CANCER RESEARCH SYMPOSIUM
ABSTRACT BOOK

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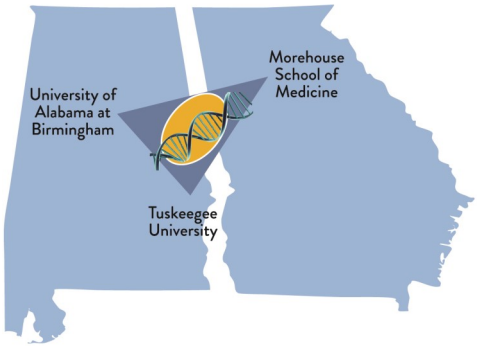
Peachtree City, GA



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A B S T R A C T S

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TABLE OF CONTENTS

Adebayo, Olayinka O., MD, PhD — Poster 1

The role of CXCL13 as a novel and putative driver of RANK-RANKL signaling in multiple myeloma.

Adebayo Olayinka MD; Kaylin Carey; Singh R PhD and Lillard W. James PhD, MBA.

Multiple myeloma (MM) is characterized by the neoplastic proliferation of plasma cells producing monoclonal immunoglobulin in blood, bone marrow, and urine. In 2019, there were approximately 30,770 new cases would be diagnosed and about 12,770 death. MM is a heterogeneous disease; characterizing its molecular phenotypes is important for effective treatment of MM and predicting relapse. CXCL13-CXCR5 signaling has been shown to be involved in malignancy cell homing, adhesion, signal transduction, and calcium flux (osteoclastic activity) in some solid and hematogenous cancers. A recent study has shown "CXCL13 causes a 5-fold increase in RANK ligand expression in Oral squamous cell carcinomas". While CXCL13 stimulation of RANKL in MM has not been studied, we seek to establish the relationship between CXCL13-CXCR5 and RANKL-RANK signaling crosstalk in MM. In other to establish the role of CXCL13 in RANKL signaling, we first analyzed RNA-sequence data from primary MM cases. Patient samples (n=150) were acquired from database Genotypes and phenotypes (dbGap) to evaluate the mRNA expression patterns of CXCL13 and RANKL co-expression and associated genes in MM. A bioinformatics strategy was used to identify genes associated with both CXCL13 and RANKL co-expression signaling and to show protein-protein interactions. Weighted gene co-expression network analysis (WGCNA) identified clusters of genes significantly associated with the molecular phenotypes of MM. Notably, activation of CXCL13-CXCR5 or RANKL-RANK signaling is known to modulate the activation of transcription factors, such as NFAT, FOS, and JUN. Ingenuity pathway analysis (IPA) was performed to analyze upstream regulators, gene interaction networks and canonical pathways. Furthermore, mechanistic studies will be performed using MM cell lines to elucidate the role of CXCL13 in RANKL signaling. Taken together, our data showed CXCL13 co-expression with RANKL signaling networks are significantly expressed and associated with MM pathogenesis, systemic osteolytic lesions, and plasma clonality. This study will provide a better understanding of the heterogeneous nature of MM and the novel interplay between CXCL13 and RANKL signaling in MM.

Ahmed, Amir, BS — Poster 2

Effectiveness of Cancer Education Course on Community Health Advisors (CHAs) in the Black Belt of Alabama

Ahmed, Amir M., BS; Tipre, Meghan, PhD; Richardson, Molly B., PhD; Hardy, Claudia M., MPA; Baskin, Monica L., PhD

Introduction: Lung cancer is the top cause of cancer deaths in the US with an estimated 142,670 expected to die and 228,229 expected new cases in 2019. African Americans, especially those in the Deep South, are disproportionately affected by lung cancer and have greater mortality compared to other racial groups or regions. Early lung cancer detection through screenings is key to reducing mortality. The U.S. Preventive Services Task Force recommends yearly low dose computed tomography scans for individuals at high risk for lung cancer. CHAs were trained to educate members of their community about lung cancer risks, prevention, and screenings. This community-centered approach is intended to reach and educate those individuals at high risk of cancer to seek possible cancer screenings.

Methods: 202 community members were selected for the Alabama Lung Cancer Awareness, Screening, and Education (ALCASE) training. They hailed from seven counties: Jefferson County and six Black Belt counties representing some of the state's poorest or most rural counties with poor access or adherence to cancer screenings. Participants were given identical pre- and post-tests designed to measure their knowledge related to cancer before and after training. Test scores were analyzed using paired sample T-tests to understand how the CHAs' knowledge changed over the training period and to assess the training's effectiveness. Results: Data analysis demonstrated that out of the 139 CHAs who completed both pre- and post-tests, they averaged 20/29 (SD=3.126) questions correct on pre-tests. Average post-test scores were 23/29 (SD=2.317), amounting to a 10% score increase.

Discussion/Conclusion: The slight improvement in scores suggests that CHAs had a modest established knowledge base and thus the training provided limited new information. Additionally, certain questions were widely missed in both pre- and post-tests, possibly due to gaps in training. Regardless, CHAs were duly trained to become better peer educators about cancer risks and prevention. They can now serve as part of a network of individuals working in their respective communities to raise awareness and dispel misconceptions about lung cancer screenings. Increasing lung cancer screenings among those communities could help to drive down the disparities in lung cancer that those populations face.

An, Jie, MD — Poster 3

Development of Knock-in Reporter Tools for Monitoring HOXA9 expression in AML

An Jie; Lu Rui

Introduction: Leukemia such as acute myeloid leukemia (AML) is a deadly disease with uncontrolled proliferation of hematopoietic progenitor cells. HOXA9 is a transcription factor playing important roles in the development of hematopoietic stem cells. In AML, due to a variety of upstream genetic alterations, HOXA9 is overexpressed in more than 50% of AML patients, and has been demonstrated as a strong predictor of poor patient survival. Significantly, many studies have shown that HOXA9 is essential for maintaining leukemic transformation, making HOXA9 an attractive target for therapeutic disruption of AML maintenance. To understand the critical factors sustaining abnormal HOXA9 expression in AML, we aim to establish a reporter cell line for simple, robust and real-time monitoring HOXA9 expression, which will provide as a foundation for high-throughput discovery of key HOXA9 regulators in the future.

Methods: In this project, we took advantage of the CRISPR/Cas9-mediated homologous knock-in system to establish a HOXA9 reporter cell line. We first used polymerase chain reaction (PCR) to amplify three approximate 800-bp fragments including 5' homologous arm (5HA), 3' homologous arm (3HA) of HOXA9, and a P2A-mCherry cassette. The full-length sequence was generated by overlapping PCR and subsequently cloned into a TA vector. Simultaneously, we designed and selected highly effective guide RNAs for constructing the final targeting knock-in vector flanked with selected guide RNA sequence. The knock-in donor vector, along with a Cas9/guide RNA expressing plasmid, were transfected into 293T cells. Genotyping with genomic DNA PCR was performed for proof-of-principle validation of successful HOXA9 knock-in.

Results: We obtained two relatively high-efficiency sgRNA sequences and constructed the final knock-in donor vector. Moreover, the genotyping PCR result showed that the P2A and mCherry DNA has been successfully knocked into the desired endogenous loci at HOXA9.

Conclusion: We successfully constructed a plasmid toolkit for establishing HOXA9 reporter cell lines. This toolkit has been validated in 293T cells. In future, we will establish HOXA9 reporter lines in human AML cells and perform large-scale screening for the discovery of novel HOXA9 regulators.

Banks, Kellon S., MPH — Poster 4

Knowledge and Awareness about Cervical Cancer and Human Papilloma Virus (HPV) Among Women Living in Macon County of Alabama

Banks, Kellon, BS; James, Crystal M., JD, MPH; Heath, John, PhD; Nganwa, David, MPH, DVM; and Abdalla, Ehsan PhD*

Introduction: Despite high screening rates for cervical cancer (CerCancer) in Alabama, Blacks still have higher mortality rates compared to Whites. The purpose of this study was to increase knowledge and intention to have CerCancer screenings (Pap test and HPV vaccination) through administration of pre and post questionnaires pertaining to CerCancer and HPV awareness before and after an educational intervention.

Methods: Pre and post questionnaires were utilized for collection of data before and after a primary educational intervention in Macon County. Descriptive statistics using chi-square and frequency tests were performed for analysis using SAS software. Of the 100 women participants, 86% self-identified as Blacks. About 65% were over the age of 35 and making less than $50,000 per year. 62% currently lived in the Tuskegee community while 34% engaged the community as students/faculty who resided outside of Tuskegee. About 25% of the participants were either married or living with their partner, leaving about 75% of the women to be single, divorced, or widowed. More than 80% were in between their 1st year of college and graduate school with only 40% of them currently working for pay.

Results: The participants who knew what CerCancer was, ever heard of HPV, and ever had an HPV-test improved after the intervention by margins of 9%, 23% and 4% respectively. Participants who ever heard of Pap test had the same knowledge (97%) before and after the intervention. Overall, the participant's knowledge increased following the education-based intervention, however there was a significant improvement level in understanding that CerCancer was caused by HPV infection (38% increased-knowledge), all HPV infections lead to CerCancer (39% increased-knowledge), and CerCancer has decreased in recent years (50% increased-knowledge).

Discussion/Conclusion: Based upon this sample, there is a compelling need for intervention implementations in underserved communities with low rates of CerCancer screenings. Although the participants lacked knowledge in certain areas, the study showed an apparent increase in their knowledge following the intervention. Different intervention methods and health behavior-change frameworks will provide an effective baseline level for CerCancer prevention. The increase of intervention occurrences can increase knowledge and contribute to the lowering of mortality rates.

Keywords: Cervical Cancer, Human Papilloma Virus (HPV), Pap test/Pap smear, HPV test, knowledge, awareness.

Becks, Alahni B. Poster 5

Reviewing and developing web-based weight loss and diet intervention for cancer survivors with a BMI higher than 30

Becks, Alahni; Curtis, Peyton; McKenzie, Jessica, MS; Modi, Bhavan, MS; Denmark-Wahnefried, Wendy, PhD; Pekmezi, Dorothy, PhD

Weight gain has experienced upward trends in the United States and have raised serious public health concerns. These trends have affected the community of cancer survivors and research shows that obesity can increase the probability of cancer recurrence. Lack of exercise and poor diet are two main contributors to the increase of weight gain and decline in health in older cancer survivors. Web-based interventions may be beneficial methods to decrease the growing rate of obesity in cancer survivors. Our approach to this problem is Amplify, a 12-month, web-based intervention for cancer survivors targeting diet and exercise lifestyle changes. Amplify is a three-arm random control trial with two sequential delivery groups and one simultaneous delivery group, with diet, exercise and both, respectively. Designing the intervention involved creating interactional articulate storylines with weekly challenges, daily diet and exercise tips, and a data input system for participants to log their calorie intake, food intake, and weight. Literature reviews provided information about prior studies using similar interventions for cancer survivors. The goal of this web-based intervention is to decrease the BMI of overweight cancer survivors and improve their overall health and diet choices.

Bellamy, Khristlyn, BS Poster 6

Effectiveness of Group Intervention Programs on Weight Loss and Weight Management

Bellamy, Khristlyn, BS

Introduction: The National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) reports that one in three Americans are suffering from obesity. Obesity is defined as having a Body Mass Index (BMI) of 30 or above. Weight loss alone significantly reduces the risk for many diseases; however, losing weight is only half the battle. Many also struggle with *maintaining* the healthy weight, and balancing an active lifestyle with work, family, and other daily responsibilities.

Methods: Primary Care Obesity Management in the Southeast (PROMISE) is a 5 year individually randomized controlled trial comparing the effectiveness of two forms of obesity treatments: the clustered campaign approach and the self-directed method. The clustered campaign approach is an alternative approach to weight maintenance. It includes 16 weeks of interactive, group sessions that share common objectives. Participants in this group will have frequent contact with treatment staff, with the intent of keeping participants motivated and engaged in long term treatment as well. The self-directed approach reflects the current standard of care, and will function as the study's control group.

375 patients, with ages ranging from 21 to 75, and BMI's ranging from 30 to 50 are recruited from 10 primary care practices primarily in the UAB/ Birmingham area and followed for a total of 18 months. We hypothesize that brief, but intensive periods of follow up care may serve to bolster patients' motivation and more effectively minimize the regain of weight.

Results:

Conclusions/Discussion: The overall intent of the project is to make low cost, feasible, evidence based lifestyle interventions for obesity available to healthcare providers and patients throughout the Southeast.

Acknowledgements: Dutton, Gareth, PhD; Glover, Renae; Phillips, Janet

Buckley, Molly, BS — Poster 7

The Effect of Oscillatory Forces in the Progression of Epithelial Ovarian Cancer

Buckley, Molly; Martinez, Alba; Berry, Joel, PhD; Birrer, Michael, MD PhD

Introduction: Epithelial ovarian cancer (EOC) is the most common cause of death among gynecological malignancies. Although the standard treatment results in an 80% response rate, the tumor will often metastasize. Ovarian cancer is subject to mechanical forces that change as the tumor progresses. These forces may be time-varying and may result from externally applied stress to the tumor tissue (arterial pulse pressure, body movement) or growth induced forces. The role of mechanical forces in the development of EOC remain a question to be answered. This work sought to understand the influence of oscillatory tension on two subtypes of ovarian cancer, clear cell and high-grade serous.

Materials and Methods: We used a Flexcell FX-6000T Tension System to apply tensile forces to ovarian cancer cells (luciferase-tagged SKOV-3 [clear cell origin] or OVCAR-4 [high grade serous]) in a 2-dimensional (2D) monolayer. Cancer cells were seeded at 250,000 cells/well in 6-well Flexcell Uniflex Culture Plates - collagen coated flexible membrane plates designed to work with this system - and placed in an incubator to reach confluence for 24 hours. After 24 hours, the cells were strained for 3 days with sinusoidal oscillating tension at 10% elongation and a frequency of 0.3 Hertz, while control samples were placed in the same incubator for the same period of time under no strain. Cells were imaged by bioluminescence (BLI) on day 0 and 3 before harvesting them for transwell migration and invasion assays with 12 and 24 hour timepoints.

Results: Mechanical forces are known to be potent regulators of cancer progression. Our initial data show consistent growth in the strained cells under oscillating strain during the 3 days. Transwell migration assays showed that the cells subjected to mechanical stress developed a more invasive phenotype than the control.

Conclusions: Mechanical forces play an important role in ovarian cancer development. Our preliminary data show that when cells were exposed to oscillating forces, they develop an increasingly invasive phenotype. Further analysis is needed to investigate the expression of epithelial-mesenchymal transition (EMT) markers that may contribute to the growing invasiveness. Next, we will study these same forces in physiologically relevant 3D models, which include extracellular matrix components and cancer associated fibroblasts. Finally, *in vivo* experiments in animal models will increasingly prove the role of oscillatory forces in cancer progression.

Caffey, Jayla M. — Poster 8

Comparison of Psychosocial Wellbeing among African American and Caucasian Metastatic Cancer Survivors

Caffey, Jayla; Bail, Jennifer PhD, RN

Introduction: Approximately 5 million Americans are living with metastatic cancer and are at an increased risk for psychosocial distress (e.g., hopelessness). Hope is vital to life and is essential for effectively coping with illness. Among early-stage cancer survivors, previous studies indicate that African Americans (AA) experience greater cancer burden than Caucasians (CAU). The aim of this study was to compare psychosocial wellbeing between AA and CAU MCS residing in the Deep South.

Methods: MCS were identified via UAB Cancer Registry and I2B2.Using a modified Dilman's method, eligible MCS (>21 years and physician permission to contact) were mailed a survey. Psychosocial wellbeing (i.e. physical and mental wellbeing, anxiety, depression, social isolation, emotional support, and hopefulness) of MCS were assessed via PROMIS® measures. Returned surveys were double-key entered into REDCap®. Data were analyzed using Excel. Descriptive statistics were used to characterize the study sample and instrument scores. Between group difference, between AA and CAU MCS, was examined via independent-samples t-test.

Results: To date, 100 surveys have been returned (AA=18; CAU=82; M_{age}=67 years; $M_{survivorship}$=3 years) with a broad representation of primary cancer sites (breast=23%; prostate=10%; gynecological=16%; colorectal=13%; lung=10%; kidney=11%; other=17%). Mean instrument scores were similar between AA and CAU MCS for physical wellbeing (44.57 vs 44.81), mental wellbeing (48.1 vs 48.46), anxiety (45.76 vs 48.45), depression (44.85 vs 46.77), social isolation (39.96 vs 40.89), and emotional support (39.96 vs 40.89). However, AA MCS reported significantly better feelings of hopefulness than CAU MCS (61.34 vs 55.39, $p>0.01$, $d=0.73$).

Discussion: Findings suggest that AA MCS may be more hopeful than CAU MCS. These preliminary findings warrant further analysis after data collection is complete (an additional 300 returned surveys are anticipated).

Castion, LeKendric — Poster 9

Myristoylated alanine rich C-kinase substrate peptide as a therapeutic in melanoma

LeKendric Castion, Hasan Alrefai, Joshua C. Anderson, Patricia H. Hicks, Nicholas Eustace, Lewis Shi, Christopher D. Willey

Introduction: Melanoma is the most fatal of skin cancers in the United States. Compared to basal and squamous cell carcinoma, melanoma is significantly more aggressive both in rates of metastasis and resistance to standard therapies such as immunotherapy, chemotherapy, & other targeted therapies. Recent studies have shown that a peptide mimetic of the MARCKS effector domain (ED), MED2 acts as a tumor suppressor, inhibiting lung cancer cell line growth. This study investigates the efficacy of MED2 in targeting melanoma cancer cell growth with or without radiation treatment.

Methods: To measure the effect of MED2 (Anaspec) pre-radiation, 1000 B16-F10 cells were plated 24hrs prior to MED2 treatment (2μM & 7.5μM). Cells were irradiated at: 0gy, 2gy, & 5gy for 30 and 60 minutes. Viability was quantified with CellTiter-Glo, 120hrs post-radiation. Similarly, to measure the effect of MED2 post-radiation, 500 B16-F10 cells were plated 24hrs prior to cell irradiation at: 0gy, 2gy, & 5gy for 30 & 60 minutes. Followed by the CellTiter-Glo reading as before.

Results: MED2 treatment was toxic in a dose-dependent manner to melanoma cell lines, and in combination when used prior to irradiation, with a monotherapy IC50 near 25μM. Figure 1 illustrates that the timing of MED2 treatment is important, with treatment at 30 mins post-radiation showing a greater reduction of B16-F10 cells than MED2 treatment at 30 mins pre-radiation & 60 mins post-irradiation.

Discussion/Conclusion: MED2 decreases B16-F10 melanoma cancer cell line viabilities pre & post-radiation. Interestingly, MED2 given post-radiation proves more effective at times closer to irradiation. This study highlights the potential for MED2 as a therapeutic tool in melanoma and emphasizes the importance of treatment when agents are used in conjunction with radiotherapy.

Chandora, Akshay N., BS — Poster 10

Implementing a Precision Cancer Medicine Protocol at a Safety Net Hospital

Akshay Chandora; Latrisha Horne; Eddie Stanley; Sha-hanna Saffold; Jamarkus Watson; Jada Johnson; Kaylin Carey; James W. Lillard, Jr.

Introduction:African Americans have higher cancer development and death rates in comparison to Caucasian counterparts. Increasing participation in clinical research related to precision medicine will alleviate such disparities. Limited genomic and transcriptomic data for African Americans (AA) results in poor biomarker research, drug target research, and thus inadequate treatment options. Most genetic cancer sequence data was collected at cancer centers that do not serve many AA. It is crucial to implement data from tumor sequencing efforts at cancer centers serving diverse groups. Here we describe such a solution at Grady Memorial Hospital (GMH) which serves > 95% AA cancer patients in Atlanta. Total Cancer Care protocol (TCCP) allows researchers to incorporate factors of genetics, environment, family history, and socioeconomic aspects via tissue and health records analysis. This work is vital for comprehensive treatment development.

Methods: After relevant information was presented and any questions were answered, patients signed the consent form. Consented patients were provided with a copy of the form and a callback number. Buccal swabs, saliva, and stored tissue samples from previous biopsies were collected from patients and sequenced at offsite facilities.

Results: Since the start of this study in April 2019, we consented 68 out of 77 cancer patients and survivors, which represents a consent rate of 88%. Comparatively, the Moffitt Cancer Center in 2015 had a consent rate of less than 30% possibly due program size, causing more attrition (of 426,284 cancer patients, 120,887 were consented). Continued improvements to TCCP protocol will allow for consent of 2,020 cancer patients by December 2020.

Conclusion: Unlike similar programs, TCCP provides CLIA-certified genetic profiles for consented patients, which can aid in more informed medical decisions. The patient genomic and health records information populating such databases will allow researchers to select proper subjects for their clinical trials, increase patient participation and improve outcomes. Patient and provider education are essential to improving consent rates. The importance of precision medicine and TCCP was emphasized to providers who were critical in the initial stages. Future scaling efforts will require such efforts and will bring this protocol within private or public medical centers serving AA populations across the southeast.

Chowdhury, Kawsar U., MS — Poster 11

Determination of Kaiso Protein-Protein Interaction

Chowdhury, Kawsar; Lin, Huixian, PhD; Karanam, Balasubramanyam,Ph.D; Yates,Clayton,PhD

Purpose: The POZ-ZF [Poxvirus and zinc finger (POZ)] transcription factor Kaiso has role in diverse biological processes in multiple human cancers including prostate, lung, colon, breast cancers. These occur mainly through its direct regulation of gene expression. Recent findings from our lab and others have demonstrated that the N-terminal, which is the protein-binding region of Kaiso interacts with different types of protein like p53, HDAC (Histone deacetylase), AR (Androgen Receptor) (refs). However, the role of Kaiso in the protein-protein is not extensively explored. Therefore, the purpose of this study is to investigate the interaction of Kaiso with these proteins.

Methods: To determination the binding of p53 and HDAC with Kaiso we used the Glomax discover system of Promega where one protein, tagged by Nanoluc Luciferase, acts as the energy donor (MDM2,pNLF1-N-Kaiso, HDAC6) and another protein fused with Halotag 618 Ligand, acts as the energy acceptor (p53,pHTN-Halo-Kaiso).Each of these vectors was cloned into HEK293 (Human embryonic kidney) cells, which do not express Kaiso, respectively. Protein-Protein interaction was measured by bioluminescence.To validate the interaction of Kaiso in a cancer model, each vector was transfected in MDA-MB-231(Human breast cancer cell line with high expression levels of Kaiso)cells, as described above. In both cases the protein pair of p53, MDM2 was used as the positive control.

Results: Our preliminary data revealed that the interaction of the positive control pair protein P53, MDM2 is very high in both cell lines but when we compared it with our desired protein pairs, in HEK293 cells the interactions demonstrated the following sequences of strength- p53, MDM2> pHTN Halo Kaiso,pNLF1-N Kaiso>pHTN Halo Kaiso ,HDAC6>P53,pNLF1-N Kaiso. When we measured the interactions using the MDA-MB-231 cells, using the same sequences the interaction of P53 MDM2, pHTN Halo Kaiso, and pNLF1-N Kaiso is higher in HEK293 cells whereas the interaction of pHTN Halo Kaiso, HDAC6 and p53,pNLF1-N Kaiso is higher in MDA-MB-231 cells. Furthermore, if we used an inhibitor to Kaiso, the HDAC6 interaction decreased in HEK293 cells, and MDA-MB-231 were more sensitive to the inhibitors.

Discussion: Taken together our findings reveal that Kaiso-Kaiso interaction is stronger in relation to other proteins (HDAC6,p53).However, Kaiso-HDAC6 interaction is also strong, and can be inhibited through direct targeting. Further, determination of Kaiso and its interaction with key proteins would result in several innovative approaches to determine new method to inhibit these interactions which will result in great innovation for the treatment of cancer.

Flanary, Victoria L. — Poster 12

Crosstalk with astrocytes establishes glioblastoma's edge

Victoria Flanary; Daisuke Yamashita; Hai Yu; Soujun Zhang; Davide Botta; Yeri Lee; Heejin Cho; Xiaoxian Guo; Saya Ozaki; Mu Gao; Sadashib Ghosh; Chaoxi Li; Joshua D. Bernstock; Ahmed Ibrahim; Shinobu Yamaguchi; Svetlana Komarova; Soniya Bastola; Takeharu Kunieda; David Crossman; Zhenglong Gu; Jeffrey Skolnick; Nicola Zamboni; Frances E. Lund; Do-Hyun Nam; and Ichiro Nakano

Prognoses for glioblastoma multiforme (GBM) remain poor despite aggressive treatment in part due to tumor invasion into surrounding brain tissue, as these cells may remain after surgical resection and facilitate GBM recurrence. The brain tumor microenvironment is thought to play a role in promoting tumoral heterogeneity, resulting in different GBM spatial phenotypes (e.g. edge vs. core). We hypothesized that the brain tumor microenvironment may influence the development of the GBM edge phenotype and affect GBM progression and recurrence. Histopathological and molecular differences between GBM edge and core tissues were analyzed via MRI-guided biopsies, immunohistochemistry, and RNA sequencing. These assays identified CD38 as a crucial molecule in the development of the edge phenotype, namely in driving the interaction between GBM edge cells and neighboring astrocytes. Using GBM derived edge and core clones as models, we found that CD38 is highly expressed in edge clones and may induce mitochondrial dysfunction in GBM edge clones to maintain the phenotype, and crosstalk between GBM cells and astrocytes occur in a CD38-dependent manner. By revealing the role of CD38 in the formation of the GBM edge phenotype, this study identifies CD38 as a potential target for clinical treatments that aim to prevent GBM progression and reduce the risk of recurrence.

Hakim, Martina J. Poster 13

Racial Disparities in Contraceptive Use and the Likelihood of Testing Positive for High-Risk Human papillomavirus (HR-HPV) Infections

Hakim, Martina; Badiga, Suguna, PhD; Piyathilake, Chandrika, DDS, MPH, PhD

Background: Infection with high risk human papillomaviruses (HR-HPVs) is the causative factor for developing cervical intraepithelial neoplasia (CIN), pre-cancerous lesions for developing cervical cancer. In the United States (US), 79 million Americans are currently infected with HPVs and every year there is an estimated 14 million new cases of HPVs. Several risky sexual behaviors including, age at first intercourse, use of douche and contraceptives, parity and history of other STDs have been studied in relation with HR-HPV infections, but the results have been inconsistent. Further, to our knowledge, no comprehensive studies have been conducted to evaluate racial differences in the relationship between those factors and risk of being infected with HR-HPVs in the US.

Purpose: The purpose of this study was to determine the racial differences in the relationship between risky sexual behaviors and risk of being infected with HR-HPVs.

Methods: The study included 852 women (African American (AA), n=434, Caucasian American (CA), n=414) who were tested for HPV infections (HR-HPV positive, n=667, negative, n=181) using Roche linear array assay who were diagnosed with higher grades of CIN (CIN2+) or ≤CIN1. A questionnaire was used to obtain demographic, lifestyle and sexual behavior related information. Information regarding fruit and vegetable intake was obtained using Block fruit, vegetable and fiber screener. Unconditional Logistic regression models stratified by race were used to test the associations.

Results: We observed racial differences in the determinants of HR-HPV infections. AA women who were <24 years and users of hormonal contraceptives were more likely to test positive for HR-HPVs (OR=1.70 P=0.0410 and OR=1.80 P=0.0441). Among CA, none of the risk factors were significantly associated with the likelihood of testing positive for HR-HPVs.

Conclusion: Younger AA women were more likely to be HR-HPVs positive. The plausible reasons for this observation could be as follows: lower socio-economic status related factors such as lower exposure to sex education, lack of HPV knowledge, lower immune response due to poor nutritional status and lower likelihood of clearance of HR-HPVs. It is likely that AA women who used contraceptives may be practicing risky sexual behavior that increases their risk for HR-HPV infections.

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Hakim, Ornin Poster 14

MP vs SP

Hakim, Ornin; Fang, Andrew, MD; Rais-Bahrami, Soroush, MD

Introduction: Over the last 10 years, robotic prostatectomies have been performed via multi-port (MP) robot systems including the da Vinci XI robot. Recent approval of the da Vinci SP Surgical System (Intuitive Surgical, Sunnyvale, CA) by the Food Drug Administration had led to several series describing safety and feasibility of a single-port robot assisted radical prostatectomy. (SP has recently been the aim of this study is to compare perioperative outcomes between multi-port robots (MP) and single port robots (SP) systems in patients undergoing radical prostatectomy

Methods: Patients who underwent robot assisted radical prostatectomies using a MP or SP robotic system at our institution between October 2019 and June 2019 were retrospectively reviewed. Both operative systems were carried out by two high-volume robotic prostate surgeons. Patient demographic, operative, perioperative course, and pathologic results were analyzed using t-test and chi-square statistical measures.

Results: In a cohort of 91 patients who underwent robot assisted radical prostatectomies, 42 (46.15%) had SP robotic surgery and 49 (53.84%) had MP robotic surgery. Average operative time in the SP cohort and MP were similar between the two groups (SP average, MP average, respectively, p = _). Additionally, blood loss was also noted to be similar between the SP and MP cohorts (SP average, MP average, p = _). However, the operative time in the SP cohort was ___ than the MP cohort (p= _).

Discussion / Conclusion: Our study is one of the first cohorts comparing the perioperative outcomes between MP and SP approaches for radical prostatectomies. Our results demonstrate that the SP approach have comparable perioperative outcomes, with the SP approach having a potentially faster operative time.

Disclaimer: Official statistics are still pending.

Hernandez, Natalie, MPH, PhD — Poster 15

The Voices of Cancer Survivors on Clinical Trial Participation at a Safety-Net Hospital

Natalie D. Hernandez, PhD, MPH; Raegan W. Durant MD, MPH; Dexter L. Cooper MPH; Desiree Rivers, PhD, MSPH; Ebony Respress, MSPH; Nedra Lisovicz PhD, MPH; Brian M. Rivers PhD., MPH

Introduction: Clinical trial participation is essential to the progress of optimizing cancer care outcomes; however, there is a paucity of African American (AA) participation in cancer clinical trials (CCTs) resulting in significant gaps in treatment efficacy. There has been a robust amount of research on ways to increase AA participation in CCTs, but few studies have examined AA recruitment at safety-net hospitals. The objective of this study is to utilize a multilevel, qualitative approach to assess the clinical and non-clinical facilitators and barriers to AA participation in CCTs at a safety-net hospital from the perspective of AA cancer survivors.

Methods: Study participants (n=16) were recruited from a cancer center at a safety-net hospital in the southeastern U.S. Eligible participants were individuals who: 1) self-identified as AA; 2) were 18-75 years old; 3) spoke and read English; 4) diagnosed with cancer; 5) had no functional limitations that would interfere with participation in a 60-minute focus group; and 6) be capable of providing written consent for study participation. Focus groups were digitally recorded and transcribed. Data was coded and analyzed to identify the most prominent themes representing unifying ideas and concepts.

Results: *Theme 1: Understanding of Cancer Clinical Trial Terminology (Barrier).* For some of the participants this focus group session was the first conversation they had where they were able to discuss their cancer diagnosis with other survivors. Participants also expressed confusion between clinical trials and treatment; many did not know the difference between the terms. There were instances where patients used the terms incorrectly, especially during discussions about willingness to participate in trials.

Theme 2: Perceptions of Cancer Clinical Trials (Barrier). Participants may have heard of clinical trials but did not know what a cancer clinical trial entailed. Once a clinical trial was explained participants expressed that they may have taken part. Some indicated that no medical professionals discussed a clinical trial or recruited them to participate. Participants who were knowledgeable about CCTs expressed that they were ineligible for the trial, although ineligibility also seemed to be confusing for them. Most reported receiving information from pamphlets.

Theme 3: Role of Patient Navigator (Facilitator). In general, participants expressed trust in their physicians, particularly for medical information; however, some preferred resources and information from a patient navigator. All agreed that they would be willing to work with a patient navigator and saw the patient navigator's role as providing social support and as a resource. Participants preferred a knowledgeable patient navigator that had cancer experience.

Conclusion: Including cancer patient navigators as part of the treatment team staff may help traverse potential barriers to CCT participation, and ultimately increase the number of AAs diagnosed with cancer participating in CCTs.

Hudaib, Sami, BS — Poster 16

Association of CCR9-CCL25 in Non-Small Cell Lung cancer and racial disparities

Hudaib, Sami; Mir, Hina; Singh, Shailesh

Lung cancer is the leading cause of cancer-related deaths irrespective of sex, especially Non-small cell lung cancer (NSCLC). Poor patient outcomes are due to the lack of precise biomarker, which impacts early detection. First line treatments have shown a race-specific gap in clinical outcomes in African Americans (AA) in comparison to Caucasian Americans (CA). Our research groups have shown that the CC chemokine and its natural ligand CCR9/CCL25 have been overly expressed in cancers, including the NSCLC cell lines of Lung Adenocarcinoma (LAC) and Squamous Cell Lung cancer (SCLC). In this study, a bioinformatic mRNA expression analysis was conducted using TCGA and Oncomine data on the UALCAN database comparing values in currently used screening biomarkers (ALK, EGFR, and KRAS) to the CCR9/CCL25 axis in CA and AA populations. We observed that overall CCR9 mRNA expression was lower NSCLC than the control group. However, mRNA of CCL25, which is the only natural ligand of CCR9 was significantly higher in both LAC and SCLC. CCR9 mRNA in AA and CA was lower than the controls in LAC and statistically low in SCLC. Whereas CCL25 mRNA was significantly higher in both LAC and SCLC in these groups. The mRNA expression of ALK, KRAS, and EGFR was inconsistent when data was compared to different races in NSCLC. Our current data analysis shows the CCR9/CCL25 axis as a novel and effective biomarker for early NSCLC, which has potential to address disparity.

Kagulire, Johnpaul, MS — Poster 17

Knowledge, attitudes and perceptions of cancer and obesity among community leaders in Macon and Bullock counties of Alabama

Kagulire Johnpaul, MSc; Dawkins Norma, PhD

Introduction: Cancer is the second leading cause of death in the US and is responsible for an estimated 9.6 million deaths. In Alabama, the incidence rate, 445.3 is higher than the national average, 441.2, and it is further estimated that there will be 27,830 new cases with over 10,000 deaths. Almost a third of cancer deaths are due to the five major leading behavioral and dietary factors namely: high body mass index, low fruit and vegetable intake, physical inactivity, tobacco, and alcohol use. The high obesity rates in the region have been associated with rising incidence of chronic diseases particularly cancer. Although several interventions have been conducted to address the obesity and cancer problem, the two conditions still disproportionately affect African Americans more than their Caucasian counterparts. Therefore, this study aims at understanding the knowledge, attitudes, and perceptions about cancer and obesity among African American community leaders in rural Alabama.

Method: The study is cross sectional and data is being collected from community leaders using a validated survey instrument.

Conclusions: Understanding of these issues will be of vital importance in informing policy as well as future efforts to more efficiently and effectively implement cancer and obesity programs that impact African-Americans that are at greatest risk in rural Alabama communities. Acknowledgements: Appreciation goes to my advisor, Dr. Norma Dawkins, Dr. Richard Whittington, Tuskegee University extension Department and the Tuskegee University - U54 CA118623 grant for supporting this study.

Karanam, Balasubramanyam, MS — Poster 18

Targeting ubiquitin receptor ADRM1 for the treatment of quadruple negative breast cancer

Balasubramanyam Karanam; Ravi Anchoor; Richard Roden; Clayton Yates

Quadruple Negative Breast cancer (QNBC) is a subtype of Triple Negative Breast Cancer (TNBC) with loss of Androgen receptor (AR). We determined the expression of AR and its relationship to breast cancer subtypes, using Gene Expression Omnibus (GEO) profiles that contained racial and clinical outcomes data totaling 1061 patients. Expression of the AR protein level was confirmed in an additional multi-institutional cohort of 197 breast cancer patients, for a total of 1258 patient evaluated. Relative to White women, African American women had higher percentage (81%) of AR-negative tumors, and, for both races, AR-negative tumors correlated with the basal subtype, a shorter time to progression, and worse overall survival (OS) compared to White women. Currently available treatments are unable to eradicate metastatic breast cancer (TNBC and QNBC), and median survival for these patients is only 2-4 years. Improving the survival rates for metastatic disease has been the subject of intense investigation, and new agents and strategies are actively being evaluated. Targeting the UPS (Ubiquitin-proteasome system) with small molecules can an effective can cancer therapy. We have developed an orally-available proteasome inhibitor bis-benzylidine piperidone (RA190) that binds to the ubiquitin receptor RPN13/ADRM1 on the 19S regulatory particle of the proteasome and directly kills cancer cells by triggering proteotoxic stress. The objective of this study is to investigate the expression of ADRM1 in QNBC patients and target ADRM1 with RA190 for the treatment of QNBC., We used forest plot analysis of TCGA patient samples to determine the expression of Ubiquitin receptor ADRM1, and invitro assays to find the sensitivity of RA190 against AR-positive and AR-Negative breast cancer cells. Our results indicate that ADRM1 is significantly elevated and in QNBC breast tumors of African American patients. Our invitro data indicates that, AR-positive and AR-Negative breast cancer cells have differential sensitivity in IC50 values to inhibitor RA190. Our findings will advance our knowledge of vulnerable pathways in QNBC/TNBCs optimal control and reveal if this mechanism is more likely to occur in African American patients.

Keller, Joycelyn, BS — Poster 19

Something Old, Something New: Evaluating the effects of gentrification on existing community health and cancer prevention

Keller, Joycelyn; Hernandez, Natalie PhD, MPH; Rivers, Brian M., PhD, MPH

Introduction: While cancer mortality is declining for most cancers among all groups, minorities, particularly, African Americans, continue to suffer disproportionately. Recent studies suggest, 42% of cancers could be prevented based on modifiable risk factors. The adoption of healthy behaviors, inclusive of physical activity, plant-based nutrition, limited alcohol consumption, and tobacco cessation, have been associated with a decreased cancer risk. However, recent studies suggest that individual ability to adopt healthy behaviors is not only predicated on their own volition but is adaptable to where they live, work, and play. In areas predominantly populated by minorities, residents commonly encounter gentrification, the process by which higher income households displace lower income households, changing the essential character and flavor of the neighborhood. Anticipated implications of gentrification include weakened social support, exacerbating health issues, and ultimately displacement. The purpose of this study is to better understand the role and impact of gentrification, a social determinant, on cancer prevention/adoption of healthy behaviors among minorities and medically underserved populations.

Methods: A convenient sampling strategy was used to identify study participants from community workshops hosted by the City of Atlanta planning efforts, Neighborhood Planning Units, and residents of communities being gentrified. We conducted 5 key informant interviews with stakeholders who reside or work in areas being gentrified. All research staff received interview training supervised by study investigators. Interviews were recorded on digital devices, transcribed and analyzed using NVIVO 11 software.

Results: Representative themes from stakeholder interviews were determined using elements of grounded theory, inter-coder reliability, and thematic saturation and will be presented.

Conclusions: The findings of this study will increase understanding on the impact of gentrification on cancer prevention. Further evaluation supposes that correlations between gentrification and specific types of cancer prevention will be more prevalent based on the minority population and gender groups dominating the affected geography. The findings will inform further systematic exploration into gentrification and health outcomes on a broader scale. Continued and completed research may lead to recommendations that city development utilize qualitative analysis of a community's health prior to development in effort to strategize initiatives that are more equitable to existing community stakeholders.

Kilian, Riley — Poster 20

Markers of adiposity are associated with the SNP72 genotype in women with breast cancer

Kilian, Riley; Behring, Michael, PhD; Manne, Upender, PhD

Background: Population-based studies show that single nucleotide polymorphisms at codon 72 of the *p53* gene (SNP72) are linked to breast cancer risk, high adiposity, and disrupted glucose metabolism. In the present study, we examined the relationship between SNP72 and body mass index (BMI) and serum lipid markers to determine if the dual functional roles of *p53* as a metabolic regulator and tumor suppressor are associated with breast cancer in African American (AA) and Caucasian (CA) women.

Methods: Primary tumor tissues were collected from a cohort of 158 breast cancer patients who underwent surgery at UAB hospital during 1988-2012 and were genotyped for SNP72 of *p53*. Clinicopathological, demographic, and other variables, including BMI, blood lipid markers (triglycerides, Tg; high-density lipoprotein, HDL; low-density lipoprotein, LDL; and complete cholesterol), and type II diabetes status at diagnosis were collected from UAB electronic health records (EHRs) and patient charts. Follow-up information was obtained from the UAB Tumor Registry and EHRs. For this cohort, the genotype status of SNP72, which was previously evaluated was used. The outcome of interest was death due to breast cancer. The chi-square and Kruskal-Wallis association tests were used for categorical and continuous variables, respectively. Kaplan-Meier and Cox methods were employed for estimations of survival.

Results: In general, measures of BMI and serum lipids exceeded healthy levels in this population. Almost all AA women were obese (BMI >30). The levels of Tg were higher and HDL levels were lower for all women with the Arg/Arg phenotype than those with other SNP72 phenotypes (Arg/Pro and Pro/Pro) (p-values 0.006 and 0.001, respectively). However, the levels of complete cholesterol and LDL were not associated with SNP72 and patient race/ethnicity. CA women (n=5) with the Pro/Pro phenotype had higher cholesterol (p-value 0.07) and LDL (p-value 0.01) levels than AAs with the Pro/Pro phenotype. Women of both racial groups who were obese and exhibited the Arg/Arg phenotype had worse cancer-specific survival (log-rank, p-value 0.04).

Conclusions: Our preliminary results suggest that the Arg/Arg phenotype in SNP72 of *p53* is associated with high BMI, high Tg, low HDL, and decreased survival of women with breast cancer.

Kovac, Stefan — Poster 21

IDH1 Mutant Epigentically Modifes Tumor Supression in Secondary Glioblastomas

Kovac Stefan, Huafeng Wang, Xiaoxue Ke, Stan Han

Introduction: Low grade glioma and secondary glioblastomas are known to have an early mutation in Isocitrate dehydrogenase 1 enzyme (IDH1), a metabolically critical enzyme of the citric acid cycle. Normal IDH1 catalyzes isocitrate to α-ketoglutarate (α-KG); presence of mutant IDH1R132H creates oncometabolite D-2-hydroxyglutarate (D2HG) from α-KG. Structurally similar, D2HG and α-KG both competitively interact to alter α-KG-dependent dioxygenase activity. α-KG-dependent dioxygenases catalyze a number of interactions, some which are relevant in DNA methylation. This project examined acute effects of D2HG in p53 activation.

Methods: Plasmids with R132H IDH1 mutation and control, both with Red Fluorescence Protein (RFP), were obtained and transformed into One Shot™ Stbl3 bacteria. The plasmid DNA was isolated and cotransfected with psPAX2 and pMD2.G plasmids into 293T packaging cells. 2 separate Lentiviruses containing mutant IDH1 and vector control were obtained and infected IP6 neural progenitor cells. Cell protein lysate was collected at 24, 48, and 72 hours post infection. Protein samples were isolated and probed by western blot for p53, pp53, p21, RFP, and GAPDH markers.

Results: Relative to control, increased p21 expression was observed in both vector and mutant cells across all time points. Vector p21 expression levels remained relatively constant for 24-72hr. Mutant samples increased p21 expression from 24 to 48hr, and then decreased thereafter at 72hr. Knowing that p21 reflects p53 activity, p21 activation encouraged a retest for p53 and pp53 markers. Additional Western blots will indicate presence of these proteins.

Discussion/Conclusion: IDH1R132H has been an attractive target for continued research as vaccines and specific inhibitors are currently in trial. We will continue to examine IDH1R132H's effects on tumor suppression via triplicated experiments to further quantify these results.

McDaniel, Bailey — Poster 22

Inhibition of epidermoid carcinoma using TLR-4 inhibitor, TAK-242

Bailey McDaniel; Monica Lewis; Mohammad Shadab; Amy Jasani; Craig A. Elmets; Nabiha Yusuf

Purpose: Non-melanoma skin cancer represents the most frequent cancer in the United States. It is estimated that more than 3 million people will be diagnosed with non-melanoma skin cancer each year. Toll-like receptor 4 (TLR4) has been recently linked to multiple cancers, including skin cancer. The purpose of this study was to evaluate if inhibition of TLR4 using a small molecule inhibitor, TAK-242, had any anti-cancer effects in epidermoid carcinoma (A431) cells.

Methods: The MTT assay was performed to determine the number of viable cells. The colony formation assay and the spheroid formation assay was performed in order to assess proliferative ability of the cells,. The scratch assay was performed to monitor migration ability of cells, whereas a Matrigel invasion assay was performed to assess the number of cells with the ability to invade. The ROS detection assay was performed in order to monitor the presence of reactive oxygen species.

Results: We found that TAK242 decreased cell viability of A431 cells at doses that were non-toxic to normal cells. Treatment with TAK242 caused a dose-dependent reduction in proliferation, as indicated by decreased colony and spheroid formation in A431 cells. TAK242 treatment also inhibited migration and invasion of these cells. We found that TAK242 treatment resulted in generation of reactive oxygen species, which might be a mechanism of TAK242-mediated cell death in these cells.

Discussion/Conclusion: In summary, TAK242 treatment exhibited anti-cancer effects against human epidermoid cells, and may be important in discovering novel therapeutic options for non-melanoma skin cancer.

These results suggest that TAK-242 should be investigated further.

McLarty, Kristiana, BS — Poster 23

Precision Oncology and BRCA1 Associated TNBC

Kristiana McLarty; Jingyao Xu; Yunlong Qin; Vaishali Reddy ; Yonte Burnam ; Anya Bazzell; Pooja Aysola; Kirat Sandhu; Shivani Punna; Sothivin Lanh; E. Shyam P. Reddy and Veena N. Rao

Background and Significance: Majority of young AA women with BRCA1 mutations have Triple negative breast cancer (TNBC) with an aggressive phenotype. Currently there is no targeted therapy nor early detection biomarkers for TNBC. Many BRCA1 missense mutant alleles, termed variants of uncertain significance (VUS) are difficult to classify as benign or malignant. Therefore, for a woman who carries a BRCA1 VUS allele, the risk of developing TNBC is unknown. AA women have a higher frequency of BRCA1 mutations and VUS.

Hypothesis: This work is based on the hypothesis that BRCA1 is a tumor suppressor gene and its RING domain can harbor several mutations some of which are driver mutations causing loss of BRCA1 function resulting in TNBC and others can be passenger mutations like BRCA1.

Results and Conclusions: We tested this hypothesis by introducing C61G, K109R and I26A mutations into TNBC cells and studied their growth inhibitory activity using colony suppression /scratch migration assays. Our results demonstrate for the first time that BRCA1 I26A to be like BRCA1. I26A mutant associates with Ubc9, has homologous recombination (HR) activity, lacks E3 Ubiquitin ligase activity and inhibits growth/migration of BRCA1 mutant TNBC and sporadic TNBC cells unlike K109R and C61G mutants. Clinically, the ability to predict which of these mutations are driver mutations that can result in TNBC offers unprecedented prospects for early detection and cancer prevention. This is the first study demonstrating the physiological link between Ubc9 binding, HR activity, loss of BARD1-dependent E3 Ubiquitin ligase activity and growth/tumor suppression of I26A mutant BRCA1 protein in TNBC cells. BRCA1, by turning off or on Ubc9 binding, regulates growth of TNBC cells. This study will accelerate precision medicine and reduce cancer health disparities in health outcomes.

Meeker, Brock, BS — Poster 24

Genistein overcomes the multidrug resistance in pancreatic cancer cells

Brock W. Meeker, Santosh Kumar Singh, and Rajesh Singh

Pancreatic cancer (PC) is the fourth most common lethal malignancy. Although numerous efforts have been made to optimize chemotherapeutic regimens, the 5-year survival rate remains 7.7%. The major reason for the failure of chemotherapy in PC is due to drug-resistant. Gemcitabine (GEM) is well-known chemotherapy, frequently used as first-line treatment for the patient with advanced PC. The development of GEM resistance leads to a low response to chemotherapy and remains a significant limitation to its use. ATP-binding cassette (ABC) transporters are common of interest in cancer therapy, as they play a crucial role in the efflux and/or influx of drugs across the cell surface; therefore, they play an important role in the multidrug resistance (MDR) of cancer cells. Due to drug resistance of PC cells, treatment often demands high doses of chemotherapy; therefore, patients experience severe adverse side effects. Thus, there is an increasing demand for more effective therapeutic agents to combat PC cells, such as natural compound which affects multiple targets including MDR, have limited side effects, and are less toxic. In this study, we analyzed the GEM-induced drug resistance of the PC cell lines (AsPC-1, and BxPC-3) and compared upon treatment with a natural compound, Genistein. Genistein, an isoflavone soybean isolated from the Leguminosae (*Fabaceae*), it has anti-oxidation, anti-proliferation, anti-cancer activities which possess the cytotoxicity against various cancer, including PC. In particular, upon a treatment, the response of ABC transporters was examined, which play a critical role in cancer cell drug resistance. The effects of the treatments were analyzed through an MTT assay and qRT-PCR. The individual IC_{50} value was calculated; the optimal treatment concentration (IC_{50} value) of genistein was found 573 µM, and 340 µM at 48h for AsPC-1, and BxPC-3 respectively. Furthermore, we observed that treating PC cells with genistein exhibited a reversal of ABC-transporter markers thereby limiting the MDR phenotype of the cancer cells. Overall, our results have revealed that genistein is applicable as a promising agent for overcoming GEM-resistant PC and might allow a reduction in GEM doses while maintaining the therapeutic effect in PC patients.

Mitchell, Caris, BA, MS — Poster 25

Disparity in Incidence and Severity of Peripheral Neuropathy in Multiple Myeloma

Mitchell, Caris, MS; Innis-Shelton, Racquel, MD

Introduction: Several chemotherapeutic agents utilized in the treatment of multiple myeloma (MM) can cause mild to severe peripheral neuropathy (PN). Among these drugs, the proteasome inhibitor, bortezomib, and immunomodulators like thalidomide, are most commonly linked to the development of PN. It has been our clinical observation that African-Americans (AAs) treated with these anti-myeloma agents suffer more intense and sustained PN compared to other ethnic groups. We hypothesize that there is a disparity in incidence and severity of PN in patients receiving treatment for MM.
Methods: To test our hypothesis, we will conduct a subgroup analysis of patient reported symptoms captured in the dataset of an existing IRB approved MM protocol at UAB [(UAB#1738) IRB -300000121]. Cases of PN will be analyzed based on patient survey entries, physician documentation, and a medication list review. These reports will then be stratified according to age, race and ethnicity, PN incidence and severity, chemotherapy exposure, the year of MM diagnosis, and other comorbidities.
Results/Conclusion: There is limited data reported on the existence of ethnic disparity in MM associated neuropathy. Prior to designing our subgroup analysis, a PubMed search using the keywords African American, multiple myeloma, disparity, and peripheral neuropathy revealed only three results further indicating that this is an area that is not well understood or studied. We anticipate that we will find a higher grade PN in AA MM patients, and that age and diabetes, will be confounding variables. Final results of the query are pending.

Mohamedelhassan, Rania, MD — Poster 26

The Role of CD206 Targeted Peptide in Tumor Associated Macrophages

Mohamedelhassan Rania MD, Janyes Jesse Ph.D, Yates Clayton Ph.D

Purpose: Cancer is the second leading cause of new cases of death in the United State in 2019, With cancer cells induce an immune response including the infiltration of Tumor-associated Macrophages (TAMs). TAMs are linked with poor prognosis of cancer as (M2 like -TAMs) promote tumor progression by enhancing angiogenesis, metastasis and by preventing anti-carcinogenic immune response. Immune defense Regulator (IDR) such as RP-182 are synthetic host defense peptides that selectively induces a conformational switch of the mannose receptor CD206 expressed in macrophages displaying M2-like phenotype. Our Lab has shown that the expression of CD206 receptors is high in (M2-like TAMs) in contrast with the low expression of the CD86 receptors (M1 marker), Our previous studies have demonstrated that (IDR) peptides possess the capability to -re-program (M2 like TAMs) to M1-like phenotype macrophages under unique polarization conditions to enhance CD206 expression. However, the level of CD206 required for IDR activation of the CD206 receptor on M2 macrophages is not well understood.

Method: To validate the CD206 expression, murine BMDM (Bone Marrow Derived Macrophages) were extracted and polarized into M2-like Macrophages in comparison with M1-like Macrophages. Polarization of the two phenotype groups of Macrophages was induced using IL-4 and IFN-G for M1 and M2 like phenotypes respectively. Markers for M1 and M2 Macrophages (CD206, CD86 and CD 11b) antibodies were added to the polarized cells and then observed by immunocytochemistry Assay.

Results: Our preliminary data reveled that Macrophages polarized to M1-like phenotype showed high expression of the antigen CD86 following binding to the specific antibody, Macrophages polarized to M2-like Macrophages showed high expression of CD206 antigens and low expression of CD86 antigens following binding to the specific antibodies. Both phenotypes showed High expression of antigens bind to CD11b antibody.

Discussion/ Conclusion: Taken together, Our findings reveal that Mannose Receptor CD206 expressed in M2-like Tumor associated Macrophages is a valid target for peptides that has the capability to repolarize M2 macrophages to M1- like macrophages, which suggest the potential of these peptides to booster the immunoediting cancer microenvironment to fight cancer.

Nesbitt, Dazjane M. Poster 27

Understanding Mitochondrial Sensitivities in Environmental Carcinogen Transformed Cells

Dazjane' Nesbitt; Danitra Parker; Bryan Spurlock; and Kasturi Mitra

Background: 2,3,7,8- Tetrachlorodibenzon-P-Dioxin (TCDD) is a potent toxicant and environmental carcinogen that is a result of industrial accidents. TCDD which is released from organic compounds, is known to cause oxidative damage that can lead to skin cancer. In previous laboratory manipulations, HACAT (immortal keratinocyte), a human skin cell line, was treated with TCDD, with hopes of turning the cells into malignant or neoplastic transformative cells. TCDD transformed the cell line forming two new cell lines indicative of the doses 1nM and 10nM used in the experiment. One characteristic of this experiment is that the 1nM transformed cell line proliferates faster and had a different morphology than the parental and 10nM transformed cell line.

Purpose: The goal of my experiment was to further characterize the mitochondrial properties of the cells and to further test tumor promotion with Phorbol 12-Myristate 13-Acetate (PMA) using a two-stage assay where the first carcinogen initiates the carcinogenesis process and the second carcinogen promotes tumor formation. Along with testing the effects of PMA on human skin cells, mitochondrial sensitivities to an inhibitor of ATP were also performed. In this case, the effects of oligomycin on HACAT cells was investigated, as well as how the cells differed in their levels of SOX2, a stem cell marker. Oligomycin is a drug that inhibits ATP synthase, and is known to increase the redox of the cell, while Sox 2 is a transcription factor that is essential for maintaining the pluripotency of stem cells.

Methods: First, a two-stage *in vitro* cell transformation assay with PMA was performed. The goal was to enhance the tumorigenecity of the HACAT cells that were previously transformed with TCDD. Then, an oligomycin dose response was conducted to test the effects of oligomyecin on cells that were induced with SOX2. Lastly, an immunoblotting and immunofluorescence techniques to identify Sox2 expression between 1nM and 10nM cell lines.

Conclusion: Awaited.

Niranjan, Soumya, PhD Poster 28

"I'd want to know, because a year's not a long time to prepare for a death": Patient and provider perspectives regarding prognostic information in shared decision making among women with metastatic breast cancer

Niranjan, Soumya J., PhD; Wallace, Audrey, MD; Williams, Beverly, PhD; Turkman, Yasemin, PhD; Williams, Courtney, MS; Bhatia, Smita, MD, MPH; Rocque, Gabrielle, MD, MPH

Background: Increasing emphasis on patient-centered care has led to highlighted importance of shared decision making, which aims to better align medical decisions with patient preferences for care. Effective shared decision-making in metastatic breast cancer (MBC) treatment requires adequate understanding of prognosis, without which patients may receive treatments for this incurable disease that are inconsistent with their preferences. We assessed perspectives of MBC patients and providers regarding the role of prognostic information in treatment decision making.

Methods: Semi-structured interviews with 20 MBC patients and 6 community oncologists, as well as 3 separate focus groups involving lay navigators, nurses, and academic oncologists, were recorded and transcribed. Qualitative data analysis was conducted utilizing content analysis approach that included a constant comparative method to generate themes from the transcribed data.

Results: Of 20 interviewed patients with MBC, 30% were African American. Academic oncologists were mostly women (60%), community oncologists were all Caucasian, and nurses were all women and 28% African American. Lay navigators were all African American and predominately women (86%). Five emergent themes were identified. (1) Most patients were aware or inclined to learn about their prognosis, (2) Emotional distress was a critical reason for not discussing prognosis, (3) religious beliefs shaped preferences for seeking or declining prognostic information, (4) Timing of prognostic discussion impacted treatment decisions and (5) Providers acknowledged that an individualized approach towards timing and extent of prognostic information that takes into account patient values and preferences would be most beneficial for patients.

Conclusion: Although most MBC patients wanted prognostic information, they varied in timing of the information they received from their oncologists. Understanding why patients want limited or unrestricted prognostic information can inform oncologists' efforts to guide/facilitate patients in the decision-making process.

Nkengbeza, Leopold, BS — Poster 29

Stress Induced Phosphoprotein 1(STIP1) Is Overexpressed in Lung Adenocarcinoma and Predicts Poor Patient Survival

Nkengbeza, Leopold, BS; Robinson, Alyncia, MS; Chandrashekar, Darshan, Ph.D; Agarwal, Sumit, PhD; Varambally, Sooryanarayana, PhD

Lung cancer is among the leading causes of cancer deaths in the U.S., accounting for 1.5 million deaths in 2012. Furthermore, it metastasizes quickly resulting in very high mortality; in fact, the 5-year survival rate is at an abysmal 15%. Lung adenocarcinoma (LUAD) is the most common type of non-small cell lung carcinoma, accounting for 85% of the lung cancer cases. Multiple genomic alterations lead to cancer initiation and progression of LUAD. Stress-induced phosphoprotein-1 (STIP1) has been identified to be overexpressed in LUAD. STIP1 acts as a co-chaperone to heat shock proteins (Hsp70/Hsp90). These proteins have major regulatory functions in cell proliferation, protein folding, and signal transduction. In this study, the expression of STIP1 was characterized using publicly available RNA-sequencing data using the UALCAN web-portal (<http://ualcan.path.uab.edu>). Furthermore, by using prediction resources, we evaluated its interaction with heat shock proteins 70 and 90 (Hsp70/90) to determine its significance in LUAD. Cross-examination of various scientific journals and data was employed to investigate the role of STIP1 on lung cancer. Immunohistochemical staining suggests overexpression of STIP1 protein in LUAD. Our findings indicate that STIP1 is overexpressed and under-methylated in individuals with LUAD, particularly for people who smoke. STIP1 overexpression is also shown to have a poor survival rate, making it a potential target for lung cancer therapy.

Nweke, Chinedu B., BS — Poster 30

A Patient-Centered Approach to Understanding the Barriers and Facilitators Affecting Minority Participation in Cancer Clinical Trials

Nweke, Chinedu B., BS; Enis, Shawn; Rivers, Brian, PhD, MPH

Introduction: Cancer Clinical Trials (CCTs) are vital to the advancement of clinical oncology research and practice, as they represent the gold standard for the development and implementation of effective cancer therapies. However, there is a disparity in CCT participation as less than 3% of all CCT participants in the United States (U.S.) are African Americans (AAs). This is important as AAs experience an overall higher cancer incidence and cancer-related mortality compared to other racial and ethnic groups. Recent studies have suggested factors impacting AA participation are not only at the individual-level, but also at the system- and policy-level. Commonly reported barriers include an overall mistrust of the healthcare system, a lack of awareness of CCT, and historical instances of unethical research. Documented facilitators include patient/provider communication, potential benefits of trial participation, and an overall feeling of altruism. The objective of this two-phase pilot study is to utilize a multilevel, qualitative approach to assess the clinical and non-clinical facilitators and barriers to AA participation in CCTs (Phase I) and develop and pilot a novel, innovative multilevel intervention (Phase II).

Methods: Safety-net hospitals serve a high number of non-White, uninsured, underinsured, Medicaid, Medicare, and low income individuals. Study participants were recruited from a cancer center at a safety-net hospital in Atlanta, GA with over 80% of the patient population being AA Medicaid, Medicare, or uninsured. Focus groups were conducted with AA patients treated for cancer at the safety-net hospital. The focus group interview guide was adapted from the NIH-funded EMPACT study and were recorded on digital devices upon which the data was transcribed and subsequently analyzed. Representatives themes from the focus groups were determined using elements of grounded theory, inter-coder reliability, and thematic saturation.

Results: The focus group transcripts were analyzed using a combination of hand coding and NVIVO 11 software. Content analysis was conducted using an immersion/crystallizing analysis plan. Common themes regarding Barriers and Facilitators within the context of Institution-level, Participant-level, System-level, Trial-level, will be presented.

Conclusion: The findings of phase I will ensure the development of a patient-centered, culturally and linguistically appropriate intervention to increase AAs participation in CCTs.

Okekenwa, Sonia, BS — Poster 31

Chronic Myeloid Leukemia

Okekenwa, Sonia; Patel, Sweta; Welner, Robert, PhD

Chronic Myeloid Leukemia (CML) is formed from the translocation between chromosome 9 and chromosome 22 in stem cells. This oncogene gives rise to the active tyrosine kinase BCR-ABL1 in blood cells. CML results in an abnormally high number of myeloid cells (white blood cells) in the bone marrow and blood. Drug inhibitors against this active oncogene targets the kinase activity leading to the death of leukemic cells. In some patient's cases, these CML transformed stem cells don't die and become drug resistant. It is reported that the cell environment (stromal cells) provides protection to the CML cells from the effects of Tyrosine Kinase Inhibitors (TKIs). The stromal cells contribute to this resistance through CML-stroma adherence molecules and secretion of cytokines which protect the leukemic cells from the apoptotic effects of drug treatment. Therefore, my hypothesis is that CML cells co-cultured with stromal cells will survive when treated with drugs. The goal of this research is to characterize the reasons that CML cells when co-cultured with stromal cells survive drug treatment. CML cells were cultured and passaged, and the growth curve was made for CML cells and CML cells +/- drug. CML cells without drug grew significantly while CML cells with drug showed a decline in growth. The results show that TKI (drug) leads to the death of leukemic cells.

Peagler, Katie M., BS, MS — Poster 32

Expression and clinical significance of mito- ATPase and COX subunits in colorectal adenomatous polyps patients

Peagler, Katie, MS; Aikhionbare, Felix, PhD

Most patients with colorectal adenomatous polyps are asymptomatic in early-stage disease and usually present with invasive colonic adenocarcinoma or villous adenocarcinoma. Patients with adenomatous polyps have a threefold higher risk of colon cancer over the general population, which increases to sixfold if the polyps are multiple and with lower survival among African American subgroup patients. The reason for this disparity is not known. There is a pressing need to identify accurate genetic predictive factors in colorectal cancer progression to improve survival especially among minority populations. This study will further this goal by analyzing molecular phenotypic profiles of colorectal cancer linking them to racial differences in mortality of those patients. Variants in mitochondrial protein expressions have been correlated with several clinico-pathological features of cancers as the majority of the energy for tumor transformation are of mitochondrial origin. Therefore, differences in mitochondrial efficiency may be reflected as in adenoma-carcinoma sequence. Reports have shown that cytochrome c oxidase (COX) is a key player in oxidative phosphorylation and reactive oxygen species (ROS) formation. In addition, ATPase subunits are also associated with ROS formation and mtDNA maintenance. Here, we specifically searched for differentially expressed ATPase and COX subunits in early adenoma of CRC tissues as compared to late stages of CRC tissues. Both RT-qPCR and western blot techniques were used to assess ATPase and COX expression level differences. Results from this study show that gene expression and protein levels of ATPase 6 progressively increased from early adenomas to late stage adenomas. COX subunit 1 progressively increased 4-fold in cancer samples when compared to TA tissues. Lastly, COX subunit 4 isoform 1 decreased 5-fold in protein expression as adenomas progressed to cancer. Therefore, this study provides evidence that changes in mitochondria gene and protein expression may play an important role in tumor progression by altering colorectal cells energy metabolism levels.

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Randolph, Que'nyah, BS, MS — Poster 33

Comparative Analysis of Lactobacilli and Bifidobacteria Species as Probiotics and Assessment of Their Anti-Inflammatory Abilities

Randolph, Que'nyah. BS, MS; Alexanader, A. Deloris, BS, MS, PhD

Probiotics are live microorganisms that provide a variety of health benefits, including anti-inflammatory properties. However, in order to be effective probiotics must have the ability to survive in the gastrointestinal (GI) tract. *Lactobacillus reuteri* is a probiotic bacterium that was previously shown, by our lab, to be effective in reducing inflammation associated with inflammatory bowel disease (IBD) and ulcerative colitis-related colorectal cancer. Due to the success of *L. reuteri*, eight additional probiotics were tested; *L. brevis, L. casei, L. delbrueckii, L. gasseri, L. helveticus, L. sakei, Bifidobacterium infantis*, and *Bifidobacterium bifidum* strains. The purpose of this study was to determine whether the aforementioned bacteria could function as probiotics in a mouse model of IBD. 129SvEv IL-10 -/- mice were placed in Specific Pathogen Free housing conditions and Pre- or Post- treated with a probiotic bacterium, or left untreated (controls). Some animals were also dual-associated with an *E. faecelis/E. coli* mix. We found that *L. casei*, or *L. delbrueckii* supplementation appeared to reduced inflammation, while other probiotics were ineffective in reducing inflammation or exacerbated it.

Sharpe, Shannalee, MD — Poster 34

Novel Precision Therapeutic Agent to Pancreatic Cancers

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Purpose: Pancreatic Adenocarcinoma is one of the leading causes of cancer deaths in United States of America. This cancer is known for its extreme fatality rates, and there are no therapies available at this time that will improve the survival rates of pancreatic cancer patients. Therefore, developing any meaningful therapeutic agent to treat these cancer patients is utmost important to improve survival rates of pancreatic cancer patients. We have identified several members of ETS oncogene family and some of them are directly involved in several cancers including prostate cancer, Ewing family of tumors and leukemias. ETS family transcriptional factors are also shown to play a role in pancreas development. In addition, some of the ETS family members (ETV1, ETV4/PEA3/E1AF and ESE3) were shown to play a critical role in regulating differentiation and proliferation of pancreatic cancer cells. Interestingly, we have observed that ETS proteins ERG, Fli-1 and ETV1 which are involved in prostate cancer and Ewing sarcoma, inhibit RXR alpha transcriptional activity. It is suggested that this RXR-inhibitory activity by these ETS proteins may be responsible for these cancers. Since RXR alpha plays a role in the regulation of differentiation and proliferation of cancer cells including pancreatic cancer cells, we hypothesized that RXR-inhibitory activity of these ETS proteins may be partly responsible for the transformation of pancreatic cancer cells.

Methods: Using a novel cell-based assay, we have identified one small molecular weight compound that reverses the inhibitory activity of ETS on RXR alpha activity. This compound (RED 1025H3) effectively inhibited the growth of pancreatic cancer cells and has no significant effect on normal cells. This small molecule has no homology to retinoids. We intend to study the mechanism by which RED 1025H3 activates RXR alpha activity in pancreatic cancer cells. We also propose to evaluate in vivo efficacy of this small molecule compound using mice Xenograft models.

Conclusion: Therefore, RED 1025H3 may represent novel therapeutic agent that can specifically target pancreatic cancer cells. Thus, these studies will not only explain the molecular mechanism of inactivation of RXR in human pancreatic cancers, but also provide clues for precision therapeutic intervention.

Shobayo, Titilayo, BS — Poster 35

The role of ACA's Medicaid expansion on the incidence and mortality rates of Colorectal Cancer among minorities.

Shobayo, Titilayo BS; Rivers, Desiree, Ph.; Orewa, Bernard MD; Lee, Caplan, M.D., PhD, MPH

Introduction: As of mid-2018, 19 of the 50 states have yet to expand Medicaid. Enacted in 2010, the ACA had three primary goals in mind namely; increase the number of insured Americans, improve the quality of care, and reduce the costs of health care. The failure of these states to expand creates an imbalance in the distribution of health resources. This disparity has a strong impact in cancer. We hypothesize that the colorectal cancer community, the third most common cancer diagnosed in both men and women in the United States, would benefit significantly from Medicaid expansion. The purpose of this study is to assess the impact ACA's Medicaid expansion has had on the incidence and mortality rates of colorectal cancer among minorities.

Method: Using the North American Association of Central Cancer Registries (NAACR) and the US Mortality Data, we assessed the incidence rate and mortality rates of CRC in states that have and have not adopted Medicaid expansion. Data from 9 years pre- and 8 years post ACA enactment, based on race and ethnicity, was analyzed using Microsoft Excel and SPSS to calculate the difference in the incidence rate between Non-Hispanic Whites and Non-Hispanic Blacks. This difference indicates the effects of lack of accessibility to affordable health insurance such as Medicaid, on minority residents. The change in the incidence and mortality rates pre ACA and post ACA, indicates access to early diagnosis and treatment resources.

Result: Our research findings reveal an increase in the incidence of CRC and a decrease in the mortality rates in states that have expanded pre and post ACA enactment.

- States that expanded had an apparent increase in the incidence of CRC in the minority population. They also experienced a decrease in mortality rates.
- States that have yet to expand experience the same or an increase in the Incidence and mortality rates.

Discussion/Conclusion: State that adopted the ACA's Medicaid expansion, are experiencing an increase in CRC incidence and a decrease in CRC mortality rate. The increase in incidence that occurred post ACA is indicative of increase rate of CRC diagnosis. In states that expanded Medicaid, this increase is likely due to the availability of Medicaid to the underserved and minority population in these states. The decrease in mortality rates is a result of early diagnosis of CRC and accessibility of CRC treatments that was/is insured by Medicaid.

Simons, Janice L., BS — Poster 36

"If my insurance won't pay for this, I can't afford to do it": Interviewing ovarian cancer patients experiencing financial distress about the costs or related stress of treatment

Simons, Leahgrace; Liang, Margaret, MD, MSHS

Introduction: We previously conducted a cross-sectional survey of 121 gynecologic cancer patients receiving systemic therapy and found that 54% screened positive for financial distress, which was associated with a negative impact on patients' employment, spending, and quality of life. The objective of this follow-up interview phase is to obtain the patient perspective about challenges they faced managing the costs of care (COC) and desired characteristics of financial assistance programs.

Methods: We will recruit 32 women with newly diagnosed or recurrent ovarian cancer who have received treatment during the last three months and screen positive for financial distress. Patients will be screened using a patient reported outcome measure, Comprehensive Score for Financial Toxicity (COST), with COST <26 used as a threshold for financial distress. Participants will be purposively recruited from three treatment groups: 1) chemotherapy alone, 2) bevacizumab +/- chemotherapy, or 3) oral PARP inhibitors. We developed an interview guide based on findings from the completed cross-sectional survey and will refine it based upon the first two pilot interviews. Participants will complete a 60-minute in-person interview with two trained interviewers discussing their experience dealing with the COC. Interviews will be transcribed and then coded by two data analysts. Descriptive statistics and thematic analysis will be performed using SAS and NVivo.

Results: We have consented, screened, and interviewed one participant. Several preliminary themes were identified: psychological distress due to the COC, impact on both the patient's and caregiver's ability to work, and objective financial strain in the form of debt and consideration of bankruptcy. Identified barriers included lack of transparency of healthcare costs and insurance coverage during cancer treatment. Identified facilitators included health care team members acting in a timely and sensitive manner.

Discussion/Conclusion: Our findings indicate the need for additional resources for ovarian cancer patients focused upon psychological well-being, employment concerns, and caregiver burden. Methods to improve cost transparency from the payor or healthcare system standpoint may also ease financial distress. Continued recruitment will allow for more detailed evaluation in this population in order to inform the development of interventions to better prepare patients for the COC and decrease financial distress.

Stallworth, Martravious T., BS — Poster 37

Racial Disparities in the Predictors of Abnormal Anal Cytology in HIV+ Men Who Have Sex with Men (MSM)

Martravious Stallworth, BS; Suguna Badiga, PhD; Chandrika Piyathilake, DDS, MPH, PhD

Background and Purpose: Several studies have reported that HIV positive (HIV+) men who have sex with men (MSM) are at a greater risk for developing precancerous lesions of the anus compared to HIV negative MSM. Infection with high-risk human papillomaviruses (HR-HPVs) is the primary cause for those lesions. Risky lifestyle and sexual behaviors and poor immune status have been associated with acquisition and persistence of HR-HPVs in HIV+. Although there are several studies determining the risk factors of anal lesions in Caucasian American (CA) men, there are limited studies in African American (AA) men. Therefore, the purpose of this study was to determine the racial differences in the demographic, lifestyle, sexual behavior and CD4/viral load (VL) status in predicting HPV related abnormal anal lesions in HIV+ MSM population.

Methods: Study population consisted of 81 HIV+ MSM (AA, n=47 and CA, n=34) who attended the University of Alabama at Birmingham (UAB) 1917 HIV Outpatient Clinic who were diagnosed with atypical squamous intraepithelial lesions of undetermined significance+ (ASCUS+ n=57) or negative for intraepithelial lesions/malignancy (NILM n=24). Patient related information that are relevant to the current study (age, race, smoking, lifetime number of sexual partners) and laboratory data (CD4 counts-current /nadir and VL) were obtained from UAB Research and Informatics Service Center. Improvements in the CD4 counts were calculated for each patient based on the difference between nadir CD4 and CD4 count at the time of diagnosis of anal precursor lesions. Logistic regression models stratified by race were used to test the associations.

Results: AA men with detectable VLs (>20 copies/mL) were 7 times more likely to be diagnosed with ASCUS+ compared to those with undetectable VLs (<20 copies/mL) (P=0.0232). CA men who had lower improvement in CD4 counts (<500 cells/mm^3) were 18 times more likely to be diagnosed with ASCUS+ compared to those who improved ≥500 cells/mm^3.

Conclusions: As previous studies have reported that AAs who receive HIV care may not have sustained viral suppression, future studies need to investigate factors that are associated with poor viral suppression in this ethnic group. In CAs, lower improvement in CD4 at the time of lesion diagnosis are likely to be better predictors of disease risk suggesting the importance of adherence to cART to improve and maintain CD4 counts.

Tiesi, Koriann R. — Poster 38

Psychosocial Wellbeing and Supportive Care among Metastatic Cancer Survivors in the Deep South

Tiesi, Koriann; Bail, Jennifer, PhD, RN

Introduction: Of the 16 million cancer survivors in the U.S., 10-30% are living with metastatic disease. Metastatic cancer survivors (MCS) are at an increased risk for psychosocial distress, which may negatively influence quality of life. The National Comprehensive Cancer Network guidelines recommend that supportive care begin at diagnosis and continue through end of life. The aim of this study was to better understand psychosocial wellbeing and supportive care needs among MCS residing in the Deep South.

Methods: MCS were identified via UAB Cancer Registry and I2B2.Using a modified Dilman's method, eligible MCS (>21 years and physician permission to contact) were mailed a survey. Psychosocial wellbeing (i.e. physical and mental wellbeing, anxiety, depression, social isolation, emotional support, and hopefulness) of MCS were assessed via PROMIS® measures. Two survey questions queried supportive care use and interest. Returned surveys were double-key entered into REDCap®. Data was analyzed using Excel. Descriptive statistics were used to characterize the study sample and instrument scores. Between group difference, between female and male MCS, was examined via independent-samples t-test.

Results: To date, 100 surveys have been returned (female=60; male=40; M_{age}=67 years; $M_{survivorship}$=3 years) with a broad representation of primary cancer sites (breast=23%; prostate=10%; gynecological=16%; colorectal=13%; lung=10%; kidney=11%; other=17%). Mean instrument scores were within "normal limits" among MCS. However, females reported better physical (45.35 vs 43.89) and mental wellbeing (49.29 vs 47.88), anxiety (47.54 vs 48.60), depression (45.91 vs 47.91), social isolation (40.68 vs 40.79), emotional support (56.90 vs 55.04), hopefulness (57.51 vs 54.89) and history of supportive care use (40% vs 23%), than males, respectively. Both females (65%) and males (58%) reported interest in future supportive care, with the greatest interest in nutrition classes (37%). Gender differences were seen in program preferences (i.e., gardening, yoga, art therapy). Twenty percent of men who reported interest in supportive care desired "other" programs.

Discussion: Findings suggest that female MCS may be more likely to use supportive care, which may enhance psychosocial wellbeing. Gender preferences may play a role in supportive care uptake. Further research is needed to better understand preferences, facilitators, and barriers to supportive care use among male MCS.

Tran, Emily — Poster 39

Impact of Fisetin and Geraldol on Ribosomal Biogenesis: Preventive/Curative Implications on Breast Cancer

Tran, Emily ; Bailey, Sarah ; Weeks, Shannon ; Samant, Rajeev, ph.D.

Introduction: Fisetin, a nutraceutical compound found in various fruits and vegetables, has been shown to have cancer preventative and curative properties. Fisetin metabolizes to its active form, geraldol, by action of enzyme catechol-O-methyltransferase (COMT). The nucleolus is a structure within the nucleus and the site for most of ribosomal biogenesis. Recent discoveries in our group have shown that treatment with both fisetin and geraldol results in decreased nucleolar number in a variety of breast cancer cell lines. I verified this in 4T1 mouse mammary cancer cells. Cancer cells have more nucleoli due to cancer's demands to keep producing ribosomes, which make proteins for the cancer to proliferate. Less nucleoli represent an inhibiting effect on ribosomal biogenesis by suppression of RNA polymerase I (PolI), which transcribes for ribosomal RNA and sets the rate of ribosomal biogenesis. Mechanistic target of rapamycin, mTOR, is a signaling pathway that positively affects ribosomal biogenesis; its control over PolI is one way that affects ribosomal biogenesis. We have found that consistent with decrease in nucleolar count, fisetin and geraldol downregulate PolI activity. Our purpose was to test if geraldol affected ribosomal biogenesis through inhibition of the mTOR pathway.

Methods: RT^2 Profiler PCR arrays were used to profile gene expressions in the mTOR pathway-this was examined with geraldol treated RNA as compared to a DMSO control group.

Results: Positive Upstream Regulators of mTOR pathway such as Pld2, Rheb, Rraga, were downregulated. Consequently, many downstream effectors such as Eif4e, Rps6, Vegfb, Vegfc, Cdc42, Ilk, Prkce were also downregulated. There was an overall negative impact on expression of mTORC1 complex member levels (Mtor, Rptor) that most negatively affected cellular processes related to translation.

Discussion: Fisetin and geraldol have shown suppression of PolI activity. The downregulation of various genes in the mTOR pathway after geraldol treatment show a downregulatory effect on ribosomal biogenesis of cancer cells.

Conclusion: We noted several changes in gene expression of the mTOR pathway that solidified our hypothesis and provided an insight into possible mechanism of the action of geraldol. Further validation of our results with independent techniques will strengthen our findings.

Wallace, Erin J., BS — Poster 40

Association of Matrix Metalloproteinase and Fibrillin-2 in colorectal cancer

Erin Wallace, Shailesh Singh and Hina Mir

Introduction: Colorectal cancer (CRC) is the third most diagnosed and second leading cause of cancer-related deaths in both men and women within the United States. Most cancers, including CRC, progress by up-regulating cell growth and survival signaling while evading cell death. and ultimately disseminating and metastasize from their primary location. Matrix metalloproteases (MMP) play significant role in remodeling extracellular matrix, facilitating migration, invasion, proliferation and hence carcinogenesis. While Fibrillin-2 (FBN2), a connective tissue microfibrillar protein is also known to play significant role in progression and evading cell death in urothelial and non-small lung cancer. Goal of this study was to establish the association of MMP and FBN2 in CRC.

Methods: Gene Expression Omnibus database (GSE123390) consisting of twenty-eight rectal cancer samples and five normal tissue samples was used to analyze expression and association of MMP and FBN2 in CRC. Statistical analysis was performed using Graphpad Prism.

Results: Expression of Matrix metalloprotease-3 (MMP3) and Fibrillin-2 (FBN2) were significantly upregulated in CRC compared to normal.

Conclusion: MMP-3 is found to be a negative prognostic marker for pancreatic and cervical cancer while FBN2 is a negative prognostic marker for urothelial cancer. However, our analysis shows significant increase in expression of both of these proteins in CRC patients emphasizing their clinical significance.

Yarlagadda, Archana, BS Poster 41

Flap Structure-Specific Endonuclease 1 (FEN1) overexpression predicts poor survival in lung adenocarcinoma

Yarlagadda, Archana; Robinson, Alyncia, MS; Chandrashekar, Darshan S., PhD; Agarwal, Sumit, PhD; Varambally, Sooryanarayana, PhD.

Lung cancer accounts for nearly 1.5 million deaths worldwide in 2012, making it the leading cause of cancer deaths. It metastasizes quickly with very high mortality. The 5-year survival rate is only about 15%. There are two different types of lung cancers: non-small cell carcinoma and small cell carcinoma. Non-small cell carcinoma is the most common kind of lung cancer. 85% of non-small cell carcinoma cases are lung adenocarcinoma, which smokers and nonsmokers suffer from equally. DNA damage has long been known as a factor for cancer development. Inappropriate DNA repair may lead to the transformation of normal cells to cancer cells. Because of this, there are therapies that interfere with DNA replication and induce DNA damage which lead to cell apoptosis or killing of the cancer cells. However, these therapies are not the most effective due to the resistance of cancer cells. Instead of killing all the cells like chemotherapy does, there are genes that code for proteins can be targeted for treatment. Flap endonuclease 1(FEN1) codes for a protein that is critical in DNA repair pathways. In addition, FEN1 is involved in base excision repair which is one of the main methods of DNA damage repair and plays a key role in the development of cancer. DNA damage has been identified as a major factor in cancer, and improper DNA repair may lead to activation of oncogenesis. Overexpression of FEN1 has been observed in different of cancers, including lung adenocarcinoma. In this study, the expression of STIP1 was characterized using publicly available RNA-sequencing data using UALCAN web-portal. The survival analysis using Kaplan-Meier method suggested that higher expression of FEN1 predicts poor lung adenocarcinoma patient survival. Our analysis found a positive correlation between smokers and overexpression of this gene, suggesting that it is an oncogene and further studies can be done to target this gene.