseline ECHO than Non-Hispanic Whites (NHW) (OR=0.75; [95% CI, 0.63-0.88]; p=0.0006) or any baseline cardiac surveillance (OR=0.76; [95% CI, 0.64-0.89]; p=0.001). Compared to NHW patients, Hispanic patients received significantly less cardiac surveillance at 6M (OR=0.84; [95% CI, 0.72-0.98]; p= 0.03) and 12M (OR=0.85; [95% CI 0.74-0.98]; p=0.03) timepoints, respectively.

Conclusions: There were significant racial and ethnic differences in cardiac surveillance among cancer survivors at baseline and following anthracycline-based treatment in NHB and Hispanic cohorts. Healthcare providers need to be cognizant of these social inequities and initiate efforts to ensure recommended cardiac surveillance occurs following anthracyclines.

Poster Number: 4

Abstract Title: *Candidate Genes for Survivorship with Coronary Heart Disease Include Cancer and Tumor Suppressor Genes*

Presenter: Jennifer Dungan (Research Faculty in the College of Nursing, Department/Division of Biobehavioral Nursing Science)

Additional Authors: Xue Qin, Simon Gregory, Rhonda Cooper-Dehoff, Julio Duarte, Huaizhen Qin, Carl Pepine, Elizabeth Hauser, William Kraus

Abstract: Introduction: In the search for genetic contribution to survivorship outcomes with coronary heart disease (CHD), our group has found top candidate genes having biologic relevance to cancer. Others have reported evidence of antagonistic pleiotropy (risk/protection trade-off effects) between CHD and cancer. However, scientific gaps remain around the genetic underpinnings for cardiac-cancer pleiotropy. The purpose of this abstract is to describe our cardio-oncology related candidate genes with potential for future hypothesis-building.

Methods: In our recent published works, we have used the following methodological approaches to identify common allelic variation associated with longitudinal, 10+ year survival outcomes among people with clinically-diagnosed CHD: a priori candidate gene (Dungan, et al., 2016), genome-wide association (GWAS; Dungan et al., 2021), and sex-stratified GWAS (Dungan et al., 2022). Our results for top hits included replications and meta-analyses.

Results: The cancer-related candidate genes and their subsequent survival effects among people with CHD are: LSAMP (tumor suppressor gene associated with epithelial ovarian cancer) showed allelic heterogeneity of survival effects; DAB2IP (ras/GAP tumor suppressor gene associated with resistance to prostate cancer), showed improved survival; TSSC1 (implicated in breast cancer) conferred increased risk of mortality; BRMS1L and MANCR (both associated with breast cancer) having male-associated mortality risk (BRMS1L) and female-associated mortality risk (MANCR); and finally, SLC9A9 (colorectal cancer), PRKD1 (malignant epithelial salivary tumors) and VTCN1 (t-cell regulated tumor progression), all demonstrating female-associated increased mortality risk.

Conclusions: Understanding genomic underpinnings of the complex relationships between CHD and cancer may provide clinical insights for future precision health screening or therapeutic targets.

Poster Number: 5

Abstract Title: *The Effects of Moderate Intensity and High-intensity Interval Training on Cardiorespiratory Capacity and Body Composition During Doxorubicin Treatment*

Presenter: Branden Nguyen (Graduate Student in the College of Health and Human Performance, Department/Division of Applied Physiology and Kinesiology)

Additional Authors: Dryden R. Baumfalk, Imtiaz M. Dowllah, Franccesco P. Boeno, and Ashley J. Smuder

Abstract: Doxorubicin (DOX) is a highly effect chemotherapeutic agent used to treat a variety of solid tumor malignancies including breast cancer. However, following DOX treatment breast cancer patients often suffer from reduced cardiorespiratory capacity and weight gain with increased body fat. These adverse changes following DOX treatment increase the risk of developing cardiovascular disease, thus, increasing patient mortality. In this regard, exercise has been shown to be effective in improving cardiorespiratory capacity and body composition. Therefore, using a clinically translational rodent model of DOX treatment we determined whether participation in moderate intensity aerobic training or high intensity interval training (HIIT) during treatment is sufficient to prevent these adverse effects. The DOX dosing protocol was administered in adult female Sprague-Dawley rats following a standard clinical treatment regimen (i.e., intravenous infusion of DOX once every 3 weeks for 4 total cycles, cumulative dose of ~160 mg/m2). Rats prescribed exercise training followed current exercise guidelines for cancer patients. Moderate exercise consisted of treadmill running at ~70% VO2max 1 hour/day 3x/week. HIIT consisted of 4x4 minute bouts of running at ~90% VO2max 3x/week. Dependent measures were assessed prior to the initiation of DOX treatment and one-week following the last cycle. Similar to patients, DOX reduced cardiorespiratory capacity and increased fat mass in sedentary rats. However, moderate intensity exercise and HIIT improved exercise tolerance, reduced fat mass and increased lean mass in DOX treated rats. These findings mimic the clinical effects of DOX on cardiorespiratory capacity and body composition in breast cancer patients and demonstrate that exercise training during treatment can reduce these negative effects of DOX chemotherapy.

Poster Number: 6

Abstract Title: *Effects of uncertainty in left ventricular border delineation on global longitudinal strain and versus LV jection fraction calculations.*

Presenter: Walter O'Dell (Research Faculty in the College of Medicine, Department/Division of Radiation Oncology)

Additional Authors: Abraham S, Lucas D, Siva Kumar S, O'Dell W

Abstract: BACKGROUND: There are many clinical scenarios in which early detection of small changes in cardiac function can be critical in determining a patient's treatment. Two of the most common imaging-based metrics in the clinic are left ventricular ejection fraction (LVEF) and border-length-based global longitudinal strain (GLS_BL). The aim of this project was to analyze the inherent biases and uncertainties in GLS_BL relative to LVEF with respect to uncertainties in delineation of the LV endocardial border.

METHODS: In silico 3D human LV endocardial surface models were created at end-diastole and end-systole based on MRIs of a healthy human volunteer. Twelve virtual long-axis imaging slices (radially-prescribed) were projected through the surface models to generate noise-free endocardial contour points. To simulate border delineation uncertainty, the set of endocardial contour points was subjected to inward and outward shifts of 1-5 mm, incrementally (Fig 1A). The expected uncertainty when using EC is around 3 mm. The % error vs. offsets were calculated for bi-plane GLS_BL, bi-plane area-length LVEF_AL, and bi-plane method-of-disks LVEF_MOD, with 3D conformal surface-fit LVEF_SURF as ground-truth.

RESULTS: The GLS_BL %error vs. offset was linear (R2 = 1.000) with a slope of 4.92 (Fig. 1B). Each of the LVEF measures were quadratic with offset (R2 = 1.000), with slopes: LVEF_AL: 6.68; LVEF_MOD: 6.71; and LVEF_SURF: 10.26.

CONCLUSIONS: GLS_BL is less sensitive to boundary delineation uncertainty than LVEF, due to the linear vs. quadratic dependence on offset. LVEF_AL and LVEF_MOD consistently underestimate true LVEF, making them less sensitive to boundary uncertainty. However, if border uncertainty is negligible, then LVEF is more sensitive than GLS_BL to small changes in LV volume, and LVEF_SURF is more sensitive than either LVEF_AL and LVEF_MOD.

Poster Number: 7

Abstract Title: *Detection of radiation-induced defects in heart perfusion in breast cancer patients*

Presenter: Walter O'Dell (Research Faculty in the College of Medicine, Department/Division of Radiation Oncology)

Additional Authors: Zhang V, Shephard A,

Abstract: Introduction

Today, it is estimated that more than 50% of patients with cancer are treated with radiotherapy, and among them, breast cancer patients constitute the largest population of patients exposed to chest radiation. As the radiation dose to the irradiated chest wall is frequently practiced and is thought to be highly predictive, the ability to quantify regional wall mechanical function and perfusion becomes crucial in regards to understanding of radiobiological response of the heart. Our hypothesis is that radiation to the heart wall causes decreased regional perfusion which is measurable and will predict regional mechanical dysfunction. Moreover, the decrease in perfusion will correlate with the amount of regional radiation dose given during treatment.

Methods

Heart MRI/CMRI images were acquired in breast cancer patients both before treatment and then 6-9 months after treatment for comparison purposes. The MRI examination included a perfusion scan where 3 slices locations through the top, middle and bottom of the left ventricle were chosen. In the study, a contrast agent was injected into the patient's arm vein for CMRI acquisition to be conducted once the injection starts; one image was acquired per heart beat with measurements acquired over 50 consecutive heartbeats. To process and analyze the images, custom software created by Dr. O'Dell and built upon the NIH ImageJ platform was used.

Results and Discussion

The qualitative preliminary results in one patient suggested that there is a decline in regional wall perfusion over time and that correlates with regional wall radiation exposure. Additional patient datasets are being analyzed and the software improved to provide more quantitative results with statistical support.

Poster Number: 8

Abstract Title: *Proton therapy preserves acute left ventricular ejection fraction relative to conventional X-ray therapy in breast cancer*

Presenter: Walter O'Dell (Research Faculty in the College of Medicine, Department/Division of Radiation Oncology)

Additional Authors: Siva Kumar S, Bradley J, Terracino B, Waler A, Zeng E, Klassen C, Rutenberg M, Mendenhall NP, Mailhot R, O'Dell W

Abstract: BACKGROUND

Radiotherapy (RT) yields a survival benefit for breast cancer patients but can result in harmful radiation exposure to the heart. Proton therapy (PT) can reduce the heart dose relative to photon RT, but a clinical benefit in terms of decreased cardiac toxicity has not yet been quantified. The study hypotheses are: (1) pre-symptomatic decline in global left-ventricular (LV) function can be quantified using detailed analysis of cardiac magnetic resonance images (CMRI); 2) the severity of cardiac dysfunction scales with heart dose; and (3) cardiac function is better maintained in patients treated with PT than with photon RT.

METHODS

Under an IRB-approved study, CMRI datasets were acquired before RT (but after chemotherapy) and at 6-13 months post-RT in 9 left-sided breast cancer patients: 4 treated with photons and 5 with PT. The LV endocardial border from end-diastole to end-systole was segmented in multiple image slices and views by 3 independent readers blinded to the treatment modality. Conformal 3D models of the LV geometry provided precise LV ejection fraction (LVEF) estimates. Mean heart dose, age, time to follow-up, and type of chemotherapy were also tabulated.

RESULTS

There was a moderate difference ($p = 0.044$) in mean LVEF before therapy between the PT (51.96 ± 2.0%) and photon (57.89 ± 2.0%) cohorts, but no significant difference in age or follow-up duration. At 6-13 months post-RT, LVEF improved for all patients receiving PT (+8.5 ± 1.6%) but decreased for all patients receiving photons (-11.0% ± 2.4%) with $p < 0.00001$. The mean heart dose for the PT cohort (0.24 +/- 0.25 Gy RBE) was significantly lower than the photon cohort (2.92 +/- 1.76 Gy). Mean heart dose negatively correlated with LVEF change ($r = -0.87$, $p<0.003$, for 7 degrees of freedom,) with a slope of -5% LVEF/Gy.

Poster Number: 9

Abstract Title: *Dexrazoxane and all-cause mortality in anthracycline-treated cancer patients.*

Presenter: Nathalie Roumi (PharmD Student in the College of Pharmacy, Department/Division of Pharmacotherapy and Translational Research)

Additional Authors: Yan Gong, Roy Williams

Abstract: Dexrazoxane is a potent intracellular chelating agent that interferes with iron mediated oxygen free radical produced by anthracyclines. It is recommended for primary prevention in anthracycline-treated patients with high cardiovascular toxicity risk. We assessed the benefit of dexrazoxane on all-cause mortality in patients receiving anthracycline treatment. This retrospective cohort study assessed overall survival in 6,193 cancer patients receiving anthracycline treatment with or without dexrazoxane using OneFlorida data from January 1st, 2012 to April 30th, 2020. Kaplan Meier analysis and Cox proportional hazard regression analysis were performed to estimate the hazard ratios (HR) and 95% confidence intervals. We also analyzed the overall survival of 5,715 patients with ICD codes for breast cancer (n=1840), lymphoma (n=1,961), leukemia (n=949), and sarcoma (n=965). . In the overall analysis, the mortality rate was significantly lower in patients treated with dexrazoxane + anthracyclines (26%) compared to those treated with anthracyclines alone (29%) (unadjusted HR 0.86 (0.74 – 0.996), p=0.044). However, this difference was no longer significant after adjusting for age, sex and race/ethnicity (adjusted HR, 0.89 (0.76-1.03); p=0.12). In the analyses of specific cancer types, lymphoma patients treated dexrazoxane had higher mortality rate compared to those treated with anthracycline alone (HR: 1.76, 1.12-2.51, p=0.01), likely due to the more advanced cancer stages of patients treated with dexrazoxane. In this retrospective analysis of cancer patients treated with anthracyclines in real-world clinical settings, we did not find evidence for survival benefit of dexrazoxane.

Poster Number: 10

Abstract Title: *Proteomic Analysis of Carfilzomib (CFZ) Related Heart Failure in Multiple Myeloma (MM) Patients from Prospective Study of Cardiac Events During Proteasome Inhibitor Therapy (PROTECT) Study*

Presenter: Samia Shabnaz (Graduate Student in the College of Pharmacy, Department/Division of Pharmacotherapy and Translational Research)

Additional Authors: Roy Williams, Samuel M. Rubinstein, Michael G. Fradley, Rachid C. Baz, Carl J. Pepine, Daniel Lenihan, R. Frank. Cornell, Yan Gong

Abstract: Introduction: Carfilzomib is effectively used in multiple myeloma (MM) patients, but heart failure (HF) occurs in 7.2- 22.7% of patients. Our previous metabolomics study discovered that patients with cardiovascular disease (including HF) had lower levels of TUDCA than no cardiovascular disease. TUDCA can prevent cardiomyocyte contractility by activating the PI3K/Akt/eNOS axis. This study aims to identify protein biomarkers that can differentiate patients at high risk of HF.

Methods: OLINK proteomics analysis was performed on the baseline (BL) and post-treatment (PT) plasma samples of 28 MM patients from PROTECT study including 14 HF patients and 14 age-, sex-matched no HF patients. t test was performed to identify proteins that are differentially expressed in HF vs no HF patients. The proteins with nominal significance $p<0.05$ were included in LASSO Cox regression.

Results: Overall, 27 proteins were differentially expressed between the two groups at BL or PT. The baseline proteins with lower intensity in the HF patients compared to the no-HF patients showed a significant difference between the two groups such as β-NGF (Beta Nerve Growth Factor) (FC= 0.77, p = 0.00077), HB EGF (Heparin-binding epidermal growth factor) (FC= 0.86, p = 0.0074). β-NGF was the only protein in the LASSO Cox regression model with a C-index of 0.83. PT proteins also significantly differentiated between the patients with HF and without HF eg β-NGF (FC= 0.74, p = 0.0015), HB EGF (FC= 0.85, p = 0.0080). β-NGF has a cardio-protective effect by activating the PI3K/Akt/NOS axis. HB EGF can decrease iNOS expression, increase NO production, and protect epithelial cells from apoptosis.

Conclusions: Our study on metabolomics and proteomics identified biomarkers with different levels in HF vs no HF patients that suggest the importance of the PI3K/Akt/eNOS pathway, NO production in carfilzomib-related HF.

Poster Number: 11

Abstract Title: *TMSB10/TRABD2A Locus associated with carfilzomib related cardiotoxicity in patients with multiple myeloma: a whole-exome sequencing analysis*

Presenter: Marwa Tantawy (Postdoc/Fellow in the College of Pharmacy, Department/Division of Pharmacotherapy and Translational Research)

Additional Authors:

Abstract: Background: Multiple Myeloma (MM) is the second most frequent hematologic cancer in the United States. Carfilzomib (CFZ), an irreversible proteasome inhibitor used to treat relapsed and refractory MM, has been associated with cardiovascular adverse events (CVAEs), including cardiomyopathy and heart failure (HF). Our study aimed to determine genetic variants associated with CVAE among patients with MM treated with CFZ.

Methods: We performed an exome-wide association analysis and gene-based analysis on the whole-exome sequencing data of 603,920 variants in 219 patients with MM of European ancestry, including 34 CVAEs, from the Oncology Research Information Exchange Network (ORIEN). As a replication, we genotyped the variants (with $p< 10\text{-}4$) in the 51 patients with MM of European ancestry in the Prospective Observation of Cardiac Safety with Proteasome Inhibitor (PROTECT) study using Taqman SNP assays. Multivariable logistic regressions were performed to estimate the odds ratios (OR) and 95% confidence intervals (CI) adjusting for age, sex and principal components for ancestry, followed by a meta-analysis of the two studies.

Results: A missense variant rs7148 in TMSB10 (Thymosin Beta 10) gene, also an eQTL for TRABD2A, was associated with CVAE with OR of 10.92 (4.4-26.9) ($p = 2.04\text{x}10\text{-}7$). This SNP has OR of 1.97 (0.41- 9.59) ($p = 0.39$) in the PROTECT study, with the meta-analysis p of $2.08\text{x}10\text{-}7$. The gene-based analysis also yielded TRABD2A as a significant gene ($p = 1.06\text{x}10\text{-}6$). TMSB10 is a ubiquitous protein and member of the β-thymosin family and has been previously reported to be dysregulated in dilated cardiomyopathy. TRABD2A was previously associated with troponin elevation and HF.

Conclusion: In this study, we identified TMSB10/TRABD2A locus to be associated with CFZ-CVAEs. Further investigation is ongoing to validate these findings.

Poster Number: 12

Abstract Title: *MiRNA-125a association with Carfilzomib-Related Cardiovascular Adverse events in Multiple Myeloma Patients: Prospective Observation of Cardiac Safety with Proteasome Inhibitor (PROTECT) study*

Presenter: Marwa Tantawy (Postdoc/Fellow in the College of Pharmacy, Department/Division of Pharmacotherapy and Translational Research)

Additional Authors:

Abstract: Background: Carfilzomib (CFZ) is a proteasome inhibitor used in Multiple Myeloma (MM) patients and is associated with cardiovascular adverse events (CVAE). microRNAs (miRNAs) regulate gene expression at the posttranscriptional level through binding to target mRNA. Our study aimed to identify circulating miRNA in plasma as a potential biomarker and explore the CFZ-related CVAE (CFZ-CVAE) mechanism.

Methods: Circulating miRNAs were profiled in the baseline and post-treatment (Six- month visit for non-CVAE patients, and at the CVAE visit for CVAE patients) in plasma samples of MM patients from the PROTECT study using TaqMan Open-Array Human MicroRNA panels (~750 of miRNAs). We performed logistic regression adjusted for age, gender, Brain-natriuretic peptide (BNP), and ancestry, for baseline and after treatment.

Results: A total of 60 patients (31 patients with CFZ-CVAE) were included in this study. The Relative expression level of miR-125a was significantly higher in the CVAE patients than in the non-CVAE patients at baseline. The odds ratio (OR) and 95% confidence interval (CI) were 1.25 (1.05-1.48) (P = 0.014, fold-change [FC] =12.9). However, this miRNA was not significantly changed in post-treatment patients, the OR and 95% CI were 1.12 (0.96-1.32) (P=0.15, FC= 3.87). The changes in the expression of miR-125a before and after treatment was also not significantly different between CVAE and non-CVAE patients (P=0.55). It was reported that this miRNA is upregulated in the plasma of humans, and cardiac cells in rats with heart failure and acute ischemic stroke.

Conclusion: Elevated circulating miR-125a at baseline may be a biomarker for CFZ-CVAE. Further study with a larger sample size is warranted.

Poster Number: 13

Abstract Title: *Planning, protocol, expected outcomes for a communal coping intervention to support DASH adherence among African American colorectal cancer survivors*

Presenter: Melissa Vilaro (Research Faculty in the College of Agricultural and Life Sciences, Department/Division of Family, Youth, and Community Sciences)

Additional Authors: Jeanette Andrade, Fidela Gjnondreka, Kathryn Hitchcock, Juan Vilaro, Janice Krieger

Abstract: Introduction: Cardio-oncology is a subspecialty of cardiology that addresses shared risk factors between cancer and cardiovascular disease (CVD) (e.g., obesity, inflammation). Disparities in cardio-oncology burden African Americans (AA), who experience low adherence to recommended CVD risk-reducing behaviors and overall worse CVD outcomes. Communal coping (a process of cooperative problem-solving framing illness as a joint vs. individual issue) may have important implications for CVD risk reduction behaviors (e.g., dietary and medication adherence) yet few interventions exist that leverage communal coping to support adherence.

Methods: This hypothesis-generating, pilot study uses a cross-sectional design to achieve two specific aims: 1) Assess communal coping and adherence strategies for CVD risk reduction with two focus groups with AA cancer survivors (n=5) and their social unit members (n=5) and conduct individual interviews with oncologists (n=5) and cardiologists (n = 5). Aim 2, asks social unit dyads to prepare a recipe (food box provided by researchers) during a single audio/video-recorded meal preparation task where salivary biomarkers of inflammation, blood pressure, FFQ, and self-reported diet/medication adherence are collected. Transcripts will be analyzed for illness appraisals ("our" vs. "your" problem), illness actions (shared vs. individual problem solving), and linguistic features of communal coping ("I" vs. "we" ratios) using LIWC-22.

Results: Feasibility of recruiting, enrolling, and retaining African American colorectal cancer survivors and social unit members, acceptability of design, and relationships between communal coping (LIWC-22) and adherence. recommendations will yield insights to inform a preliminary codebook, sample content, and key variables.

Conclusions: Long-term we aim to prevent CVD among at-risk groups using community-engaged strategies and inform key variables for a larger study.