# TABLE OF CONTENTS

Asante, Affie, BS (Poster 1) ................................................................. 2
Asim, Tariq H. (Poster 2) ................................................................. 2
Berry, Kaala S., BS (Poster 3) ........................................................... 3
Blair, Isabella A., BS (Poster 4) ....................................................... 3
Bowens, Gyasi, BS (Poster 5) .......................................................... 4
Brock, Briana A., BS (Poster 6) .......................................................... 4
Chevrin, Jamirah Y., BS (Poster 7) .................................................. 5
Condoll, TaMiko A., BS (Poster 8) .................................................... 5
Cross, Michael A. (Poster 9) ........................................................... 6
Daniels, Tia N. (Poster 10) ............................................................... 6
Dominguez, Santana E. (Poster 11) .................................................... 7
Dwarampudi, Sindhu (Poster 12) ....................................................... 7
Earvin, Jalyn A. (Poster 13) ............................................................. 8
Eltoum, Mustafa I. (Poster 14) ........................................................ 8
Feng, Bryan D. (Poster 15) .............................................................. 9
Fouche, Iman R. (Poster 16) ............................................................ 9
Gerrick, Rebekah N., BS (Poster 17) ............................................... 10
Giles, Emily (Poster 18) ................................................................. 10
Hollins, Zaria, MPH (Poster 19) ....................................................... 11
House, Prada C., MPH (Poster 20) .................................................. 11
Howell, Evan N. (Poster 21) ........................................................... 12
Hughes, Kamal, BS (Poster 22) ...................................................... 12
Hurley, Eric A. (Poster 23) ............................................................. 13
Isaac, Brittany, BS, MS (Poster 24) ............................................... 13
Jackson, Harrison P. (Poster 25) ..................................................... 14
Levy, Rusheka R. (Poster 26) ......................................................... 14
Mahaffey, Carolina, BS (Poster 27) ................................................ 15
Mansur, Ramy, (Poster 28) ............................................................. 15
Metcalf, Nyla (Poster 29) ............................................................... 16
Mwazembe, Irene, BSN (Poster 30) ................................................ 16
Nagaraj, Nayana H. (Poster 31) ....................................................... 17
Naveen, Nabaa, BS (Poster 32) ....................................................... 17
Nixon, Rachel D., BS (Poster 33) .................................................... 18
Norfleet, Vonneta D., BA (Poster 34) ............................................ 18
Notice, LilyJasmine (Poster 35) ....................................................... 19
Rao, Kirthana, MS (Poster 36) ........................................................ 19
SAZIB, Sazaul Morshed, BS (Poster 37) ......................................... 20
Siddique, Md Abu Talha T., BS (Poster 38) ....................................... 20
Vadranam, Meghana, BS (Poster 39) ............................................. 21
Williams, Caleah, None (Poster 40) .............................................. 21
Williams, Grace, None (Poster 41) ................................................ 22
Willis, Shannon D. (Poster 42) ........................................................ 22
Introduction As of today, Triple-Negative Breast Cancer (TNBC) is one of the four deadliest cancers with the lowest survival rates. From the years 2012 to 2016, TNBC was responsible for about 12% of all breast cancer diagnoses and African Americans had higher mortality rates from this disease than their white counterparts. One common denominator is the endocrine disrupting chemicals (EDCs) present in hormone containing personal care products more commonly used by African American women (AA). However, little research has been done on chemical hair relaxers and their effect on AA. This literature review will examine the association of EDC containing hair products and TNBC among African American women.

Methods This paper provides a narrative overview on chemical hair relaxers that are predominately performed on AA hair by focusing on three main EDCs: estrogens, phthalates, and parabens. The literature reviews utilized the following keywords: Triple-Negative Breast Cancer, EDC, African Americans, hair products, chemical relaxers. The search parameters included articles published between the years 2014 and 2022.

Results Past papers have shown that chemical hair relaxers were associated with estrogen receptor negative disease and increased early onset breast cancer in AA. It was found that incidence rates for breast cancer were higher among AA under the age of 45. EDCs were said to mimic the effects of estrogen and affect endocrine functionality. EDCs contribute to abnormal sexual development and cause younger women to experience higher incidence of breast cancer. This acceleration only regressed after hormonally active hair product use was discontinued.

Discussion We found that there was an association between EDC containing hair products and breast cancer risk among AA, but not enough research is known to conclude its direct association to TNBC. The hair products focused on were chemical relaxers, hair dyes, and cholesterol or placenta-containing conditioners. EDCs increase risk by mimicking the effects of estrogen and changing the rate of mammary gland development.

Conclusion These results demonstrate that there is an association between EDC containing hair products and triple negative breast cancer among African American women.

Introduction: Colorectal cancer (CRC) is the third mostly commonly diagnosed cancer among Black men and women and is third as it relates to cancer-related deaths among Black men and women residing in the United States (U.S.). It is estimated there will be about 20,000 new cases of CRC and about 7,200 deaths from CRC among Blacks in 2022. Men are at a significantly higher risk for these cancers than women. CRC incidence is about 20% in Black people than in White people among both men and women. Comparatively, CRC mortality rates are 44% higher in Black men and 31% higher in Black women compared to Whites. Studies have postulated various factors associated with this disparate outcome, such as access to care, unhealthy diet, insufficient physical activity, high alcohol consumption and smoking. The purpose of this literature review is to examine the role and impact of red and processed meat consumption among Blacks and its association with CRC risk.

Methods: A literature search was performed, using the biomedical databases: PubMed. The parameters of the search included articles that were published between March 2012-March 2022. The search included key words such as “red meat” “processed meat” “colorectal cancer” “Blacks” “cancer” and “cancer disparities”.

Results: Preliminary findings suggest red and processed meats contribute to an increased risk of CRC. The World Cancer Research Fund found the risk of CRC is increased by 18% for every 50 grams/day of processed meat and 12% for every 100 grams/day of red meat. Both red and processed meats are classified as carcinogenic to humans according to the International Agency for Research on Cancer. We will present our comprehensive findings upon completion of the data abstraction process.

Discussion/ Conclusion: CRC disproportionately impacts Blacks when compared to men and women of other racial and ethnic groups. This is largely a result of the “lifestyle choices” that members of this community are forced to take part in. We will present evidenced based, culturally appropriate interventions to address the consumption of red and/or processed meats among Blacks with the goal of advancing cancer health equity.
Impact of Therapeutics on Cardiovascular Health and Breast Cancer Outcome

Berry, Kaala S., BS; Mir, Hina, PhD; Singh, Shailesh, PhD

**Introduction:** 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 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 cardioprotective 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 and Discussion:** Chemotherapy, radiation therapy, hormone ablative 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 outcome.

**Conclusion:** 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.

Development of cost of care (CoC) interventions to decrease financial distress in cancer patients

Blair, Isabella A., BS; Liang, Margaret, MD, MS; Pisu, Maria, PhD

**Introduction:** Financial toxicity affects approximately 50% of gynecologic cancer patients. It can result from out-of-pocket costs and reduced income-earning potential of cancer patients, which can lead to psychological distress or health-related coping behaviors. Few evidence-based interventions are available to decrease financial distress. Our objective is to develop a proactive Cost of Care (CoC) intervention, which includes providing cost information (education about insurance and employment) with a cancer-specific cost estimate or a cost tracker.

**Methods:** We used publicly available patient education resources and prior pilot study data to develop the proactive CoC materials along with an intervention delivery script. We will review the proactive CoC materials in two focus groups of stakeholders to ensure that the materials are patient friendly and relevant. The first group will include three cancer patients/survivors and two caregivers. The second group will include health care team members, including a social worker, financial counselor, nurse, patient navigator, and two oncologists. The focus groups will assess the actionability and understandability of the proactive CoC intervention materials. We’ve prepared questions to guide the focus group sessions and a questionnaire for stakeholders to evaluate the insurance/employment information.

**Results:** The stakeholder group feedback will be used to refine the CoC intervention materials and intervention delivery.

**Discussion/Conclusion:** We plan to conduct a pilot randomized controlled trial of cancer patients to measure the impact of using the proactive CoC intervention materials that we have developed on financial distress, measured by the Comprehensive Score for Financial Toxicity (COST).
**Introduction.** Breast cancer is the most diagnosed cancer among Black women, with an estimated 36,000 new cases expected to be diagnosed in 2022, and the leading cause of cancer death with an estimated 6,800 deaths expected in 2022. Interestingly, Black women are more than twice as likely as women of other racial and ethnic groups in the United States (U.S.) to be diagnosed with triple negative breast cancer (TNBC). Clinically, TNBC refers to tumors that lack estrogen receptors, progesterone receptors, and human epidermal growth factor receptor-2. Studies suggest, women diagnosed with TNBC experience poorer outcomes due to the lack of effective treatments. The purpose of this literature review is to examine through a multifactorial model, the factors associated with disparities in breast cancer and better understand interventions that may help with these disparities, reducing the prevalence and mortality rate among black women and TNBC.


**Results.** We are currently synthesizing literature. Data is being abstracted to examine through a multifactorial model, the factors associated with disparities in breast cancer and better understand interventions that may help with these disparities, reducing the prevalence and mortality rate among black women and TNBC. We will present the most common themes identified for the various factors attributable to the disparate outcomes among Black women at risk, diagnosed, and/or treated for breast cancer.

**Conclusion/ Discussion:** Black women continue to be disproportionally impacted by breast cancer and the evidence is clear. With an increased focus on subtypes of breast cancer among populations at increased risk, such as Black women, we expect to present evidence-based, multi-level intervention strategies to overcome these disparate outcomes.

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**Brock, Briana A., BS**

**Poster 6**

**Racial Disparities in Lung Cancer**

Briana Brock, Hina Mir, and Shailesh Singh

**Introduction:** 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.

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

**Results and Discussion:** 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 White. In women, the highest rates are in white 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:** 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.
Chevrin, Jamirah Y., BS

A Systematic Literature Review: Society, Genomics, and Black Cancer-related Outcomes and Mortality; Intrinsic Reversibility for Cancer Prevention
Chevrin, Jamirah Y., BS

Introduction: Black persons residing in the United States have unique life experiences that may serve as an adverse trigger for toxic cellular stress 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. This systematic review provides a comprehensive evaluation of these associations, interventions, and prevention strategies.

Methods: This is a scoping review design. The “PubMed” database were searched for articles published through July 2022. Three research questions were proposed “Are allostatic load and systematic chronic inflammation more present or associated with increased risk to develop cancer or more aggressive cancer forms in Blacks in the United States?”, “What interventions are effective at preventing and reducing allostatic load and systematic chronic inflammation?” 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.

Discussion/Conclusion: 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.

Condoll, TaMiko A., BS

Transgender People Get Cancer Too!
Condoll, TaMiko A., BS

Introduction: Transgender people (TGP) in America face real and perceived disparities in healthcare leading to delayed cancer screening and treatment. Barriers include societal stigma and discrimination associated with higher levels of smoking and alcohol consumption. Fatalistic views about cancer, reluctance to disclose transgender status, scarcity of providers experienced in transgender healthcare, structural and financial barriers such as unemployment and the lack of health insurance further exacerbate disparities. As a result, many TGP are diagnosed in late stages of cancer, particularly lung cancer. Limited research on transgender oncology care has left an estimated 1.4 million transgender Americans underserved. Thus, the purpose of my research is to explore the impact trusted sources of health information may have on improving TGP’s cancer screening rates.

Methods: This study was guided by a review of the literature to understand who is included in the transgender population and TGP’s experiences with the healthcare system. Data from two quantitative studies were reviewed—the Health Information National Trend Survey (HINTS) 5, Cycle 1, 2017; and the 2016-2018 TransPop National Probability Study of TGP conducted by the Inter-university Consortium for Political and Social Research. My study asked the question, do trusted sources of health information affect cancer screening rates for TGP? Results: A weighted linear regression was used to explore the variables ‘health information-seeking behaviors’, ‘fatalistic views of cancer’, and ‘trusted health care information’ in the HINTS data set (117 lesbian, gay, bisexual (LGB) and 2857 heterosexual respondents). Descriptive statistics were used to explore similar variables in the TransPop data set (247 TGP and 1184 cisgender/heterosexual respondents).

Discussion/Conclusions: Much of the literature highlight the lack of national studies, limited number of studies overall, and small sample sizes as problematic in determining the level of disparities TGP experience and which population-specific interventions work best to improve TGP’s cancer health outcomes. Therefore, future research should include regional and national qualitative and quantitative studies to understand how TGP would like to receive healthcare information and what the actual numbers reveal about TGP’s access to prevention and treatment of cancer within the healthcare system. Key Words: transgender, transgender people (TGP), sexual minority, cisgender, heterosexual, lesbian, gay, bisexual (LGB), gender minority (GM), cancer, screening, treatment, access, health disparities
Cross, Michael A.

**Poster 9**

**Vaping Impairs Airway Surface Hydration and Cilia Beat Frequency**

Cross, Michael A.

Chronic Obstructive Pulmonary Disease (COPD) is the third leading cause of death in the U.S. It is mainly caused by long-term cigarette smoking. Smoking is known to impair mucociliary clearance (MCC), the primary defense of the lungs responsible for capturing and removing inhaled particulate matter and pathogens from the airways. MCC is regulated by ion transport by Cystic Fibrosis Transmembrane conductance Regulator (CFTR) channel that hydrates the Periciliary Liquid (PCL) layer on the airway surface. As observed in Cystic Fibrosis patients, inherited defects in CFTR result in the loss of airway surface hydration, MCC, and opportunistic infections. Our lab has previously shown that cigarette smoking reduces CFTR activity and impacts PCL depth and MCC by reducing Cilia Beat Frequency (CBF). However, the lung effects of using newer nicotine products such as e-cigarettes (e-cigs) called vaping are unknown. Thus, my research aims to test if e-cig aerosols affect airway MCC defense. I examined the trachea isolated from ferrets exposed to e-cig aerosols for 5 weeks. Ferrets are similar to humans in mucus gland distribution in the lungs, making them an excellent model for investigating respiratory pathology. Excised ferret tracheae were imaged with micro optical coherence tomography (μOCT) that simultaneously captures PCL depth, CBF, and mucus clearance, followed by CFTR activity measurement by Ussing Chamber electrophysiology. Data generated will help inform the health effects of vaping and the risk of developing lung diseases such as COPD.

Daniels, Tia N.

**Poster 10**

**Targeting TRIP13 With The Small Molecule Inhibitor DCZ0415 Reduces Growth Of Pancreatic Ductal Adenocarcinoma Through Induction Of An Immune Response**

Daniels, Tia, BS.; Afaq, Farrukh, PhD.; Bajpai, Prachi, PhD.; Kim, Hyung-Gyoon, PhD.; Diffalha, Sameer Al, MD.; Khushman, Mohammad, MD.; Bae, Sejong, PhD.; Varambally, Soory, PhD.; Manne, Upender, PhD

**Introduction:** Since pancreatic ductal adenocarcinoma (PDAC) is an aggressive malignancy and the fourth leading cause of cancer-associated mortality, there is a need to identify molecules as therapeutic targets. Thyroid receptor-interacting protein 13 (TRIP13), an enzyme of AAA-ATPase family, facilitates the assembly and degradation of protein complexes and participates in various biological functions, including the spindle assembly checkpoint and DNA repair pathways. Overexpression of TRIP13 has been associated with the progression and poor prognosis of other cancers, but its role in PDAC is unknown. Thus, we evaluated the role of TRIP13 in PDAC and investigated if targeting it with DCZ0415 had an inhibitory effect on tumor growth and immune modulation.

**Methods:** Immunocompetent mice (C57BL/6) were used to examine the effect of DCZ0415 on tumor growth and the immune response. KPC cells (0.1 x 10⁶), a syngeneic PDAC model, were subcutaneously injected into mice. When tumor volumes reached approximately 200 mm³, the mice were divided into two groups and treated with vehicle or with DCZ0415 (25 mg/kg body weight) intraperitoneally every alternate day for up to 3 weeks. Tumor size was measured at regular intervals, and tumor weights were determined at the time of sacrifice. In the KPC tumors treated with DCZ0415 or the vehicle, expression of TRIP13, cytotoxic mediators (perforin, granzyme B), PD1, and the infiltration of T cells (CD3 and CD4) were determined by western blotting and immunohistochemistry, respectively.

**Results:** DCZ0415 treatment reduced the growth of PDAC tumors (P<0.001) as compared with vehicle control mice. Western blot analyses revealed that DCZ0415 treatment induced an immune response by increasing expression of granzyme B and perforin and decreasing expression of PD1. DCZ0415 treatment facilitated immune cell infiltration as evident from elevated staining of CD3 and CD4 cells.

**Discussion/Conclusion:** These results demonstrate that treatment with the TRIP13 inhibitor, DCZ0415, reduces PDAC tumor growth by enhancing antitumor immunity through the infiltration of T cells, secretion of cytotoxic mediators, and inhibition of the PD1 immune checkpoint. Our findings show that DCZ0415 could be developed as an immunostimulatory agent to improve treatment for PDACs, particularly for those with high expression of TRIP13.
An examination of the consumption and influence of the ‘Western Pattern Diet’ (WPD) and associated risk for Breast Cancer among the black community.

An examination of the consumption and influence of the ‘Western Pattern Diet’ (WPD) and associated risk for Breast Cancer among the black community. Breast cancer is the most common cancer worldwide, contributing 12.5% of the total number of new cases diagnosed in the world and with 290,560 new cases expected in the United States in 2022. Black women especially are being affected with African American women having a 33% breast cancer mortality rate - the highest of any U.S. racial or ethnic group. In addition to breast cancer incidence being higher among African American women than White women younger than 45. Women are at significantly higher risk for breast cancer however about 1 out of every 100 breast cancers diagnosed in the United States is found in a man. Black women have an 8% LOWER cancer incidence rate compared with White women, yet, are 41% MORE LIKELY to die from breast cancer, despite these similar or lower incidence rates. Studies have posited several factors associated with the previous outcomes such as unhealthy diet, inadequate physical exercise, and denied access to care voluntarily and involuntarily. The purpose of this literature review is to examine the role and impact of the ‘Western Pattern Diet’ (WPD) among the black community and its association with Breast Cancer.

Methods: A literature search was performed, using the biomedical databases: PubMed. The parameters of the search included articles that were published between March 2012-March 2022. The search included key words such as “breast cancer”, “fried and processed foods”, “breast cancer disparities”, and “western pattern diet effects”. Results: Preliminary research and findings suggest that the “Western Pattern Diet” (WPD) also known as the Standard American Diet (SAD) consisting of high amounts of processed foods, high-fat dairy products, high-sugar foods, and reduced intake of plant-based fibers contribute to an increased risk of breast cancer. These processed foods with tremendous fat and calorie content result in one's body creating fat tissue which secretes estrogen. The American Cancer Society found that a woman’s risk of breast cancer is related to the estrogen and progesterone made by her own ovaries, so as a result of additional estrogen this hormone stimulates hormone receptor-positive breast cancers to grow causing breast tumors. We will present our comprehensive findings upon completion of the data abstraction process.

Discussion/Conclusion: Breast Cancer disproportionately impacts the black community but African American women especially when compared to men and women of other racial and ethnic groups. The results are mainly due to the Western Pattern Diet and limited food access besides this diet that are forced upon underserved black communities. We will present evidenced based, culturally appropriate interventions to address the consumption of the WPD among the black community with the goal of advancing cancer health equity.

**Dwarampudi, Sindhu**

The Role Of Traditional Healers In Cancer Care In Sub-Saharan Africa

Dwarampudi, Sindhu; Gutnik, Lily, MD MPH

**Introduction** Traditional healers play a significant role in the healthcare system in Sub-Saharan Africa, often serving as trustworthy sources of information and care in their local communities. Cancer incidence rates are high in the Sub-Saharan Africa region and mortality due to cancer is rising. There is limited information surrounding the role of traditional healers in cancer care. Given the prevalence of patients consulting these healers, it is necessary to precisely understand how they impact cancer care delivery in Sub-Saharan Africa.

**Methods** We are conducting a systematic review to obtain this relevant information. Inclusion criteria are a focus on the role/function of traditional healers on at least one aspect of the cancer continuum and inclusion of interviews or intervention with healers. Exclusion criteria are: lack of perspectives on the role of traditional healers, focus on herbal medicines, not about cancer, based in a non-Sub-Saharan Africa country, non-English language, not a primary research article, and unavailable full-text. For each study, data is extracted to include: country, aim of study and design, type of cancer treated, start and end dates, type and gender of healer, religion, highest level of education, source of training, and location of healer, inclusion and exclusion criteria, participant recruitment method, healer’s participation in cancer continuum and role in cancer care, and the healer’s interaction with the biomedical healthcare system. Results: 1187 titles and abstracts were screened, with 168 moving forward to full-text review. During full-text review, 125 were excluded and 43 studies were included for final data extraction. Currently, data extraction is ongoing.

**Discussion** While the data extraction phase is ongoing, we are finding that traditional healers are an important part of cancer delivery. Traditional medical practitioners contribute to the various parts of the cancer care continuum such as diagnosis through observation and bone throwing, treatment using various medicinal plant preparations, and referring patients to biomedical facilities. A greater understanding of their role and partnership between traditional healers and healthcare providers is needed to improve cancer outcomes for all.
**Check the effect of CRISPRa Activation Of Gene Promoter On The Expression Of mir3662**

Earvin, Jalyn; Kumar, Pradeep; Wang, Lizhong

**Introduction:** miRNAs (miRNAs) are small, highly conserved noncoding molecules involved in the regulation of gene expression. miRNA inactivates genes by degrading the target mRNA or simply inhibiting the translation of specific proteins. miR-3662 plays a critical role as an oncogene in different malignancies. Also, the expression of miR-3662 varies in different cell lines, for example some cells have high expression of MDA-MD-231, while other cell lines have very low expression of MDA-MD-468. However, it’s still unknown that miRNA-3662 expresses through coding gene promoter, or it has its own promoter.

**Result:** miR-3662 is present in the exon 19th of the HBS1L gene at the estimated position of 75,313 bp far from starting codon ATG. To investigate the expression of miR-3662 depends on the gene promoter, or it has its own promoter, we designed 3 sgRNAs on the CpG island of the HBS1L promoter using the online tool (https://chopchop.cbu.uib.no). Further these sgRNA were cloned in the lenti-sgRNA-(MS2)-zeo backbone (Addgene #61427). Further for single colony isolation, we selected 7 colonies and some cells from each colony were lysed and sample used to check the expression of Cas9 protein. Immunoblot analysis confirmed that colony number 5 show the expression of Cas9 (Fig. 1). This colony was used to further transfection of sgRNA plasmids with the control plasmid. Sample was collected at different time point such as 0h, 24h, 72h and 96h. Henceforth, miRNA was isolated and performed by quantitative real-time PCR (qPCR). Cells that contain sgRNA have high miR-3662 levels as compared to control.

**Conclusion:** Although miRNA-3662 expresses through its own promoter as identified in our previous study, the present study shows that miR-3662 is also regulated by the promoter of its host gene HBS1L.

**Materials And Methods:** Cell lines, antibodies, reagents, CRISPRa cell lines, lenti-sgRNA-(MS2)-zeo backbone

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**SON DNA-Binding Protein Regulates The Dna Hydroxymethyltransferases Tet2/Tet3 In Hematopoietic Stem Cells And Leukemia Cells**

Mustafa I; A Eltoum; L Vukadin; EY Ahn

**Introduction:** SON is an RNA-/DNA- binding protein that functions in pre-mRNA splicing and transcriptional repression. Altered expression of SON is associated with many disorders, specifically malignant and non-malignant hematopoietic disorders: leukemia, polycythemia vera and anemia. Because abnormal gene expression in hematopoietic stem cells (HSCs) is fundamental for leukemia development, we investigated molecular SON function in hematopoiisis through genetically engineered mouse models and cell lines. Preliminary studies in Son knockout mouse models demonstrate that depletion of Son alters lineage bias in HSC compartments. Our RNA sequencing data from mouse HSCs suggest that depletion of SON leads to compromised expression of ten-eleven translocation (TET2/TET3). TET2/TET3 proteins hydroxylate methylated DNA to activate gene expression, and TET2/TET3 mutations are implicated in abnormal HSC expansion and leukemia. Therefore, we hypothesize that altered SON expression causes abnormal gene expression during hematopoiesis by targeting TET2/TET3.

**Methods** We developed a mouse model with conditional Son knockout mice which has Son deletion only in hematopoietic cells by Vav promoter-mediated Cre expression (Vav-iCre Son f/+). The LSK (Lineage-, Sca-1+, c-Kit+) HSC population was isolated from these mice for RNA sequencing. Various bioinformatics tools were used to determine and visualize Tet2/Tet3 expression. To examine the role of SON in TET2 and TET3 expression in human leukemia, we performed small interfering RNA (siRNA)-mediated silencing of SON in K562 leukemic cell line. Polymerase chain reaction (PCR) methods analyzed splicing changes and quantified relative mRNA level changes (qPCR) of target genes. To evaluate SON binding to promoter regions of TET2 and TET3 genes, we performed chromatin immunoprecipitation (ChIP) in K562 cells using SON antibody; SON enrichment was determined by qPCR.

**Results/Conclusion** Our preliminary results confirm Tet2 and Tet3 mRNA decrease in bone marrow hematopoietic cells from Son knockout mice. We also found that TET2 and TET3 expression decreased upon siRNA-mediated SON knockout in K562 cells. ChIP experiments demonstrate SON binding to TET2 and TET3 promoters, suggesting that SON is a transcriptional activator of these target genes. This is the first time SON is implicated in direct activation of transcription. Therefore, we conclude that SON is important for hematopoietic development through transcriptional regulation of chromatin organizing proteins.
Feng, Bryan D.  
**Poster 15**  
**Role of Serine-Threonine Kinase Receptor-Associated Protein (STRAP) in Cancer**  
Feng, Bryan; Datta, Pran K., PhD

The Serine-threonine kinase receptor-associated protein (STRAP) is a 39 kDa protein of WD40 family involved in chaperoning function during the formation of multiprotein complexes shown to be active in tumor formation and progression. STRAP, first identified in our laboratory as an inhibitor of TGF-b signaling, interacts with Smad7 and synergizes with it in suppression of the canonical TGF-ß signaling. Deletion of Strap in mice leads to early embryonic lethality due to abnormal differentiation of embryonic stem cells. STRAP is upregulated in human cancers including breast cancer, lung cancer, colon cancer, and neuroblastoma. Its upregulation causes activation of a number of oncogenic signaling pathways, like MEK/ERK, Notch, and Wnt/ß-catenin pathways. STRAP induces mesenchymal morphology; promotes cell proliferation, migration, invasion and growth of tumors; and increases stemness of cancer cells. In colon cancer patients, upregulation of STRAP is associated with worse survival following adjuvant therapy. In contrast, patients carrying tumors with normal or low STRAP expression benefits from the treatment. Therefore, understanding the novel functions of STRAP in cancer development and finding new insights into the molecular basis of chemoresistance are critical. Because of its prevalence and importance in oncogenic processes, STRAP is an immense topic of interest. More comprehensive studies on STRAP could potentially yield breakthroughs on cancer treatment. Here, we will describe the TGF-beta-dependent and -independent functions of STRAP and provide a context for the significance of STRAP activity in the development of cancer.

Fouche, Iman R.  
**Poster 16**  
**Differential gene expression in control vs RAGE -/- mice in the context of pancreatic cancer**  
Fouché, Iman; Dudeja, Vikas, MD

**INTRODUCTION:** Pancreatic cancer is a highly lethal disease, being the fourth leading cause of cancer mortality in the USA. The most common type of pancreatic cancer is known as pancreatic ductal adenocarcinoma (PDAC) which occurs when cancer cells form in the cells that line the ducts that carry digestive enzymes out of the pancreas. Risk factors for this malignant disease include smoking, family history of chronic pancreatitis, advancing age, obesity, etc. Patients with PDAC show a very poor prognosis and resistance to conventional therapies. Therefore, to improve prognosis new therapies against pancreatic cancer are needed.

**METHODS:** Before tumor implantation, pancreatic cancer cells (KPC) were cultured in DMEM/F12 media with 10% FBS and maintained at 37°C and 5% CO₂, then 10,000 cells were implanted orthotopically into the tail of pancreas of C57BL/6J (control) and mice with disruption of the AGER gene (RAGE -/-). Tumor growth was measured at the end of the experiment and tumor samples were isolated and divided into 3 fractions -one fraction was used to analyze immune cell infiltration in the tumor by flow cytometry. The second fraction was used to isolate mRNA, from which cDNA was made and then used for gene expression analysis by RT-qPCR. The third fraction was used to isolate total proteins.

**RESULTS:** We observed that RAGE -/- mice develop smaller tumors as compared to control mice. Flow cytometry was utilized to analyze the immune cell infiltration of the tumor cells. Tumor cells were isolated and stained with a cocktail of antibodies against CD45+ (common leukocyte marker), CD3+ (Pan T-cell marker), and CD8+ (Cytotoxic T-cells). We observed that CD8+ T cells were significantly increased in the tumors of RAGE -/- mice. Obtaining high quality RNA is the most critical step in performing molecular techniques such as PCR. mRNA from tumor samples was successfully isolated with a high purity to use in downstream applications. Therefore, RT-qPCR was performed using SYBR green. The expression levels were normalized to 18SRNA and gene expression was calculated using the 2−ΔΔCT method.

**CONCLUSION:** A better understanding of the biology of pancreatic cancer is needed to develop new therapies against it.
**Gerrick, Rebekah N., BS**

**Poster 17**

**Health Care Use, and Costs of Acute Respiratory Distress Syndrome Survivors**

Gerrick, Rebekah N., BS

1. The long-term effects and expenses of acute respiratory distress syndrome survivors are little understood (ARDS).
2. To describe the functional and quality of life outcomes, health care utilization, and financial expenses of ARDS survivors 2 years following ICU discharge.
3. In Toronto, Canada, we selected a cohort of ARDS survivors from four academic tertiary care ICUs, and we prospectively followed them from the time they entered the ICU to two years following release.
4. The use of healthcare services, clinical and functional results, and direct medical expenses.
5. The overall 2-year death rate was 49%, with 85 percent of ARDS patients who were discharged from the ICU surviving to that point. Even though 65 percent of survivors had returned to work after 2 years, they were nevertheless limited in their ability to exercise. Although there was a trend toward greater physical function at 2 years, there was no statistically significant increase in health-related quality of life as judged by the Short-Form General Health Survey between 1 and 2 years. All other domains remained below that of the general population, with the exception of emotional role and mental health. The initial hospital stay, which included ICU costs, accounted for the majority of the survivor's health care expenses from ICU admission to two years following ICU discharge. Health care expenses following the initial hospitalization were associated with inpatient rehabilitation and readmissions to hospitals.
6. Two years after being released from the intensive care unit, ARDS survivors continued to have functional impairment and poor health-related quality of life. Hospital readmissions and inpatient rehabilitation were the main causes of subsequent medical use and expenditures.

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**Giles, Emily**

**Poster 18**

**Identification Of New Regulators Of Melanoma Growth And Progression**

Giles, Emily; Gupta, Romi, PhD

**Introduction** Melanoma is the deadliest form of skin cancer, accounting for ~65% of deaths associated with cancers of the skin. Genetic and non-genetic factors contribute to melanoma initiation and progression. Mutations in various genes are involved in melanomagenesis. Large-scale genomic DNA sequencing has identified activating mutations in *BRAF*, a component of the MAPK (ERK) signaling pathway, in ~50% of melanoma cases. In addition, by activating the MAP kinase (MAPK) pathway, mutations in neuroblastoma RAS viral oncogene homolog (*NRAS*), neurofibromin 1 (*NF1*), cyclin-dependent kinase inhibitor 2A (*CDKN2A*), microphthalmia-associated transcription factor (*MITF*), and phosphatase and tensin homolog (*PTEN*) are involved in melanoma initiation and progression. Since more than 50% of melanoma patients harbor oncogenic mutations in *BRAF*, several BRAF inhibitors (*BRAFi*) and inhibitors of MAPK/ERK kinase (MEKi) have been approved by the US Food and Drug Administration for treatment of melanomas. This includes the BRAF inhibitors, vemurafenib and dabrafenib, and the MEK inhibitor, trametinib, combined with immunotherapy for the treatment of metastatic melanoma. Although BRAF and MEK inhibitors produce impressive initial clinical responses against *BRAF*-mutant metastatic melanoma, the durability of the response is limited by the emergence of acquired resistance to the inhibitor, often within a few months of treatment initiation. Therefore, it is essential to identify regulators of melanoma growth that can be targeted to provide durable and effective melanoma therapy.

**Methods** In the present study, we focused on identifying new downstream targets of the oncogenic MAPK pathway, which is a major driver of melanoma initiation and progression. To do so, we treated *BRAF* mutant melanoma cells with various MAPK pathway inhibitors and measured the expression of new targets such as MAPK regulated phosphatases by immunoblotting.

**Results** Our results revealed that MAPK pathway inhibitors led to inhibition of expression of MAPK regulated phosphatases. By studying the role and regulation of these phosphatases we revealed new molecular mechanisms by which melanoma cells grow and proliferate.

**Conclusions/Discussion** Our results revealed new targets and mechanisms that are necessary in driving *BRAF*-mutant melanoma growth. Their detailed study can aid in the discovery of new therapeutic options for melanoma patients.

**Acknowledgements** MSM/TU/UAB O’CCC Partnership 2U54CA118948-17, Dr. Romi Gupta
**Hollins, Zaria, MPH**

**Poster 19**

**FOSL1**

Hollins, Zaria, MPH; Hollins, Zaria; Liu, Mingli

**Background:** FOSL1 (FOS-Like1; also named FRA-1), encoding FRA-1, is an AP-1 transcription factor with important 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 tumor cell proliferation and survival and acts as a master switch of epithelial-to-mesenchymal transition (EMT). Our previous results showed that FOSL1, which is regulated by TRPM7, is responsible for sustaining glioma cell growth and glioma cell invasion in vitro and is an unfavorable prognostic marker for GBM patients. As we previously found that TRPM7 channels regulate glioma stemness, it is worth further investigation into the regulation of FOSL1 in remodeling glioma stemness in support of glioma cell invasion and progression.

**Methods:** Flow cytometric analysis to assess CD133 expression in A172, U87MG and PDX-L14 glioma cells by FOSL1 silencing. ALDH1 enzymatic activities were determined by the ALDEFLUOR assay which was performed in FOSL1-knockdown A172, U87MG and PDX-L14 cells.

**Results:** It was revealed that once FOSL1 was knocked down by siFOSL1, the number of CD133+ cells were decreased in A172, U87MG and PDX-L14 cells. These results indicated that the downregulation of FOSL1 resulted in a reduced GSC population. To address the question of whether FOSL1 would affect ALDH1, another GSC marker, the ALDEFLUOR assay was performed on an identical model of glioma cells aforementioned, in which FOSL1 was under expressed by siFOSL1 construct. FOSL1 silencing decreased the number of ALDH1-positive cells in A172, U87MG, and PDX-L14 cells. In accordance with the relationship of FOSL1 and CD133, here, our results provided further evidence that FOSL1 knockdown resulted in a decreased GSC population as defined by the ALDH1+ population.

**Conclusion:** These results indicated that down-regulation of FOSL1 causatively reduced the expression of GSC markers CD133 and ALDH1 and FOSL1 may play a critical role in glioma stemness.

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**House, Prada C., MPH**

**Poster 20**

**Analysis of Mitochondrial Genes Variants and Expressions in Early Colorectal Tumors**

House, Prada; Aikhionbare, Kylie; Wallace, LaShanale, PhD.

**Introduction** Colorectal cancer (CRC) is the third most commonly diagnosed cause of cancer-related deaths in both men and women in the United States. Numerous studies have analyzed mitochondria DNA mutations in CRC and other tumors. Results from these studies have detected high mutation rates which may lead to mitochondrial deregulation and tumor progression. Most CRCs develop from adenopolyps via the adenoma-carcinoma sequence.

**Methods** Analysis of mitochondrial mutations and gene expression may provide a mechanism for inhibiting this tumoral sequence in individuals with a high risk of developing CRC. In the present study, PCR-based sequencing and reverse transcription-quantitative PCR (RT-qPCR) were used to determine if mutations in mitochondrial encoded genes and levels of expression of these genes could influence the progression of the adenoma-carcinoma tumoral sequence. Genes analyzed included MT-RNR1, MT-COI, MT-ATP6, MT-MT-CYB, and mitochondrial ND genes that are involved in the normal metabolism of mitochondria. Measurements were made for 34 tissue sample pairs obtained from various types of colorectal adenomas and corresponding adjacent normal tissues. Additionally, mitochondrial complexes I (NADH: ubiquinone oxidoreductase) and III (CoQH2-cytochrome c reductase) protein was analyzed. Results There was progressive differential expression of mt-genes and complexes I and III proteins among the colorectal tumor stages relative to their paired normal samples. The level of complexes I and III was higher in tumor tissues relative to adjacent normal tissues. Noticeably, the expression of MT-COI was higher in late-stage carcinomas among, all studied transcripts. We detected 54-point mutations in one region ranging from 11871-13950. The frequency of these mutations in all stages was as followed; 71.5% in tubular adenoma, 57% tubulovillous, and 43% in villous and carcinoma.

**Conclusion** Our results suggest that alterations in mt-gene expression play a role in the transformation of the colorectal tumoral stages.
Cancer Clinical Trials are responsible for many new drug developments and give cancer patients a sense of hope when it comes to cancer therapies. Unfortunately, they do not fairly represent all people, with underrepresentation seen in all racial and ethnic groups, especially African Americans. This leads to receiving suboptimal care, with the potential for their specific needs not being met. They represent 13.3% of the total population in the U.S. but comprise less than 4% of patients in clinical trials. Furthermore, access to clinical trials in the indigent population is either non-existent or limited. With the combination of underrepresentation and the prevalence of cancer within this community, African Americans will only continue to be not given the care they deserve on a humane level, as biological factors are needed to make specific, precision medicine. Therefore, a cross-sectional survey was administered to patients with cancer seen at Cooper Green Mercy Health Services Authority (CGMHSA). This will be followed by interviews with patients and with the healthcare team with specific questions to ultimately use their results to tailor a specific recruitment design to promote cancer clinical trial participation in patients seen at CGMHSA. Through a 22-item Clinical Trial Knowledge scale, knowledge will be assessed across psychosocial and clinical factors. Qualitative interviews provide exploration of potential barriers to referral to clinical trials at the UAB O’Neal Comprehensive Cancer Center. Currently, 29 patients with cancer have responded to the surveys with the goal of 100 responses. The next step needed is to increase participation by recruiting more patients and having trainees and assistants help encourage participation. Our research is the beginning of a much-needed solution to an overlooked problem within the medical community that will soon reap its benefits.

Hughes, Kamal, BS

An Examination of Hookah Usage and Its Association with Lung Cancer Risk in the United States: A Systematic Literature Review
Hughes, Kamal, BS; Hughes, Kamal; Rivers, Brian, PhD, MPH

Introduction: Lung cancer continues to be the most prevalent form of cancer, behind skin cancer, in the United States. The American Cancer Society estimates 236,740 new lung cancer cases and 130,180 lung cancer-related deaths will occur in 2022 alone. Although on the decline for some groups, cigarette smoking is the most substantial risk factor for lung cancer due to tobacco smoke’s carcinogenic substances. However, little research has thoroughly investigated waterpipe tobacco smoking (WTS), also known as hookah, as an alternative modality for tobacco use and its association with lung cancer risk.

Methods: A literature search of articles was performed utilizing the biomedical database: PubMed. Search parameters included articles published between September 2012 and September 2020. Search keywords 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 were more likely to smoke hookah than their white counterparts in the US. Moreover, waterpipe tobacco has similar carcinogenic substances present in cigarette smoke, such as tobacco-specific nitrosamines (TSNA), polycyclic aromatic hydrocarbons (PAHs), and volatile organic compounds (VOCs). WTS usage patterns typically extend between 45-60 minutes in which users can potentially inhale 100-200 times the amount of smoke that a single cigarette would produce.

Discussion/Conclusion: WTS is becoming a growing alternative to traditional cigarette smoking. As hookah stores and lounges continue to emerge, the increasing access and 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. The Food and Drug Administration must impose stricter federal regulations on waterpipe tobacco and its corresponding parts and accessories to mitigate additional lung cancer risk and incidence. Furthermore, there needs to be a more concerted effort to implement WTS interventions (i.e., anti-WTS campaigns) to stifle the growing use in adolescents and young adults.
**Hurley, Eric A.**

Clinical Characteristics of UAB Glioblastoma Cases 2018

Hurley, Eric A.; Nabors, Burt; Abdelrashid, Moaaz,

**Background:** Glioblastomas are the most common form of primary brain cancer. Patients diagnosed with glioblastomas often face a poor prognosis, despite the aggressive treatment. So far, the most accurate predictor of response to treatment is the activity of O6-methylguanine-DNA methyltransferase (MGMT). The MGMT gene encodes a protein necessary for DNA repair. Methylation of this gene’s promoter reduces or eliminates its activity, causing poor DNA repair and no longer inhibiting the effect of alkylating drugs. Past research has shown that methylation of the MGMT promoter is correlated with higher overall survival (OS) in patients undergoing chemotherapy with drugs such as temozolomide (Temodar).

**Methods:** This study focuses on the effects of MGMT methylation on the survival of patients with glioblastomas. 141 patients diagnosed with glioblastomas in 2018 at the University of Alabama at Birmingham (UAB) Hospital were observed in the study, and their demographic data (age, sex, race, income, and Rural-Urban commuting area [RUCA] codes) were collected. MGMT methylation status was collected for 126 of the 141 patients. Finally, the OS was calculated using the date of diagnosis and either the date of death or date last seen (for living patients).

**Results:** Patients expressing methylated MGMT showed a markedly longer OS than those without. The 46 patients with methylated MGMT had an average overall survival rate of 719 days after diagnosis. Those with unmethylated MGMT (80 patients) had an average OS of 458 days.

**Conclusion:** MGMT methylation is correlated with a greatly increased overall survival rate in the UAB glioblastoma population.

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**Isaac, Brittany, BS, MS**

Regulation Of Ultraviolet Radiation Induced Responses By K-Homology Splicing Regulatory Protein

Isaac, Brittany, MS; Chung, Minh; Harunur, Rashid, PhD; Yusuf, Nabiha, PhD

**Introduction** Sunlight is the main source of the three forms of ultraviolet light. One form, UVB radiation (290-230nm), causes deoxyribonucleic acid (DNA) damage in the form of cyclobutene pyrimidine dimers (CPD). Repair mechanisms correct damage from acute exposure to UVB and other forms of ultraviolet light. Lack of repair releases a cascade of proinflammatory responses to the sites of UVB damage. Chronicity of this inflammatory process promotes tumor development. K-homology type splicing regulatory protein (KSRP) is an adenylate uridylate rich-element (ARE)-binding protein that binds to mRNAs resulting in decreased expression due to increased mRNA decay. This project aims to determine the role of KSRP in UVB mediated DNA damage, inflammation, and skin cancer.

**Methods** KSRP knockout (KO) and their wild type (WT) counterparts were treated with a single dose of UVB (100 ml/cm2) on their dorsal skin. Twenty-four hours after exposure, their skin was harvested for estimation of CPDs by immunohistochemistry. In a separate experiment, under similar conditions, skin-bi-fold thickness was measured. For detection of KSRP in human samples, biopsied skin tumor patient sample and normal skin samples were collected from Whitaker Dermatology Clinic. RNA was isolated from human skin and skin tumor samples and was analyzed for KSRP by quantitative polymerase chain reaction (qPCR).

**Results** KSRP KO had significantly fewer (***p<0.001) CPDs following cutaneous exposure to a single dose of UVB (100 ml/cm2). UVB induced skin inflammation was significantly lower (***p<0.001) in KSRP KO mice in comparison to WT mice. We have discovered that KSRP expression was found to be higher in human skin tumors compared to normal skin, and KSRP deficiency resulted in fewer UVB induced CPDs in mouse skin.

**Discussion/Conclusion** KSRP is a protein involved in the innate immune response and contributes to the proinflammatory state of the injured organ. Research of this protein in UV-exposed mice skin has discovered less CPD with KSRP deficient skin in comparison to skin with KSRP present. From the findings of this project, KSRP is significantly expressed in human skin tumor cells, which support the idea that it has a role in tumor producing inflammatory process.
Succinate dehydrogenase (SDH) is a metabolic enzyme that links the TCA cycle to the electron transport chain (ETC, where it is called complex II). SDH is composed of 2 membrane-bound subunits and 2 soluble subunits, facing the mitochondrial matrix. SDH enzymatically converts succinate to fumarate, donating high energy electrons from succinate to FAD and eventually to complex III via coenzyme Q. Under conditions of stress or hypoxia that prevent electron flow through complexes III and IV, oxidized coenzyme Q (CoQ) can be excessively reduced to CoQH2, which reduces the available CoQ pool. When the oxidized CoQ pool is significantly reduced, NADH and FADH2 cannot effectively shuttle electrons through the ETC, and free radicals are generated.

It has been recently reported that SDH can function in reverse, allowing CoQH2 to be converted back to CoQ by utilizing fumarate as an electron acceptor, converting fumarate to succinate (the reverse reaction of the normal TCA cycle). We hypothesize that a novel proteolytic processing step in the large subunit of SDH (SDHA) is involved in facilitating this physiologic response to mitochondrial stress. Western blot demonstrated that, when electron flow through the ETC is blocked by inhibition of complex III with antimycin A, that cleaved versions of SDHA are present. Our mass spectrometry data showed most cleaved polypeptides occur after amino acid 574. To visualize the tertiary structure of SDHA in both normal and stress-induced states, we utilized the Alphafold2 program to compare the known crystal structure of SDHA to the predicted, stress-induced cleaved structure, which retains all important enzymatic features of full-length SDHA. The largest of these cleaved bands corresponds to a novel cleavage event that we have identified in mass spectrometry datasets. Based on these data, cleaved SDHA is formed by stress-induced proteolytic cleavage between amino acids 574 and 575, which causes release of the C-terminal, matrix-facing portion of SDHA (which is not involved in the core enzymatic mechanism). These data demonstrate that Complex II subunit SDHA can be proteolytically processed following electron transport chain stress, which we hypothesize facilitates the use of SDH reverse flux to regenerate the oxidized coenzyme Q pool.

Introduction: Known as one of the top 10 most common cancers in both men and women, kidney cancer is increasing globally. Located at the back wall of the abdomen and guarded by the lower rib cage, these organs carry out the responsibility of flushing excess water, salt, and waste products carried in the blood from the renal arteries. Clear cell renal cell carcinoma, ccRCC, is the most common form of renal cancer. ccRCC is given its name due to the clear appearance of the cells when studied histopathologically. The survival rate for tumors confined to the kidney is excellent. However, survival from this cancer is dramatically reduced when there is spread to other organs i.e., metastasis.

L-2-Hydroxyglutarate (L2HG) is a oncometabolite which is elevated within renal cancer cells (RCC). This increase is due to loss of the enzyme L-2HG dehydrogenase (L2HGDH). This increase is known to suppress the activity of α-ketoglutarate dependent enzymes that include various epigenetic and epitranscriptomic dioxygenases e.g., DNA demethylase (TET), histone demethylases (KDMs), RNA demethylases (ALKBHS), etc. It is believed that higher levels of L2HG promote tumor growth.

Methods: 786-O is a ccRCC cell line. It has an elevated level of L2HG that can be lowered by restoring the L2HGDH enzyme in the cell. 786-O cells expressing either control plasmid or L2HGDH expressing plasmid were grown in RPMI media in six-well plates. When the plates were confluent, a scratch was made in each of the wells harboring either control or L2HGDH expressing 786-O cells. The width of the scratches was measured at different time points (0hr, 6hr, 12 hr., 16hr) from n=3 biological replicates of each group and finally, the percentage of scratch or wound closure was measured at different time points.

Results/ Conclusion: We observed that the wound healing capacity of L2HGDH containing 786-O, which have low L2HG levels, is lower than control (high L2HG) 786-O cells. This data suggests that lowering L2HG level can be a means to downgrade the metastatic potential of RCC cells.
**Mahaffey, Carolina, BS**

**Factors Associated with Genetic Counseling and Testing For Black Women Diagnosed with Triple Negative Breast Cancer in the United States**

Mahaffey, Carolina, BS; Mahaffey, Carolina; Rivers, Brian, PHD, MPH

**Introduction** Breast cancer is the second leading cause of death in women. Triple-negative breast cancer (TNBC) displays negative expressions of estrogen, progesterone, and human epidermal growth factor receptor-2. 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 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. Genetic counseling and testing allow for the discovery of mutations that would allow for decision-making on screening, chemoprevention, and prophylactic measures that would reduce morbidity and mortality. The purpose of this literature review is to better understand the factors associated with Black women’s receptivity to genetic counseling and testing.

**Methods** A literature search was performed using the online database of PubMed and hand-searching. Search parameters were 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** Numerous studies 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. The literature search did also reveal that there were women wanting to know their status who were open to genetic counseling and testing if it was recommended by a medical provider. 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.

**Discussion/Conclusion** 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. An increase in the education on the importance of these resources is warranted in the Black community to cause receptivity and use of these vital services.

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**Mansur, Ramy**

**Type I Interferons Enhance Repair of Ultraviolet Radiation Induced DNA Damage and Regulate Photocarcinogenesis In Mice**

Mansur, Ramy; Sherwani, Mohammad Asif, PhD; Yusuf, Nabiha, PhD

**Introduction:** Type I interferons (IFNs) are important enhancers of immune responses which are downregulated in human cancers, including skin cancer. Solar ultraviolet (UV) B radiation is a proven environmental carcinogen, and its exposure contributes to the high prevalence of skin cancer. The carcinogenic effects of UV light can be attributed to the formation of cyclobutane pyrimidine dimers (CPD) and errors in repair and replication of DNA.

**Methods:** Type I IFN receptor 1 knockout (IFNAR1 KO) and their wild type (WT) counterparts were treated with a single dose of UVB (100 mJ/cm2) on their dorsal skin. Twenty four hours after exposure, their skin was harvested for estimation of CPDs by immunohistochemistry and ELISA. The expression of DNA repair gene xeroderma pigmentosum A (XPA) was determined by quantitative polymerase chain reaction (qPCR). For photocarcinogenesis experiment, mice were pretreated with anti-IFNAR1-Ab (250 ug/mouse three times per week) prior to each UVB (180 mJ/cm2) exposure up to 30 weeks. Statistical analysis. In all experiments, UVB exposed and unexposed groups were compared separately using two-way analysis of variance (ANOVA). In each case, a value of p < 0.05 was considered to be statistically significant.

**Results:** IFNAR1 KO had significantly more (**p<0.001) CPDs following cutaneous exposure to a single dose of UVB (100 mJ/cm2). UVB induced expression of XPA was significantly higher (**p<0.001) in wild type (WT) mice in comparison to IFNAR1KO mice. When mice were subjected to photocarcinogenesis,treatment with anti-IFNAR1-Ab resulted in significant increase (**p<0.001) of tumor number compared to the mice pretreated with isotype control antibody.

**Discussion/conclusion:** Overall, our studies reveal a previously unknown action of type I IFNs in repair of photodamage and regulation of photocarcinogenesis. Strategies that enhance type I IFN production may be useful for prevention of pre-malignant skin lesions before they develop into skin cancer.
**Analyzing the Degree of Cytotoxicity for Combined Therapeutic Approaches for Glioblastoma Radiation Resistant and Radiation Sensitive Patient Derived Xenografts.**

Metcalf, Nyla; Schanel, Taylor, BS; Nassour, Lauren, BS; Willey, Christopher, MD., PhD

**Introduction:** Glioblastoma (GBM) is characterized as a highly aggressive brain tumor with rapid rates of cell division. With its targeting of localized regions, the affected areas of the brain cease functioning, resulting in the patient succumbing to the tumor. Standard treatments are in practice to combat the activity of the glioblastoma; however, its intratumoral heterogeneity offers little to no remedial success, therefore labeling GBM as currently incurable. To design improved forms of treatment, it is important we investigate the various tumor cells present within the tumor microenvironment (TME). Therefore, we generated patient-derived xenograft (PDX) glioma stem cell lines with radiation-resistant counterparts to better model this concept. We hypothesize that the radiation-sensitive line will be susceptible to the cytotoxic effects of radiation-therapy (RT) and temozolomide (TMZ) therapy, however, we suspect the radiation-resistant line will only be susceptible to TMZ.

**Methods:** We developed the radiation-sensitive (JX14P) and -resistant (JX14P-RT) PDX GBM cell lines through the implantation of primary tumors into immunodeficient nude mice. To achieve radiation resistance, these mice were treated with 6 fractions of 2 Gray (Gy) of radiation over the span of 14 days. Cells were infected with an mCherry lentivirus for visualization via fluorescent imaging. Cells were then plated on a geltrex matrix and divided into the following groups: untreated cells (control), cells treated only with temozolomide (TMZ), cells treated only with RT, and cells treated with both RT and TMZ. Two plates were utilized per condition -0Gy (control), 3Gy, and 5Gy. The cell lines were imaged using a Cytation 5 cell imaging multi-mode reader after each treatment to monitor proliferation via fluorescence intensity. We also investigated viability using the gold-standard CellTiter-Glo viability assay which quantifies the amount of ATP present.

**Results:** The JX14P-mCherry (radiation-sensitive) line is susceptible to cytotoxicity from RT and TMZ, but JX14PRT-mCherry (radiation-resistant) is only susceptible to temozolomide cytotoxicity. This was measured based on viability and proliferation.

**Discussion/Conclusion:** The various cell populations present in the GBM tumor microenvironment enhance the tumor’s ability to evade cell death and spread chemo-radioresistance throughout the brain. However, if we design adequate GBM tumor models that properly recapitulate the intratumoral heterogeneity, we will be able to obtain a more accurate representation of treatment response.

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**Mwazembe, Irene, BSN**

**Poster 30**

**Receptor Tyrosine Kinase Activation In Castration Resistant Prostate Cancer**

Mwazembe, Irene, BSN; Haseena Polk,Honghe Wang

Castration resistance prostate cancer (CRPC) is the leading cause of poor health and death in patients with prostate cancer. Present therapies for CRPC are non-curative; the treatments are aimed at controlling symptoms and slowing progression of the disease. Receptor tyrosine kinases (RTK) are cell membrane proteins involved in normal cell proliferation and survival. However, abnormal activations of RTK are implicated in the development, progression, metastasis, and therapy resistance of various cancers including prostate cancer. The current study aims at identifying targetable RTK, which are activated in CRPC. The study will screen and identify RTK that are hyperactivated in CRPC and evaluate the effect of different RTK inhibitors such as dasatinib and axitinib on the growth and survival of CRPC cells. The Human Receptor Tyrosine Kinase Phosphorylation Antibody Array and single cell western blot protocols were used to identify RTK activation after Androgen Deprivation Treatments. The effect of RTK inhibitors on growth and survival of the prostate cancer cells will be evaluated using MTT assay and the flow cytometry. Preliminary results confirmed that thirteen RTKs were hyper-phosphorylated following hormonal manipulation. Specific RTK inhibitors inhibited proliferation of castration resistant cells. The current study suggests, RTKs are differentially phosphorylated following hormone therapy and identified RTKs activation may be as part of the resistance mechanism. Thus, these findings provide a rationale for exploration of combining different RTK pathway inhibitors for treating CRPCs in the clinic and would significantly improve treatment outcomes.

**Keywords:** Prostate cancer, Castration resistance prostate cancer, Receptor tyrosine kinase and Receptor tyrosine kinase inhibitors.
Nagaraj, Nayana H.  

**Generalizations of Aurora Kinases and their effects on Ovarian Cancer**  
Nayana Nagaraj; Karthikeyan Mythreye PhD; Rajkarnikar Resha BS; Page Emily BS

Ovarian Cancer is caused by the abnormal growth of the cells in the ovaries. The major problem is the lack of early detection and non-specific symptoms. Disruption of checkpoints in the cell cycle can cause the progression of cancer. The cell cycle is regulated by many kinases, which are proteins that regulate the growth and function of cells. Kinases act by adding a phosphate group to a molecule, increasing the potential energy in the molecular bonds leading to either activation or inactivation of signaling pathways. Aurora Kinases (AURK) are serine/threonine cell cycle kinases that impact Ovarian cancer by regulating mitosis. AURKA regulates spindle fibers in the beginning stages of mitosis. AURKB plays a part in chromosomal segregation monitoring. AURKC is similar to AURKB but is associated with meiosis. Aurora kinases have also been shown to be a part of DNA repair mechanisms, making them a target of cancer treatment therapies through AURK inhibitors. Such inhibitors have also been shown to reduce cancer progression by influencing tumor vascularization. Here, I have presented a literature-based analysis of the mechanism of onset and symptoms of Ovarian Cancer, the role of Aurora Kinases in individuals with Ovarian Cancer, and the implications of AURK inhibitors in cancer treatment methods.

Naveed, Nabaa, BS  

**Ribitol Testing in Zebrafish Models Of FKRP Dependent Dystroglycanopathies**  
Naveed, Nabaa, BS; Karuppasamy, Muthukumar; Alexander, Matthew

**Background:** A subset of congenital muscular dystrophies called Dystroglycanopathies are caused by gene mutations that occur in the FKRP gene and affect the glycosylation of the alpha-Dystroglycanopathy protein (α-DAG1). FKRP is a Fukutin related protein and can be found in the skeletal and cardiac muscle, in addition to the brain. Pathogenic FKRP variants in human patients can result in a wide spectrum of Dystroglycanopathy phenotypes ranging from progressive muscle weakness (limb girdle muscular dystrophy 2I/R9) to severe muscle and brain malformations (Walker Warburg Syndrome/WWS). The zebrafish is an excellent model for neuromuscular disorders due to its low costs, ease of genetic manipulability, large offspring size, and most importantly the ability to rapidly evaluate drugs through direct administration to the fish water. Recently, several groups have identified ribitol as an important molecule that can glycosylate α-DAG1 protein in the absence of FKRP in LGMD2I mice. However, little is known about ribitol supplementation in other FKRP animal models.

**Hypothesis:** Ribitol administration can correct FKRP-associated disease pathologies in the zebrafish. Ribitol treatment in our FKRP mutant fish models will correct muscle physiology and overall lifespan in the zebrafish.

**Methods:** The Alexander lab has generated FKRP knock-in C318Y (WWS) and L276I (LGMD2I) models that I will determine if ribitol administration can rescue. I will administer 3 optimal doses (1, 2.5, and 10 µM) of ribitol in fish water in the 1 day post fertilization (dpf) FKRP WWS zebrafish. At 3 to 5 dpf, I will evaluate zebrafish motility, histology, and overall phenotypes.

**Results:** Early results indicate that ribitol supplementation can restore some muscle and overall locomotive function in the FKRP LGMD2I and WWS zebrafish models.

**Conclusions/Future Directions:** Ribitol supplementation may be effective in restoring FKRP-associated pathologies in Dystroglycanopathies. Additional Dystroglycanopathy causative genes may and potentially patients may benefit from ribitol as a corrective molecule.
**Nixon, Rachel D., BS**

**Poster 33**

**Racial Disparities In Endometrial Cancer Patients At A Single Academic Institution**

Nixon, Rachel D.; Elrod, Savannah; Harsono, Alfonsus Adrian Hadikusumo, MD; Wall, Jaclyn M. Arquiette, MD; Boitano, Teresa, MD; Dholakia, Jalak, MD; Evans, Elizabeth, MD; Moore, Jenna; Foxall, McKenzie; Arend, Rebecca, MD, MSPH

**Introduction:** Historically, Black patients with endometrial cancer (EC) have worse survival than non-Black patients. Obesity has been associated with poor survival in various cancers, though a relationship between race/obesity and survival is not well understood. We sought to explore the relationship between these factors and survival in patients with EC.

**Methods:** EC patients between 2007-2021 were included. Demographic/death information was collected from the EMR and public records. Effect of BMI (body mass index)/race on overall survival was analyzed using Kaplan-Meier survival methods and Cox hazard ratios.

**Results:** 1042 women were included. Black women had higher death rates than non-Black women (17.4% v. 11.3%, p<0.01) and decreased five-year cancer-specific survival (68.6% vs 83.4%, p=<0.001). Black women were more likely to be morbidly obese (35.7% v 23.5%, p<0.001), but there was no difference in presentation of obese/overweight/normal BMI patients (HR=0.66, 95% CI:0.35-1.24; HR=0.61, 95% CI:0.36-1.02; HR=065, 95% CI:0.39-1.10). There was no difference in risk of EC death in morbidly obese/obese/overweight patients compared to normal BMI patients (95% CI: 0.35-1.24; 0.36-1.20; 0.39-1.10). There was no difference in age at diagnosis between Black and non-Black women, although age at diagnosis increased risk of death in populations 60-69, 70-79, and >80 years compared to <49 years (HR=8.76, 95% CI:1.16-66.00; HR=10.51, 95% CI:1.35-81.78; and HR=22.00, 95% CI:2.75-176.14).

**Conclusions:** Black women at our institution had higher EC-specific mortality than non-Black women. This disparity cannot be contributed to differences in age or BMI; investigation into other contributing factors is needed to diminish disparities and improve the survival of Black women with EC.

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**Norfleet, Vonnetta D., BA**

**Poster 34**

**Impact of COVID-19 on Cervical Cancer Screening in Primary Care Clinics of a Safety-net Hospital System in Georgia from 01/2018 to 12/2021**

Norfleet, Vonnetta D., BA; Lee, Regina, MD; Flowers, Lisa, MD

**Introduction:** Cancer is the second leading cause of death for adults in the United States. Cancer screenings have developed over the past century to help healthcare providers detect cases of cancer in their initial, more curable stages. Government mandated shut-downs and stay at home orders due to the COVID-19 virus, have disrupted daily activities, such as preventive primary care visits where women often get gynecological care. In this study, we aim to show the impact of COVID-19 protocols on the cervical cancer screenings (CCS) from January 2018 - December 2021 at primary care clinics (PCC) within a safety-net hospital in Atlanta, Georgia.

**Methods:** Women ages 40-65 who underwent CCS by cervical cytology and high-risk human papillomavirus (hrHPV) testing during January 2018- December 2021 in PCC according to current guidelines were included. The aggregate data for number of clinic visits during the time period and percentage of women who underwent screening were pooled from routine quality improvement surveillance. The data was converted to see the overall trend changes from 2018-2021 on the rate of CCS.

**Results:** Total 178,000 women ages 40-65 eligible for a cervical cancer screening were seen in the PCC during the study period. For 2018, the percentage of women who received recommended CCS was 50% out of 32,066 women. For 2020, the percentage was 53% out of 30,054 women, while in December of 2021, the total was 59% out of 24,910 women. Although the total number of patients seen were reduced during the quarantine, the percentage of women who underwent timely cervical cancer screening did not decrease.

**Discussion:** Based on the rates of CCS across the nation during the COVID-19 pandemic, we expected that CCS for women ages 40-65 at the PCC would have been negatively impacted. Our data reveals that rates of CCS during the pandemic did not decrease for patients who were able to complete in-person visits during the quarantine. Both the rates of CCS and the total number of patients increased towards the end of the pandemic, which may be a result of the healthcare system’s concerns for delayed cancer screening due to COVID-19.
Notice, LilyJasmine  

**Disparity of Cervical Cancer Diagnosis by Age, Race and Residence in Alabama**  
Notice, LilyJasmine; Chowdhury, Sabrina; Shrestha, Sadeep

**Introduction:** The human papillomavirus (HPV) is the most common sexually transmitted infection and causes almost all cervical cancer. Cervical cancer is the fourth most common cancer in women. Effective primary (HPV vaccination) and secondary prevention approaches (screening and treating precancerous lesions) are available to treat cervical cancer. However, the resources are not available to everyone including in the US, where there are disparities between races and geographical regions.

**Methods:** This retrospective study assesses rural-urban and racial disparities in age at cervical cancer diagnosis, using the 2010-2019 data from the Alabama Cancer Registry. Rurality was defined based on 2010 U.S. Census Bureau's rural-urban classification. Median age was calculated for each race and residential area group and also categorized using the CDC standard. Distribution of age was assessed by race (White vs Black) and further in their geographical region of residence (urban vs rural).

**Results:** During the study period, there were 479 Black and 1131 White women diagnosed with Cancer. There were 51 women from other races combined. Overall, median age of diagnosis was older for Black women versus White (51 vs 48 years). However, white women in urban areas were diagnosed at younger age (47 years) then black women in urban areas (52 years) or both white (49 years) and black women in rural areas (50 years).

**Discussion/Conclusion:** It is plausible that white women have genetic predisposition that make them susceptible to cervical cancer at earlier age. However, this was only observed in urban setting and not among those who lived in the rural areas. Rural areas do not have resources for screening regardless of race, so it is possible that they all are diagnosed at late stage. On the other hand, it is possible that there is disparity in access to care in urban settings such that while whites are diagnosed earlier, they may be diagnosed at earlier stage of cancer, that can be cured with better treatments. Future studies will be warranted to examine this disparity by staging of cancer and in other states in southern US to validate if this disparity is real or confounded by other factors.

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Rao, Kirthana, MS  

**Therapeutic Strategies To Overcome Drug Resistance In Ovarian Cancer**  
Rao, Kirthana, MS; Singh, Santosh Kumar PhD; Singh, Rajesh PhD

**Introduction:** Ovarian cancer (OvCa) is the most common malignancy in the gynecological system. More than 70% of patients received a delayed diagnosis with widespread metastases. For those individuals, cytoreductive surgery and platinum-based chemotherapy are the standard treatment regimen. However, many patients acquire therapeutic resistance and do not respond to anti-cancer therapy, which is the main obstacle to curative OvCa treatment. Consequently, it is crucial to understand the molecular mechanisms behind the resistance. In this study, we detailed the numerous factors that influenced OvCa progression and resistance and discussed strategies to improve patient outcomes.

**Methods** Different internet sources or databases, including PubMed, Google Scholar, Scopus, and Science Direct, were searched for relevant information and scientific data. The search terms included phrases like "Paclitaxel (PTX) resistance in OvCa," "Carboplatin (CB) resistance in OvCa," and "Multi drug resistance (MDR) or P-glycoprotein (P-gp) function in PTX and CB resistance."

**Results** Refractory OvCa is often treated with the platinum-based anticancer drug, and it remains a substantial unmet need. However, studies have found that the emergence of multidrug resistance (MDR) significantly reduces the effectiveness of platinum-based drugs, such as PTX, Cisplatin, and CB in OvCa cells. The mechanisms driving drug resistance to chemotherapy are complex. Various pathways, including the post-translational modifications of tubulin, increased expression of specific MDR efflux transporters (P-gp, ABCC1, ABCG2), NF-κB, and PI-3K/Akt pathways involved in drug resistance to PTX. Cellular processes such as autophagy, oxidative stress, dysregulated microRNAs, cell metabolism, and epigenetic changes can also affect the development of resistance. Therefore, our comprehensive drug resistance analysis demonstrates the clinically relevant information and more precise therapy for OvCa.

**Conclusions** These results imply that accumulative knowledge of complex underlying therapy-resistance mechanisms may help to reduce OvCa fatalities. This study offers fresh perspectives on approaching patients with drug-resistant OvCa and a novel molecular conceptual framework for identifying the most likely MDR-mediated mechanism affecting OvCa.
Androgen deprivation therapy (ADT) is a common method for prostate cancer (PCa) treatment for a long time. But, the major side effects of ADT caused to search for a better treatment option for the PCa. Dihydrotestosterone (DHT), which is testosterone, at lower concentrations should stimulate cell growth. This has been published in many experiments in the literature. In our experiment, we tried to investigate the effect of different concentrations of DHT on PCa cells. Interestingly, we found that DHT acts differently in higher concentrations.

Based on cell viability, the effectiveness of DHT was preliminarily investigated for possible biological anti-cancer activity on different prostate cancer PCa cell lines (LnCap, RC77). We used the MTT assay which detects the cell viability by using a colorimetric technique for assessing cell metabolic activity of the PCa cells. In our experiment, we used 2 sets of LnCap cells with Serum Free and charcoal-stripped media and tested MTT the in consequent 3, 5 and 7 days after adding DHT. It is already established that the DHT treatment proliferates all CRPC cells and also accelerates STAT5 phosphorylation. On the contrary, according to our study, though low DHT concentration (5,10 & 20 nM) increases cancer cell proliferation and metastasis, the high concentration (50 & 100 nM) kills the prostate cancer cells instead.

Further investigation for ‘protein extraction and gene expression’ will be done to know more about the cause of cancer cell death at high DHT concentrations.

As one of the most common malignancies among men and globally, prostate cancer is still affected by racial disparities relative to a different response to treatment and poor overall survival among patients. Androgen receptor has a pivotal role in the progression of prostate cancer. Therefore, Androgen deprivation therapy (ADT) is the front-line treatment for prostate cancer. But ultimately, they turn into castration-resistant prostate cancer (CRPC), leading to earlier mortality and inadequate prognosis. Kaiso as a transcriptional regulator has been implicated in African American and Caucasian American prostate cancer patients, while the therapeutic role of androgen receptor antagonist enzalutamide was underscored. In an attempt to give further insight into the role of kaiso in cancer disparities, we set to evaluate the effect of low and higher doses of enzalutamide treatments (3,5 & 7 days) on LnCap and RC-77T cell lines while focusing on kaiso-protein and Kaiso-DNA interactions. MTT, RT-qPCR, (Rapid immunoprecipitation mass spectrometry of endogenous protein (RIME) and Cleavage Under Targets and release using nuclease (Cut and Run) assays have been determined to be performed based on their standard procedures. So far, based on the MTT assay, the enzalutamide was cytotoxic to LnCap and RC-77T cells maximally at 60μM across different treatment conditions. In addition, the result from qPCR on kaiso mRNA levels shows the effect on different concentrations of enzalutamide. The treatment with enzalutamide maximally downregulated the expression of kaiso at 30μM concentration and which by 0.2-fold change related to control. Therefore, it implies that kaiso has a correlation with the treatment of the androgen blocker enzalutamide. So, to treat the CRPC kaiso can be potential therapeutic target in both African American and Caucasian American men. Efforts are being made to conduct cut and run and RIME assays based on the MTT and qPCR data.
Among all cancers, lung cancer has the highest mortality rate. In efforts to reverse this statistic, increasing clinical trial participation is necessary. Cancer patients of minority groups in the U.S. have been proven to have significantly less involvement in cancer clinical trials, even though they make up 13.3% of the general U.S. population. Prior research has shown that the obstacles to minority participation in cancer clinical trials are "multilevel within cancer centers". In additional studies, it showed the effects of low patient awareness and biases on the healthcare provider and research staff side. The main goal of this research study is to develop effective intervention methods to increase lung cancer clinical clinical trial engagement among the African American community while also forming lasting impressions on those who will be implementing the methods for the future. In this study, we will survey and record one-on-one interviews in hopes of gaining the participants objective and opinion towards clinical research.

Validated surveys will be distributed to patients through REDCap. Upon completing the surveys, the participants are offered an optional one-on-one interview in which they will indicate their motivations to participate in clinical trials. Interviews will take place on the phone and will be audio-recorded.

There will be a Community Advisory Board that will assess the design and experimentation of this research study. The purpose of UAB O’Neil Community Advisory Board is to offer a community view and provide leadership and structure to the outreach research programs. The diverse group of people will offer feedback and tips on the outreach programs and strategic goals. Clinical trial participation is crucial and increasing minority group involvement is the second step to gaining more accurate results.

**Background:** Metastasis of breast cancer cells is a critical event that drives poor prognosis. Solid tumors such as breast cancer have intratumoral regions of low oxygen tension. This intratumoral hypoxia promotes cancer progression and metastasis. Hypoxia drives immune evasion, alters metabolism of cancer cells, and promotes angiogenesis. Hypoxia Inducible Factor (HIF-1α) is the main transcription factor responsible for cellular signaling influenced by hypoxia. Recent work from our laboratory demonstrated that the number of nucleoli in cancer cells increases in hypoxia compared to normoxia. Nucleoli are sub-nuclear bodies that contain ribosomal DNA and several hundred proteins, and collectively are responsible for ribosome biogenesis. Ribosomes translate proteins and are crucial for tumor cells to support continued growth and cell division, which is also important in supporting cellular pathways for invasion and metastasis. Ribosomes are comprised of ribosomal RNA (rRNA) and ribosomal proteins. RNA Polymerase I is solely responsible for rRNA transcription. Our lab has demonstrated that cells cultured in hypoxic conditions have upregulated RNA Pol I activity. Increased RNA Pol I activity led to upregulated ribosome biogenesis which was evident as increased number of nucleoli, conversely, when HIF-1α is silenced there is a decrease in RNA Pol I activity.

**Hypothesis:** EMT program is upregulated in hypoxic conditions compared to normoxic conditions.

**Methods:** To understand the impact of hypoxia on breast cancer we profiled expression of mRNA in SUM1315 triple negative (TNBC) cells in hypoxia in comparison to normoxia. Using Informatics and Gene set Enrichment (GSEA) we analyzed the data. We used western blotting to confirm the results.

**Results:** Level of EMT driving transcription factor ZEB2 are upregulated in hypoxia.

**Conclusions:** Hypoxia drives EMT in SUM1315 breast cancer cells by upregulating ZEB2.

**Future directions:** ZEB2 protein localizes to the nucleolus. Future investigations will involve studies on effect of ZEB2 levels on RNA Pol I activity and ribosome biogenesis.
Underrepresented minorities (URMs) experience significantly more stress than their white counterparts do. High levels of the stress-induced hormone cortisol increase the risk of many diseases, including breast cancer. The aim of this study was to determine whether gut microbiome dysbiosis due to stress increases estrogen levels by altering the microbial population. We hypothesize that in black women, changes in the gut microbiome due to increased stress may act as a biomarker for breast cancer diagnosis and prognosis. Ten black women (average age of 21 years) and 22 white women (average age of 19 years) completed an interview assessing their stress levels using the Perceived Stress Scale (PSS) and Minority Status Stress Scale (MSSS). Women also provided saliva, nails, and fecal samples to assess short-term cortisol and estrogen levels, long-term cortisol levels, and microbiome composition. Analysis of these samples is underway. We will analyze nail and saliva cortisol and salivary estrogen levels using ELISA. Once microbiome analysis is completed, we will review the results for bacteria containing the ß-glucuronidase enzyme, which produces estrogen. We also plan to correlate PSS, MSSS, cortisol, estrogen, and microbiome results. These data will help us to better understand the risk factors and the role of stress in breast cancer development, which could guide the development of effective preventive measures for young, high-risk women.

**Introduction:** Malignant glioma is one of the most lethal cancers of the central nervous system. Despite the standard treatment options, such as surgical removal of the tumor followed by multi-dose radiation and chemotherapy, the prognosis of this aggressive cancer is extremely poor. Patients rarely survive the 5-year mark following diagnoses. Poor survival is often attributed to the highly heterogeneous nature of glioma and the presence of a highly immunosuppressive tumor microenvironment. Immunotherapy has been shown to extend patient survival in several other cancers with the use of agents such as immune-checkpoint inhibitors, including antibodies targeting cell surface receptors programmed cell death protein 1 (PD-1) or its Ligand, programmed death-ligand 1 (PD-L1). One of the recent promising cancer immunotherapeutic approaches involves the use of engineered oncolytic herpes simplex viruses (oHSV) to directly kill tumor cells and to subsequently stimulate the immune system to attack tumor cells. However, the immune modulating effects of oHSV treatment on glioma cells are understudied. PD-L1 is frequently expressed in glioma cells and its interaction with PD-1 on immune cells prevents immune activation. In this study, we aim to evaluate the impact of oHSV treatment on the expression of PD-L1 in human glioma cell lines.

**Methods:** We will utilize M002, a type of oHSV, to treat human glioma cell lines U87MG and U251. Following treatment with M002 at a dose of multiplicity of infection of 1 for 24 or 48 hours, we will stain these cells with a fluorescently labelled antibody targeting PD-L1. Cells will then be acquired on an AttuneNXT flow cytometer and data will be analyzed using the FlowJo software.

**Results and Discussion:** We expect to see a cell surface modulation of PD-L1 expression in human glioma cells following oHSV treatment. This finding may suggest a potential therapeutic strategy by combining oHSV with PD-L1 checkpoint inhibitors to treat glioma patients with improved efficacy.