2ND ANNUAL SPEECH REGIONAL CANCER HEALTH DISPARITY CONFERENCE

Hunter College of
The City University of New York
695 Park Avenue
New York NY, 10065

Thursday, May 14th 2020
9:00 AM - 3:30 PM

HUNTER
The City University of New York

TEMPLE UNIVERSITY

FOX CHASE CANCER CENTER
TEMPLE HEALTH

SPEECH
Synergistic Partnership for Enhancing Equity in Cancer Health

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AGENDA

9:00 AM - 9:45 AM **General Session**

Welcome Remarks: Jennifer J. Raab, JD; Lon S. Kaufman, PhD; John M. Daly, MD

Overview of TUFCCC/HC Partnership: Olorunseun O. Ogunwobi, MD, PhD; Grace X. Ma, PhD

Keynote Speaker: Curtis Pettaway, MD

NCI Welcome: Emmanuel A. Taylor, MSc, DrPH

10:00 AM - 12:00 PM **Poster Presentations**

PSC Members and NCI PD Interaction with Students, Trainees, Junior Faculty

12:00 PM - 12:45 PM **Lunch**

12:45 PM - 1:15 PM **Poster Awards Ceremony and Conference Adjourns**

1:15 PM - 3:15 PM **Mentor Training Workshop**

2:15 PM - 3:15 PM **Internal Advisory Committee Meeting**

IAC Members

CONFERENCE WELCOME MESSAGE

Dear Students, Colleagues and Associates,

On behalf of the Synergistic Partnership for Enhancing Equity in Cancer Health (SPEECH), we welcome you to the 2nd Annual SPEECH Regional Cancer Health Disparity Conference.

Hunter College is proud to host the conference virtually, despite the current COVID-19 pandemic. Our hearts go out to everyone who has been directly or indirectly affected by the crisis.

Through this virtual format, conference attendees can learn about ongoing work within our partnership and institutions within our region. This meeting includes a general opening session, virtual poster presentations and a mentor training workshop.

We hope this event offers valuable opportunities for all conference attendees.

Thank you for your participation.

Sincerely,

Olorunseun O. Ogunwobi, MD, PhD
Associate Professor of Biological Sciences
Director of the Hunter College Center for Cancer Health Disparities Research
SPEECH Contact Principal Investigator
Hunter College of The City University of New York

Grace X. Ma, PhD
Associate Dean for Health Disparities
Director of Center for Asian Health
Laura H. Carnell Professor in Clinical Sciences
Fox Chase Cancer Center Primary Member, Temple University Health System
SPEECH Contact Principal Investigator
Lewis Katz School of Medicine, Temple University

HUNTER The City University of New York · TEMPLE UNIVERSITY · FOX CHASE CANCER CENTER TEMPLE HEALTH · SPEECH Synergistic Partnership for Enhancing Equity in Cancer Health

ABOUT SPEECH

The Synergistic Partnership for Enhancing Equity in Cancer Health (SPEECH) is a National Cancer Institute U54 grant funded comprehensive regional cancer health disparity partnership between Temple University/Fox Chase Cancer Center and Hunter College (TUFCCC/HC).

TUFCCC/HC Cancer Health Disparity Partnership was established as a collaborative effort to develop a regional comprehensive cancer health equity research infrastructure in the Pennsylvania, New Jersey, and New York Regions. Our goal is to develop rigorous and sustainable research, education and outreach programs at all the institutions to address cancer health disparities and train underrepresented minorities to become leaders in cancer research.

Despite advances in cancer treatment and research, there are significant cancer health disparities in underserved African, Asian-Pacific, and Hispanic American populations. The mission of the TUFCCC/HC and address critical national needs of career development in cancer research among underrepresented junior investigators and students.

The TUFCCC/HC Cancer Health Disparity Partnership consists of five cores:

1. Administrative Core led by Olorunseun O. Ogunwobi, MD, PhD, Joel Erblich, PhD, MPH, Grace X. Ma, PhD, and Jean-Pierre Issa, MD
2. Research Education Core led by Olorunseun O. Ogunwobi, MD,PhD, and Carolyn Y. Fang, PhD
3. Planning and Evaluation Core led by Sarah-Jane (SJ) Dodd, PhD and Marsha Zibalese-Crawford, PhD, MSW
4. Community Outreach Core led by Ming-Chin Yeh, PhD, Marilyn Fraser, MD, and Yin Tan MD, MPH
5. Biostatistics and Bioinformatics Core led by Konstantinos Krampis, PhD, and Eric Ross, PhD

The TUFCCC/HC Cancer Health Disparity Partnerhip has three research projects:

1. Liver Cancer Long-Term Adherence to Monitoring/Treatment in Underserved Asian Americans with Chronic HBV, led by Sarit A. Golub, PhD and Grace X. Ma, PhD. ESIs: Chibuzo Enemchukwu, MD and Nestor Esnaola, MD
2. Nicotine Dependence and Lung Cancer Genetics in African Americans, led by Joel Erblich, PhD, MPH and Camille Ragin, PhD, MPH
3. Epigenetics Factors and the Microbiome in Disparities in Colon Cancer Outcomes, led by Frida Kleiman, PhD, Carmen Sapienza, PhD, and Jean-Pierre Issa, MD

GENERAL SESSION SPEAKERS

Jennifer J. Raab, JD

Jennifer J. Raab, JD

President, Hunter College of The City University of New York

Jennifer J. Raab marked her 18th anniversary as President of Hunter College, the largest college in the City University of New York system, with 23,000 students, five schools, and an annual operating budget of more than $250 million. President Raab has led the successful transformation of Hunter College from an open-admissions institution to a selective, highly ranked college; under her leadership both graduation and retention rates have increased markedly. Since she has assumed the presidency, Hunter has significantly increased its government grants and awards and strengthened its fiscal management.

President Raab has been responsible for securing $400 million in private support for the college. She has launched a $25 million library renovation; a science research and nursing/health professions facility in partnership with Memorial Sloan Kettering Cancer Center; a floor in the new Belfer Research Building at Weill Cornell Medical College; a $131 million School of Social Work in East Harlem; a new facility in Tribeca for Hunter's renowned art graduate program and gallery; and the $25 million restoration of the historic 1908 Roosevelt House. President Raab was a litigator at Cravath, Swaine & Moore and Paul, Weiss, after which she was appointed Chairman of the New York City Landmarks Preservation Commission, a post she held from 1994 to 2001.

President Raab served on the Board of Directors of Compuware Corporation, The After School Corporation, United Way New York, and the One To World Foundation, and was a member of the 2004-05 New York City Charter Revision Commission. She is currently a member of the Council on Foreign Relations, and serves on the Steering Committee of the Association for a Better New York and the Advisory Committee for WOMEN.NYC. Frequently honored for her leadership in education, President Raab was elected to the American Academy of Arts and Sciences in 2016. Other recognition from 2018 and 2019 includes induction into the Manhattan Jewish Hall of Fame, receipt of the Chairman's Award from The New York Landmarks Conservancy, and selection for the Manhattan Power 100 Award from City & State New York. A graduate of Hunter College High School (whose campus she now oversees), President Raab received a BA with distinction in all subjects from Cornell University in 1977, an MPA from the Woodrow Wilson School of Public and International Affairs at Princeton in 1979, and a J.D. cum laude from Harvard Law School in 1985.

GENERAL SESSION SPEAKERS

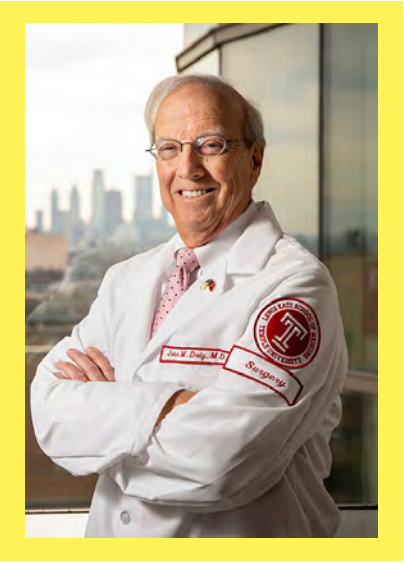
Lon S. Kaufman, PhD

Lon S. Kaufman, PhD
Provost and Vice President for Academic Affairs, Hunter College of The City University of New York

Provost and Vice President for Academic Affairs Lon S. Kaufman is an accomplished biologist, and was affiliated with the University of Illinois, Chicago (UIC) for nearly 30 years serving in a variety of administrative positions including Vice Chancellor for Academic Affairs and Provost, Vice Provost for Planning and Programs, Vice Provost for Undergraduate Affairs, Dean of the Honors College, and Head of the Department of Biological Sciences. He is deeply committed to public urban education, and his breadth of experiences as a senior administrator overseeing both the arts and sciences, coupled with a comprehensive knowledge of professional education, prepared him well for the range of responsibilities as Hunter's Provost.

His many successes at UIC include introducing a popular general education program, improving enrollment, retention and graduation rates, and implementing innovative programs to support faculty development and streamlined tracks to tenure and promotion. Dr. Kaufman is a native New Yorker who attended Stuyvesant High School and Brooklyn College, graduated from Queens College with a BA in Biology, and earned a PhD in Cell and Developmental Biology from SUNY, Stony Brook.

GENERAL SESSION SPEAKERS



John M. Daly, MD, FACS

John M. Daly, MD, FACS

Interim Dean, Lewis Katz School of Medicine at Temple University
Harry C. Donahoo Professor, Surgery
Surgical Director, William Maul Measey Institute for Clinical Simulation and Patient Safety

An internationally renowned surgeon whose clinical work and research span the fields of surgical oncology, metabolism, and nutrition, John M. Daly, MD, FACS, has led multiple medical associations, including the American College of Surgeons (Vice-President), the American Surgical Association (Vice President), the Society of Surgical Oncology (President), the American Society of Parenteral and Enteral Nutrition (President), the New York Surgical Society (President), and the American Cancer Society (President, Philadelphia Chapter). He has been listed numerous times among the "Best Doctors in New York and Philadelphia" and "318 Top Cancer Specialists for Women." Over the course of his career, Dr. Daly has been the recipient of over $20 million in research grants, predominantly from the National Institutes of Health. He has authored more than 250 peer-reviewed publications and 100 book chapters, has edited several books, and has served on the editorial boards of dozens of medical publications.

An internationally recognized educator, Dr. Daly has mentored numerous research trainees--a number of whom have risen to become chairs of surgery departments. He has been awarded honorary fellowships in the Royal Colleges of Surgeons of both Glasgow and Ireland, and has received many other teaching awards for his dedication to both medical students and surgical residents. A native of Philadelphia, Dr. Daly earned his bachelor's degree in biology cum laude from La Salle University and graduated AOA from Temple University's School of Medicine in 1973. He completed his surgical training at the University of Texas Medical School at Houston and the MD Anderson Cancer Center, where he later joined the faculty. Dr. Daly then moved to New York to join the faculty at Memorial Sloan Kettering Cancer Center (1980 to 1986), followed by a move to Philadelphia to become the Jonathan E Rhoads Professor of Surgery and Chief of the Division of Surgical Oncology at the University of Pennsylvania. In 1993, Dr. Daly was recruited by Weill-Cornell Medical College in New York to serve as Stimson Professor and Chair of the Department of Surgery, concurrent with his post as Chief of Surgery at New York Hospital. Dr. Daly then returned to Philadelphia to become Dean of his medical alma mater, the School of Medicine at Temple University. He served in the role from 2002 to 2011, and upon stepping down as Dean, returned to the practice of surgical oncology as the Harry C. Donahoo Professor of Surgery and Dean Emeritus for eight years at the Fox Chase Cancer Center, specializing in the care of patients with cancers of the gastrointestinal tract and breast. During this time, he also chaired the Institutional Review Boards for Human Research at both Fox Chase and Temple University, in addition to teaching students and residents. In 2019, Dr. Daly was appointed the Interim Dean of the Lewis Katz School of Medicine at Temple University, returning to the role he held for 9 years previously. Also in 2019, Dr. Daly became one of 91 surgeons from seven countries to be inducted into the inaugural group of Master Surgeon Educators of the American College of Surgeons. Dr. Daly has cared for thousands of patients who have cancer.

GENERAL SESSION SPEAKERS

Olorunseun O. Ogunwobi, MD, PhD

Olorunseun O. Ogunwobi, MD, PhD

Associate Professor, Biological Sciences, Hunter College of The City University of New York
Director, Hunter College Center for Cancer Health Disparities Research (CCHDR)

Dr. Olorunseun Ogunwobi obtained a medical degree from the University of Ibadan, Nigeria, a master's degree in biomedicine from the University of Hull, United Kingdom, a master's degree in clinical and translational science from the University of Florida, Gainesville, USA, and a PhD in molecular medicine from the University of East Anglia, Norwich, United Kingdom.

He is the founding Director of the Hunter College Center for Cancer Health Disparities Research, tenured Associate Professor of Biological Sciences at Hunter College of The City University of New York, and a member of faculty in the Biology and Biochemistry PhD programs at The Graduate Center of The City University of New York.

Dr. Ogunwobi is a translational cancer biologist whose work focuses on molecular mechanisms of progression of solid organ cancers with established racial disparities, such as hepatocellular carcinoma, pancreatic cancer, colon cancer, and prostate cancer. His laboratory has established novel circulating tumor cell models in hepatocellular carcinoma, and prostate cancer that are being used progressively to elucidate molecular mechanisms underlying the role of circulating tumor cells in cancer metastasis. They have also discovered that genetic alterations of oxytocin and the oxytocin receptor in some patients with pancreatic cancer and hepatocellular carcinoma significantly correlate with poor survival outcomes. And they are now investigating the molecular mechanisms of action of oxytocin/oxytocin receptor signaling in pancreatic cancer and hepatocellular carcinoma. A major focus of Dr. Ogunwobi's laboratory are studies elucidating the role of non-coding RNAs derived from the PVT1 gene locus in the development and progression of solid organ cancers. His work has been funded by the National Institutes of Health, New York State, Carnegie Corporation of New York, and the National Science Foundation, among others.

Dr. Ogunwobi is a Contact Principal Investigator of the Synergistic Partnership for Enhancing Equity in Cancer Health (SPEECH) funded by a U54 grant (CA221704) and Co-Investigator on a R01 grant (CA239603) both from the National Cancer Institute. An author of 55 peer-reviewed journal articles and 2 book chapters, Dr. Ogunwobi has been issued 3 United States patents for biotechnology inventions with potential clinical applications in cancer, and he is a Co-Founder of NucleoBio, Inc, a City University of New York start-up biotechnology company.

GENERAL SESSION SPEAKERS

Grace X. Ma, PhD

Grace X. Ma, PhD

Associate Dean for Health Disparities
Director, Center for Asian Health
Laura H. Carnell, Professor, Clinical Sciences, Lewis Katz School of Medicine of Temple University
Primary Member at Fox Chase Cancer Center, Temple University Health System

Dr. Ma is the founding director of Temple University Center for Asian Health (CAH), established in 2000, which is one of the first in the nation dedicated to reducing cancer health disparities funded by NCI/NIH. In partnership with Asian community leaders, she co-founded the first Asian Community Cancer Coalition in the U.S. eastern Region.

As a nationally recognized behavioral health scientist, Dr. Ma's research focuses on health disparities, cancer prevention, early detection, patient navigation, treatment adherence and access/quality of healthcare in underserved Asian Pacific Americans, African American and disparity populations. Her pioneering health disparity studies using community-based participatory and patient-centered approach are cited extensively. Over the past 24 years, Dr. Ma has received continuous funding awards from the National Institutes of Health. As the PI of NIH/NCI-funded Health Disparity Center grants (U54s, U01s) since 2000, Dr. Ma has led regional networks of cancer health disparity research, education/training and community engagement. She has directed over 95 intervention or observational longitudinal research studies, including large-scale randomized intervention trials, implementation and dissemination studies at worksites, community health centers, primary care clinics, community-based organizations and churches (NIH-funded R01s, R24s). She was PI for CDC-funded project, Racial and Ethnic Approaches to Community Health (REACH)" and PI for PCORI funded A Comparative Trial of Improving Care for Underserved Asian Americans Infected with HBV. Dr. Ma is the MPI for Unpacking Mechanisms of Disparities for HIV-related Hypertension in African Am and Asian Pacific Am MSM (RO1, NIH/NIMHD). Dr. Ma also conducted a number of studies focusing on multilevel risk factors and viral related diseases, evidence-based interventions for improving screening, vaccination, disease management, medication adherence, quality of life and continuum of care in underserved Asian Pacific Americans and African American populations. She is an author of 5 books, over 175 peer-reviewed publications and delivered over 650 professional presentations at regional, national and international conferences. She has trained and mentored over 200 minority trainees, that created a pipeline of diverse researchers to conduct health disparity research in underserved ethnic populations and communities.

Dr. Ma serves on numerous scientific advisory boards and NIH study sections, NIMHD-NIH national Health Disparity Science Vision Advisory Panel. She serves on national and state health disparity advisory boards and an active member of numerous Public Health association, American Association for Cancer Research, journal editorial boards. Dr. Ma, a recognized visionary leader, has received numerous distinguished awards from NIH, academic institutions, scientific associations, and community organizations.

GENERAL SESSION SPEAKERS

Emmanuel A. Taylor, M.Sc., Dr.P.H.

Emmanuel A. Taylor, M.Sc., Dr.P.H.
Health Scientist Administrator, Center to Reduce Cancer Health Disparities (CRCHD)/ National Cancer Institute (NCI)

Dr. Taylor provides leadership for program evaluation of all cancer disparities related programs within the NCI's Center to Reduce Cancer Health Disparities (CRCHD). He provides technical and scientific expertise and guidance to staff and grantees in the use of program evaluation techniques, performance measurement, community-based participatory research (CBPR), and translational research methodologies in relation to cancer prevention and control interventions in minority and medically underserved populations. He serves also as a Program Director for NCI-funded disparities research and research training programs. He manages grants, Cooperative Agreements, and contracts.

Dr. Taylor has over 30 years of experience in public health program planning, implementation, and evaluation at local/community, national, and international levels. Prior to joining CRCHD, Dr. Taylor was President and CEO of Health Information Management Associates (HIMA), Inc., as well as the Chief Epidemiologist and Director of health informatics, research and program evaluation at HIMA, Inc. He was an Associate Professor of Public Health at the Morgan State University, and Senior Epidemiologist for Minority Health at the Centers for Disease Control and Prevention (CDC). Dr. Taylor earned a doctorate in International Health/Epidemiology from the Tulane University School of Public Health and Tropical Medicine, with a specialty in the application of epidemiological methods for planning and evaluation of public health programs; a M.Sc. in Health Education and Communications, and a B.S. in Pre-med/Biology from the University of Southern Mississippi.

H. Nelson Aguila, D.V.M.

H. Nelson Aguila, D.V.M.
Deputy Director, Center to Reduce Cancer Health Disparities (CRCHD)/ National Cancer Institute (NCI)

Dr. Aguila is Deputy Director of the NCI's Center to Reduce Cancer Health Disparities. In this capacity, he represents the Center in various working groups across NCI and NIH, coordinates the day-to-day functions of the Center, and oversee and coordinates the programmatic management of CRCHD's Partnership to Advance Cancer Health Equity program. Previously, Dr. Aguila served as Chief of CRCHD's Diversity Training Branch.

Before coming to NIH, Dr. Aguila worked at the Food and Drug Administration as a Reviewer Toxicologist at the Center for Veterinary Medicine. Earlier in his career, he held senior research scientist positions in neuropathology at the University of Miami and later in cancer gene therapy at Aventis-Gencell. Dr. Aguila earned his Doctor of Veterinary Medicine degree at Austral University in Chile and trained as a neurobiologist at The University of Texas Southwestern Medical Center, Dallas.

GENERAL SESSION SPEAKERS

Keynote Speaker

Curtis A. Pettaway, MD

Curtis A. Pettaway, MD

Professor, Urology, Division of Surgery, The University of Texas MD Anderson Cancer Center, Houston, TX

Dr. Pettaway is Professor of Urology at The University of Texas MD Anderson Cancer Center in Houston Texas. His clinical practice is based on treating patients with genitourinary malignancies including penile, urethral, and prostate malignancies. The goal of his prostate related studies is to 1) further define host and molecular markers of cancer progression, and 2) to reduce disparities in prostate cancer outcome among African Americans and the underserved by studying both clinical and biologic correlates of aggressive disease.

He served as the medical director of the Prostate Outreach Project (POP) that has educated and screened over 5,000 men. He recently served as the co-director a National Cancer Institute funded U54 collaboration with the University of Puerto Rico evaluating the influence of West African Ancestry on the incidence and aggressiveness of prostate cancer among African American and Puerto Rican populations.

General Session Moderator

Joel Erblich, PhD, MPH

Joel Erblich, PhD, MPH

Associate Professor, Psychology, Hunter College of The City University of New York

Dr. Erblich was a founding member of the Mount Sinai Tisch Cancer Institute and Institute for Translational Epidemiology at Mount Sinai, where he spent a decade focusing on cancer prevention and control related to genetic factors primarily impacting African American smokers. Using a multidisciplinary-translational approach, Dr. Erblich's research in behavioral medicine has examined the effects of genetic polymorphisms on smoking behaviors and behavioral interventions to manage nicotine dependence. He has a long track record of research, training/mentorship and administrative leadership. In 2012, Dr. Erblich was recruited to Hunter College to spearhead a new doctoral program in Health Psychology in Clinical Science (HPCS), which has matriculated a number of outstanding URM students. This doctoral program built on his long-standing work in training post-doctoral students and his mentorship of students, trainees, and junior investigators in cancer prevention and control. He was a PI on numerous research grants and career development awards from diverse funding agencies, including the NCI, NIDA, American Cancer Society, Department of Defense Breast Cancer Research Program, PCORI, and others. Dr. Erblich also serves as the methodologist on several R01 grants in behavioral medicine.

2nd Annual SPEECH Regional Cancer Health Disparity Conference

Abstracts

BASIC CANCER RESEARCH

Poster #1

The role of fatty acids in crosstalk between melanoma cells and the microenvironment

Sabina Kubayeva[1], Mohita Tagore[2], Richard White[2]

[1]Macaulay Honors College at CUNY Hunter College
[2]Cancer Biology and Genetics Program, Sloan Kettering Institute

Metastatic melanoma is a lethal disease, with 5-year survival of 23%. Melanoma cells produce exosomes, extracellular vesicles that can be transferred between cells and participate in communication, "crosstalk", between melanoma and its microenvironment. Exosome transfer of cancerous RNA/protein cargo from melanoma to healthy keratinocytes can lead to keratinocyte "switching" from benign to malignant. Ceramide, composed of sphingosine and a fatty acid, is needed to produce exosomes. We hypothesized that fatty acids (FAs), which may influence ceramide generation pathways and exosome formation, could increase switching frequency. By co-culturing keratinocytes and melanoma cells in 64 FAs compounds and then using fluorescence microscopy at 2-day intervals, we were able to quantify the "switching" phenomenon, tracked by the use of a Cre-loxP system. If the number of switched cells was higher with than without the FA, the FA was considered a "hit". Compared to our control group with a normalized switching efficiency of 100%, we identified 13 hits from FA drug screening. Hits, which varied in structural features such as saturation and chain length, included oleic acid (OA) and compounds in the myristic acid family. We chose to pursue OA because prior reports indicate that OA is the most prevalent FA in the melanoma microenvironment and OA increases metastasis in breast and prostate cancer. On validation of OA, we confirmed the increase in switching frequency originally identified. Our findings may help elucidate pathways involved in crosstalk between melanoma and its microenvironment and guide development of targeted anti-cancer drugs for patients with melanoma.

Poster #2

A novel approach to modeling transcriptional heterogeneity identifies the oncogene candidate *CBX2* in invasive breast carcinoma

Daniel G. Piqué[1,2], Cristina Montagna[2], John Greally[2] and Jessica Mar[1,3,4]

Departments of [1]Systems and Computational Biology, [2]Genetics, and [3]Epidemiology and Population Health, Albert Einstein College of Medicine, Bronx, NY 10461
[4]Australian Institute for Bioengineering and Nanotechnology, The University of Queensland, QLD 4072, Australia

Oncogenes can serve as therapeutic targets for treating aggressive subtypes of cancers. Only several hundred oncogenes have been identified, primarily via DNA mutation-based approaches, in the human genome. Transcriptional (RNA) overexpression is a less-explored mechanism through which oncogenes can arise. Here, a new statistical approach, termed oncomix, which captures transcriptional heterogeneity in tumor and adjacent normal (i.e., tumor-free) RNA expression profiles, was developed to identify oncogene candidates that were overexpressed in a subset of breast tumors. Intronic DNA methylation was strongly associated with the overexpression of chromobox 2 (*CBX2*), an oncogene candidate that was identified using our method but not through prior analytical approaches. *CBX2* overexpression in breast tumors was associated with the upregulation of genes involved in cell cycle progression and with poorer 5-year survival. The predicted function of *CBX2* was confirmed *in vitro* using genetic knockdown, providing the first experimental evidence that *CBX2* expression promotes human breast cancer cell growth. Oncomix is a novel statistical approach that has the potential to uncover therapeutic targets that benefit subsets of cancer patients. *CBX2* is an oncogene candidate that should be further explored as a biomarker and potential drug target for aggressive subtypes of breast cancer.

Poster #3

MicroRNA-1205 directly binds to FRYL and suppresses aggressive prostate cancer

Michelle Naidoo[1,2], Fayola Levine[1], Xavier Graña-Amat[3] and Olorunseun Ogunwobi[1,2]

[1]Hunter College of The City University of New York, New York, NY, USA
[2]Graduate Center of The City University of New York, New York, NY, USA
[3]Lewiz Katz School of Medicine, Temple University, Philadelphia, PA, USA

High mortality rates of prostate cancer (PCa) are associated with metastatic castration- resistant prostate cancer (mCRPC) due to the maintenance of androgen receptor (AR) signaling despite androgen deprivation therapies (ADTs). Resistance to second generation ADTs leads to the progression to AR-independent neuroendocrine PCa (NEPC), which is observed in nearly 1 in 5 men with mCRPC and is associated with very poor outcome. PCa neuroendocrine differentiation (PCND) is observed in treatment-related NEPC, however, this mechanism is poorly understood. The 8q24 chromosomal locus is an important prostate cancer (PCa) susceptibility region containing genetic variants associated with increased PCa incidence and aggressiveness. *PVT1* is a gene located within this region that encodes microRNA-1205 (miR-1205), whose function is largely unknown. We have previously reported that miR-1205 is underexpressed in a cohort of histologically confirmed PCa tissue, when compared to normal tissue and is also underexpressed *in vitro* in CRPC cells, when compared to non-CRPC cells. We also demonstrated that exogenous miR-1205 significantly inhibited tumor volume in CRPC-derived xenograft male NOD/SCID gamma mice, suggesting that miR-1205 is a tumor suppressor. To understand the molecular mechanism of miR-1205, we demonstrated that miR-1205 directly targets Fry-like (FRYL) by performing an RNA pulldown assay. FRYL is predicted to regulate dendritic branching leading to the hypothesis that FRYL plays a role in PCND. To examine miR-1205 regulation of FRYL in PCND *in vitro*, LNCaP cells were cultured under androgen deprivation conditions for fourteen days. We observed FRYL mRNA overexpresssion and a significant reduction of miR-1205 expression in LNCaP-PCND cells when compared to undifferentiated LNCaP cells. To further determine whether this mechanism drives PCND, miR-1205 was inhibited in RWPE-1 and LNCaP androgen-sensitive cells. As a result, FRYL protein expression was increased along with neuroendocrine marker expression when compared to cells transfected with a negative control. To determine whether PCND is driven directly through FRYL activity, FRYL was silenced using an siRNA in PC-3 cells (model of NEPC). Interestingly, knockdown of FRYL did not reveal a decrease of neuroendocrine markers, suggesting that miR-1205 may induce PCND independently of FRYL. Further understanding this molecular mechanism may provide novel insights into overcoming ADT resistance in aggressive PCa.

Poster #4

Detection and Targeting of Triple Negative Breast Cancer with a Nuclear Directed p53 Tetramerization Domain Peptide

Gu Xiao, George Annor, Kimberly Fung, Brian Zeglis and Jill Bargonetti

Hunter College of the City University of New York

Triple negative breast cancer (TNBC) is more prevalent in African Americans (AAs) and TNBC in AA has been associated with worst overall survival. p53 is mutated in approximately 80% of patients in TNBC. A major challenge of targeting mutant p53 (mtp53) has been how to detect and block the protein *in vivo*. The high stability of mtp53 in cancer patients suggests new strategies for targeting the protein for diagnostics and therapy (theranostics). We generated a novel peptide (Cy5p53Tet) that contains the p53 tetramerization domain conjugated to a fluoroprobe for tumor imaging and inhibiting of the gain of function of mtp53. We demonstrated by live cell imaging and flow cytometry the higher uptake of Cy5p53Tet peptide for binding to mtp53 in the TNBC cell line MDA-MB- 468 than with wild type p53 in the Estrogen Receptor positive breast cancer cell line MCF-7. In addition, Cy5p53Tet signal reduction was found when mtp53 was depleted. *In vivo* analysis in mice bearing MDA-MB-468 xenografts showed that 12 minutes post injection, the Cy5p53tet peptide was able to detect tumors. The specificity of this peptide was confirmed in mice with a higher uptake of the Cy5p53tet peptide in MDA-MB-468 xenografts compared to in MCF7 xenografts. *In vitro* co-precipitation and cross-link assays validated Cy5p53tet peptide binding to tetrameric mtp53. In addition, higher nuclear Cy5p53tet peptide staining was observed in mtp53 TNBC than in normal mammary epithelial cell lines. Selective detecting by Cy5p53Tet peptide suggests the importance of targeting towards TNBC in multi-ethnic populations.

Poster #5

Reciprocal regulation of miR-1207-3p by its molecular target FNDC1 in prostate cancer

Priyanka Ghosh[1,3] and Olorunseun O. Ogunwobi[2,3]

[1]Department of Biochemistry, Graduate Center, City University of New York, NY
[2]Department of Biological Sciences, Hunter College of The City University of New York, NY
[3]Hunter College Center for Cancer Health Disparities Research (CCHDR), New York, NY

Prostate cancer (PCa) is the most common cancer among men around the world. Among the total number of new cancer cases that will occur in 2020, an estimated 23% will be that of the prostate. A subset of these men will develop the PCa subtype described as castration-resistant prostate cancer (CRPC). CRPC is metastatic, highly lethal, and frequently demonstrates resistance to current therapeutic strategies. Consequently, more work needs to be done to understand the underlying molecular mechanisms of PCa particularly CRPC with a view to identifying novel therapeutic opportunities. Human chromosome 8q24 is the most important PCa susceptibility locus. Chromosome 8q24 contains only one protein-coding gene, the oncogene *c-MYC*. However, it is rich in non-protein coding genes, including *PVT1*. *PVT1* is a long non-protein coding gene that encodes alternatively spliced transcripts and six well-annotated microRNAs (miRNAs), including miR-1207-3p. MicroRNAs are small non-coding RNAs that regulate gene expression at the post transcriptional level. It is noteworthy that many miRNA molecular targets are transcription factors (TF)**,** and the expression of some miRNAs and their molecular targets is reciprocal. Previously, our laboratory has shown miR-1207-3p to regulate molecular mechanisms and key cellular functions implicated in PCa development and progression. An important discovery is that miR-1207-3p is significantly underexpressed in human PCa cell lines as compared to normal prostate epithelial cells. Moreover, our laboratory showed that fibronectin domain containing protein1(FNDC1) is a direct molecular target of miR-1207-3p and there is a concomitant overexpression of FNDC1, fibronectin (FN1), androgen receptor (AR) and cMYC expression in metastatic prostate cancers. Preliminary data indicate that FNDC1, FN1, AR and cMYC all co-localize in PCa cell lines but do not all co-localize in normal prostate epithelial cells. Therefore, we hypothesize that miR-1207-3p is reciprocally regulated by its downstream molecular target FNDC1 in prostate cancer.

Poster #6

Population-level genetic differentiation at the PVT1 gene locus: implications for prostate cancer

Gargi Pal[1], Lia Di[1], Akintunde Orunmuyi[2], E. Oluwabunmi Olapade-Olaopa[2], Weigang Qiu[1], and Olorunseun O. Ogunwobi[1,3,4]

[1]Department of Biological Sciences, Hunter College of The City University of New York, New York, NY
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The incidence and mortality rate of prostate cancer (PCa) is highest in males of African ancestry. PCa is a multi-factorial, complex disease, but the exact mechanisms for its development and progression are unclear. Analysis of data from the one thousand genomes project revealed that a 26-kb region spanning PVT1 exons 4A and 4B shows a run of 75 SNPs with distinct allelic frequencies between African and non-African populations. The gene desert located on chromosome 8q24 is associated with aggressiveness of PCa in males of African ancestry. Interestingly, the non-protein coding gene locus Plasmacytoma Variant Translocation (PVT1) present at 8q24 is overexpressed in PCa. PVT1 gives rise to multiple transcripts with potentially different functions. To investigate if our observed population-level differences at the PVT1 gene locus have implications for PCa, we determined the expression of PVT1 exons 4A and 4B in histologically confirmed normal prostate (n=22), benign tumor prostate (n=35), and malignant tumor prostate tissue (n=28) samples obtained from males who had undergone prostatectomy or transrectal ultrasound-guided biopsy in Nigeria, a sub-Saharan Black African population. We have observed that PVT1 exons 4A and 4B are significantly overexpressed in PCa tissues in comparison to benign prostatic hyperplasia and normal prostate tissues obtained from males of African ancestry (moAA). Transient and stable overexpression of PVT1 exons 4A and 4B significantly induce greater prostate epithelial cell migration and proliferation. We anticipate that further exploration of the role of PVT1 exons 4A and 4B may lead to the possibility of exploiting them for diagnosis, therapy, and other clinical applications in PCa.

Poster #7

PVT1 exon 9 transcript regulates claudin 4 expression and migration in triple negative breast cancer

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Breast cancer (BC) is a heterogeneous disease that is classically driven by the estrogen receptor (ER), progesterone receptor (PR) and human epithelial growth factor receptor 2 (EGFR2/HER2) signaling pathways. Triple negative breast cancer (TNBC), is a lethal subtype of invasive BC tumors that are ER-, PR- and HER2-. A subtype of TNBC is claudin low (CL). Dysregulation of claudin proteins disrupts tight junctions, consequently inducing the epithelial-to-mesenchymal transition (EMT) in cancers. This leads to enhanced motility and metastasis. Patients with CL TNBC have worse prognosis than patients with other BC subtypes. PVT1 is a long noncoding RNA (lncRNA) transcribed from the 8q24 genomic locus that has been implicated in multiple cancers including BC. Amplification of the 8q24 gene locus is a common event in many malignant diseases and is associated with poor clinical outcomes. Although previous research has implicated PVT1 as an important player in BC, the underlying molecular mechanisms of PVT1 in CL TNBC was previously unknown. We assessed PVT1 expression in BC, and we observed that PVT1 exons 4A, 4B, and 9 are significantly upregulated in MDA MB 231 cells (claudin low) and significantly downregulated in MDA MB 468 cells (claudin high), in comparison to T47D (ER+). We have confirmed that claudin expression, specifically claudins 1, 3, 4 and 7, are significantly higher in MDA MB 468 cells and significantly lower in MDA MB 231 cells. Knockdown of PVT1 exon 9 expression in the MDA MB 231 cell line, led to a significant reduction in migration when compared to cells transfected with a control scramble siRNA, indicating that PVT1 exon 9 regulates migration in CL TNBC. Interestingly, we observed that claudin 4 expression, and not claudins 1, 3 and 7, was increased in cells where PVT1 exon 9 was knocked down when compared to the control cells. This indicates that PVT1 exon 9 regulates claudin 4 protein stability in CL TNBC. We also assessed the expression of EMT markers (vimentin, fibronectin, and caveolin) in MDA MB 231 cells. We observed no changes in EMT markers when PVT1 exon 9 is knocked down; however, our data suggests that EMT markers are more highly expressed in MDA MB 231 cells in comparison to MDA MB 468 cells. Taken together, our data indicate that PVT1 exon 9 regulates claudin expression and migration in CL TNBC, and may have implications for clinical outcomes in TNBC.

Poster #8

STAT2 Contributes to DSS-Induced Acute Colitis by Impairing Antimicrobial IL-22 Production

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Ulcerative colitis (UC) is one of two major forms of inflammatory bowel disease. This debilitating chronic condition causes inflammation in the colon and heightens the risk of colorectal cancer (CRC). In the United States, nearly one million people suffer from UC. It is prevalent in Caucasian populations and slightly higher in women. However, the incidence of UC is now increasing in ethnic minorities. What causes UC and its pathogenesis remains largely unclear. STAT2 is a key transcription factor that mediates the antiviral and antitumor effects of type I interferons. In recent studies we reported that in a chronic inflammation model of CRC, STAT2 promoted tumor burden whereas in a model of gastrointestinal bacterial infection, STAT2 facilitated pathogenic bacterial outgrowth. Therefore, we reasoned that STAT2 may also play a role in the onset of colitis before CRC development. We used the DSS model of acute colitis and found that mice lacking STAT2 (*Stat2KO*) exhibited mild intestinal inflammation compared to wild type control mice. *Stat2KO* mice also experienced less body weight loss, less damage to the epithelial gut barrier, and reduced immune cell infiltration. Reduced neutrophil infiltration in *Stat2KO* colons was also evident. Furthermore, colons from *Stat2KO* mice showed early and increased induction of the antimicrobial cytokine interleukin 22 (IL-22); known to be protective against colitis. Thus, our findings indicate that STAT2 may promote colitis by impairing IL-22 production, and thus bring to the forefront an unrecognized damaging role of STAT2 in the pathogenesis of UC, that when deregulated may progress CRC.

Poster #9

Is the oligomerization domain of GOF mtp53 R273H critical for chromatin-based activities?

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In triple negative breast cancers (TNBC), there is a high occurrence of missense mutations in the DNA binding domain of p53 protein. Some of these changes lead not only to loss of wtp53 function but also mtp53 proteins that acquire novel Gain-of-Function (GOF) abilities. Our group showed that mtp53 associates with MCM2-7 and PARP1 on chromatin and that GOF mtp53 R273H associates with replicating DNA. What is not clear is which domain in R273H mtp53 is responsible for its chromatin-based replication activities. We hypothesize that GOF mtp53 proteins form tetramers *in-vivo* and this tetramer formation is critical for mtp53 chromatin-based activities. We used the glutaraldehyde crosslink assay to test mtp53 oligomerization in TNBC cell lines and saw that R273H, R248Q and R280K mtp53 exist predominantly as tetramers. We used the CRISPR/Cas9 system to delete amino acid residues 347-393 in a TNBC MDA-MB-468 cell line expressing R273H mtp53 (R273HΔ347-393). Using the glutaraldehyde crosslinking assay and western blot analyses, we demonstrated that R273HΔ347-393 clone expresses a truncated version of mtp53 and is a monomer. The R273HΔ347-393 cells grow at a slower rate than the parental derivative. We used site-directed mutagenesis to introduce three OD mutations which destabilize tetramer formation of mtp53 in Li-Fraumeni syndrome patients, in plasmids expressing either wtp53 or R273H mtp53. We will test if the OD influences GOF mtp53 chromatin-based activities by determining if dual mtp53-OD variants no longer interact with replicating DNA. Our findings will increase understanding of the roles played by the OD in GOF mtp53 activities.

Poster #10

Controlled-magnetic nanorotor for delivery of RNA therapeutics

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While various nanoparticle-based gene carriers have been developed for cancer gene therapy, limitations remain in **i)** target specificity, **ii)** controlled-release of therapeutic RNAs at desired time, and **iii)** transfecting sufficient concentrations of therapeutic RNAs into cells. To reslove these issues, we developed cage-shaped magnetic iron oxide nanoparticles (IO-nanocages) as RNA carriers that can unload RNAs at the desired time when an alternating magnetic field (AMF) is applied. We hypothesize that IO-nanocages are accumulated in high concentration at the tumor site with RNAs in the cavity, and then AMF triggers spinning motion of magnetic IO-nanocages and release of RNAs. In this scheme, the centrifugal force created by the spinning motion could sprinkle RNAs at high velocity to reach deep into the cytoplasm and the nucleus of cells, resulting in high transfection efficiency, and thus the gene expression will be effectively altered for treatment. To test our hypothesis, *specific aims* include **1)** engineer therapeutic RNA- incorporated IO-nanocages, **2)** determine the efficiency of RNA release in prostate cancer cells and study effect on apoptosis/proliferation, and migration of cancer cells by delivering RNAs at desired time with spinning motion of IO-nanocages under AMFs *in vitro*, and **3)** optimize experimental conditions to maximize the therapeutic effect of RNA delivery by IO-nanocages *in vivo*. This outcome could lead to not only new treatment of prostate cancers, which is important in cancer disparity issues in the specialized community, but also broad impact in gene therapy and gene editing in general.

Poster #11

Reactive A1 Astrocytes Promote Cancer Cell Growth Within the Leptomeninges

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Scientific Discipline 2 Cancer Biology, a Cancer Biology

Leptomeningeal metastasis (LM), spread of systemic cancer into the cerebrospinal fluid-filled leptomeninges, is a fatal complication of cancer. Within the leptomeninges, cancer cells may float within the spinal fluid or adhere to the parenchyma. We observe proliferative adherent cancer cells in mouse models of LM. Beneath these proliferative cells, we observe aberrant accumulation of microglia and astrocytes. Others have shown that reactive astrocytes are induced by neuroinflammatory microglia secreting Il-1α;;, TNFα;;, and C1q cytokines. CNS injury is known to induce reactive astrocytes (A1) which may contribute to neuron death and axotomy. We hypothesize that cancer cells will secrete cytokines that induce astrocytes to adopt an A1 phenotype, resulting in the death of neighboring neurons and oligodendrocytes and proliferation of cancer cells. We measured cancer cell proliferation in vivo by immunohistochemistry (IHC) of formalin-fixed, paraffin embedded brains from our LM mouse models. To investigate the relationship between cancer cells and astrocytes, we generated a novel co-culture system with Leptomeningeal Metastatic (LeptoM) cancer cells (LLC, E0771, MDA or PC9 model systems) and primary mouse astrocytes. Gene transcription of the cancer cells and astrocytes was measured by real-time PCR (RT-PCR), allowing us to identify astrocyte transcriptomal phenotype (A1 vs. A2). Cancer cell growth was measured by Cell Titer Glo assay. IHC staining demonstrated that cancer cells adherent to the leptomeninges maintain high proliferative capacity. RT-PCR revealed significant upregulation of pan-reactive transcripts and A1-specific transcripts in astrocytes after co-culture with LeptoM cancer cells, in comparison to housekeeping genes. After three days of co-culture, LLC, E0771, and astrocytes demonstrated increased cancer cell viability compared with cancer cells alone. Our preliminary findings demonstrate that the A1 astrocyte phenotype is induced by co-culture with certain cancer cells. In addition, A1 signaling from astrocytes promotes cancer cell growth, suggesting new crosstalk between cancer cells in the leptomeninges and parenchymal astrocytes.

Poster #12

Human microbiome, colorectal cancer and health disparities: A review

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The human microbiome is the microbial ecosystem associated with the human body, which greatly impacts human physiology and health. Microbiome profiles are highly complex driven by a multitude of factors including human race and ethnicity. Recent studies have shown that alterations in microbiome composition play an important role in the development of disease such as different types of cancers. Here, we review research on colorectal carcinoma microbiomes studied across different human races and ethnicities. Colorectal cancer is the third most diagnosed cancer in the United States and former research indicated that incidence rates show clear racial/ethnic health disparities. We found that the abundance of the phylum Fusobacteria increases from healthy to tumor colorectal tissues, which was most noticeably in African Americans when compared with Asian and Caucasian colon cancer patients. Hester et al. (2015) examined fecal microbiome samples from Hispanics, African Americans, American Indians, and Caucasians and found significant differences such as an increased abundance of the phylum Firmicutes in African Americans compared to Caucasians. In summary, microbiome profiling across a diverse human population provides a more fundamental understanding of the human microbiome composition and diversity, which ultimately helps to improve the accuracy of early cancer diagnosis. Our review also highlights the need to conduct more extensive race and ethnicity-specific microbiome profiling in cancer patients, which would greatly contribute to the development of race- and ethnicity-specific biomarkers and microbiome-based therapeutic strategies.

Poster #13

Detection of Ubiquitin Ligase Activity from the *Mdm2* Splice Variant MDM2-C

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Triple negative breast cancer (TNBC), a subset of aggressive human breast cancer, is defined by its lack of hormone receptors, which limits available targeted treatment options. African American women are twice as likely to be diagnosed with TNBC than Caucasian women which poses a significant racial health disparity in the U.S. About 80% of TNBC patients harbor a mutation in the tumor suppressor gene *TP53,* which suggests mutant p53 contributes to TNBC progression. MDM2, a negative regulator of the p53, is overexpressed in 40-80% of high-grade tumors and shown to be required for migration and invasion of different cancers. MDM2 is an E3- ubiquitin ligase that ubiquitinates p53 for nuclear export, transcriptional repression, or proteasomal degradation. MDM2 has >70 known alternatively spliced transcripts, many of which display transformative properties when exogenously expressed. Our lab previously found that MDM2-C, which is missing exons 5-9, consequentially lacks a portion of the p53-binding and acidic domains, and as a result is unable to degrade p53. However, all splice variants retain exon 12, which encodes for the RING-finger domain, leading us to hypothesize that splice variants may also have ubiquitin ligase activity. Here we show that MDM-C cross-links with and ubiquitinates both wild-type and mutant p53. We also show that MDM2-C has auto-ubiquitination activity, which is abated with the addition of a competing substrate, such as p53. Our research highlights the importance of investigating how MDM2 splice variants in breast cancer drive ubiquitination of additional substrates that when modified may promote tumorigenesis in diverse populations.

Poster #14

In-Vivo Study of Orthotopic Drug and Tumor Injection into C57BL/6 Mice

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Pancreatic cancer is a lethal malignancy, but current treatment strategies (i.e. standard chemotherapy and radiation) are largely ineffective. However, emerging evidence suggests that a tumor's capability to grow and propagate is dependent on a small subset of cells within a tumor called cancer stem cells. Identification of pancreatic cancer stem cells and further elucidation of the signaling pathways that regulate their growth and survival may provide novel therapeutic approaches to treat pancreatic cancer. The overall goal of this work was to identify and therapeutically target the stem cell population and key signaling pathways that promote growth and metastasis in pancreatic cancer. We hypothesized that there would be a positive effect on human pancreatic study based on the in-vivo study we perform in mice. Presently, the expansion of human tumor tissue can only be accomplished in-vivo. The use of orthotopic mouse models is necessary in the examination of drug combinations. C57BL/6 mice were used to understand the biology of the drug in the mouse body. After the drug and mouse were prepped, orthotopic surgery was performed and the development of the tumor was observed through ultrasound. The tumor was allotted time to grow up to 2 centimeters, at which point was considered the humane endpoint. Organ harvests were performed on the mice bodies and in-vitro tests were conducted to assess whether or not the tumor had shrunk. We concluded that the tumor did, in fact, shrink after drug injection, so the drug studied here is critical to the development of possible treatments to pancreatic cancer. Further insights into and studies on this drug should provide more conclusive data in the designs of new therapies for pancreatic cancer treatments.

Poster #15

The Mutant p53 C-Terminal Domain Assists in DNA Interactions and Cell Cycle Promotion

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Triple-negative breast cancer (TNBC) is the most aggressive form of breast cancer and has the worst prognosis of any breast cancer sub-type. Because TNBC lacks estrogen, progesterone, and HER2 receptors, typical hormone deprivation treatments are ineffective. More than 80% of TNBC patients harbor mutated *TP53,* an indicator of further decreased prognosis. Some mutant p53 (mtp53) proteins exhibit gain-of function (GOF) oncogenic characteristics.
Our group previously found that mtp53 R273H interacts with newly synthesized DNA, and that mtp53 R273H increases chromatin-bound replication proteins such as MCM2-7, PCNA, and PARP1. Mutant p53 retains an intact C-terminal domain (CTD). In wild-type p53 the CTD is known to be important non-specific DNA binding. We hypothesize that the CTD of mtp53 facilitates DNA interactions at sites of DNA replication or repair and that this interaction facilitates at least some GOF oncogenic properties. To test this hypothesis, we have transiently transfected human cells to compare wild-type p53, and R273H mtp53 full–length and CTD-truncated variants. We have also used CRISPR/Cas9 to generate endogenous CTD truncations of mtp53 R273H in the breast cancer cell line MDA-MB-468. Preliminary data suggest that, for both exogenous and endogenous expression systems, mtp53 R273H and wild-type p53 with intact CTD tethers to chromatin better than CTD-truncated p53 proteins. Additionally, the CTD-truncated clone MDA-MB-468/delta380-387 demonstrates slowed cell cycle progression. Future work will focus on functional protein-protein and protein-DNA interactions mediated by CTD-mutated p53 proteins and compare how these interactions correlate with wild-type p53 interactions at sites of DNA-damage and DNA replication.

Poster #16

Immortalization of human primary prostate epithelial cells via inactivation of the *CDKN2A* locus and expression of telomerase

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Prostate cancer (PCa) is the most common cancer diagnosed in men and represents one of the largest cancer disparities of incidence and mortality rates between non-Hispanic blacks and whites; however, due in part to the limited number of cellular models available, the molecular basis of PCa development remains unclear. To tease out the molecular basis of PCa progression, it is necessary to have prostate epithelial cell lines as karyotypically normal as possible as a starting point. Immortalization of primary prostate epithelial cells (PrECs) with just hTERT expression is particularly inefficient in the absence of DNA tumor viral proteins or p16INK4A knockdown. Here, we describe the establishment of immortalized normal prostate epithelial cell line models using CRISPR technology to inactivate the *CDKN2A* locus concomitantly with ectopic expression of an h*TERT* transgene. Using this approach, we have obtained immortal cell clones of PrEC that exhibit fundamental characteristics of normal cells, including diploid genomes, near normal karyotypes, normal p53 and pRB cell responses, the ability to form non-invasive organoids and a non-transformed phenotype. Based on marker expression, these clones are of basal cell origin. Given the simplicity of this one-step methodology, it should also be suitable for the establishment of primary prostate tumor samples. This system could be used for the immortalization of primary prostate normal cells as well as cells of varied tumor grades from patients of diverse ethnicities to generate cell line models that facilitate the study of the molecular basis of disease disparity.

Poster #17

Impacts of Methylglyoxal (MGO) on Epigenetic Regulations

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Histone post-translational modifications (PTMs) control accessibility of chromatin, which regulates gene transcription. The metabolite methylglyoxal (MGO) is produced during anaerobic glycolysis. In this study, we propose MGO-induced glycation as a non-enzymatic covalent modification that acts as an epigenetic mark affecting chromatin stability. *In vitro* biochemical assays and immunoblot analysis were performed on recombinant core histones, histone octamers and nucleosomes. For *in vivo* analysis, 293T cells were treated with MGO in increasing concentrations and the histones were extracted for biochemical and biophysical analysis. We also synthesized a chemical probe that can attach to histones and function as an MGO mimic, allowing us to track the histone MGO-glycation. Western blot analysis of individual histones proved that at H3 histone is the most reactive histone, and that MGO can cause crosslinking of H3 products dose-dependently. Western blot analysis of histone octamers and nucleosome had similar results. *In vivo* assays showed that MGO decreases the signal of essential PTM sites dose-dependently, indicating that it can occupy them. To elucidate the effects of MGO on histones, a chemical probe was synthesized in a two-step route to produce a bio-orthogonal compound that mimics MGO. The results suggest that MGO-driven histone modification is an epigenetic mark that can bind to histone H3 most reactively. It can also occupy essential PTM sites and affect the assembly of nucleosomes, chromatin architecture and gene transcription. However, to fully understand the biological role of histone MGO-glycation, we synthesized a probe for click chemistry labeling and will use it for ChIP-sequencing.

Poster #18

Regulation of PINCH protein to slow progression and prevent recurrence of glioma

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Particularly *i*nteresting *n*ew *c*ysteine-*h*istidine-rich protein (PINCH) is a LIM-domain-only adaptor protein involved in protein recruitment, assembly of multi-protein complexes and subcellular localization of these complexes. PINCH is developmentally regulated and is critical for cytoskeletal organization and extracellular matrix adhesion. Although PINCH has no catalytic abilities, it serves as a link between integrins and components of growth factor receptor kinase and GTPase signaling pathways. Accordingly, PINCH-mediated signaling induces cell migration, spreading, and survival. After development, PINCH is nearly undetectable in health but developmental functions of PINCH extend to disease states and cellular responses to damage. PINCH expression in cancer has been correlated with gliomas, breast, colon, skin, lung, squamous cell carcinoma, throat and gastric cancer. In fact, PINCH stromal labeling has been defined as a prognostic indicator in colorectal cancer. *In vitro* studies indicate that silencing PINCH in cancer cells increases their vulnerability to radiation and chemotherapeutic agents. Importantly, studies by Loof *et al* reported that strong PINCH immunolabeling in adjacent normal tissue in colorectal patients was associated with poor survival, suggesting that PINCH expression is an early event in cancer development with deleterious effects. In this context, our preliminary data investigating PINCH in glioma have uncovered several important clues as to the potential biological role of PINCH. Our data show that PINCH is dramatically increased in some types of brain cancers, but less robustly in others. In this project, we assessed the mechanisms of activation of PINCH in glioma, the role of PINCH in survival of brain cancer cells, and signaling pathways responsible. We utilized primary astrocytes, glioma cell lines, and a newly developed conditional inducible PINCH knockout mouse model to investigate these pathways. In addition, we have assessed PINCH levels, patterns and cell type specific expression in tissue from brain cancer patients. Taken together, this multi-level approach has uncovered biologically important targets for prevention of recurrence or progression of glioma.

Poster #19

CUGBP1 and calreticulin can bind both a long non-coding RNA and full-length transcript of *CDKN1A* gene resulting in post-transcriptional regulation of p21 expression

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Regulation of *CDKN1A* gene expression, and hence p21 protein levels, has long been studied. The tumor suppressor p21 is a CDK inhibitor that regulates cell-cycle progression to prevent aberrant transmission of damaged DNA. The expression of *CDKN1A* under normal conditions and during DNA damage response (DDR) is tightly regulated. Our studies show that *CDKN1A* undergoes intronic alternative polyadenylation (APA) during DDR, and the resulting transcript is a long non-coding RNA (lncRNA). Like p21, lncRNA levels increase following UV treatment. Our recent data indicate that the loss of this lncRNA through siRNA-mediated knockdown produces a significant decrease in p21 protein, without a change in *CDKN1A* full-length mRNA levels, suggesting a role for this lncRNA at the post-transcriptional level.

Previous studies showed that RNA binding proteins (RBPs) calreticulin (CTR) and CUGBP1 can bind the *CDKN1A* full-length mRNA and regulate p21 translation (Iakova et al. 2004). CUGBP1 is a p21 translational activator, whereas CTR blocks translation of p21 via stabilization of a stem–loop in *CDKN1A* mRNA. Using RNA-immunoprecipitation assays, we show that CTR and CUGBP1 binding to the *CDKN1A* full-length mRNA increases in cells depleted of *CDKN1A* lncRNA. Both RBPs can bind the *CDKN1A* lncRNA directly. Additionally, after UV damage, CTR binding to the lncRNA is favored over full-length mRNA, whereas CUGBP1 binding to the full-length is favored over the lncRNA. Together, these results suggest a new regulatory mechanism for p21 expression at the post-transcriptional level, whereby the *CDKN1A* lncRNA sequesters p21 translational regulators CUGBP1 and CTR at different stages of DDR.

Poster #20

Race- and ethnicity specific signatures in colorectal carcinoma microbiomes

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Over the past decade, human microbiome research has made strides in relating disease to microbiome composition and diversity. More specifically, certain human microbiome profiles have been associated with an increased risk of cancer. In addition, racial/ethnic health disparities in incidence and mortality for cancer persist. Here, we utilized 16S rRNA sequence data to compare microbiome profiles associated with colorectal carcinoma tissue samples collected from African American, Caucasian, and Asian patients (n = 63) in the United States. Specifically, we performed differential abundance analysis using R packages *DESeq2* and *egdeR* to characterize microbiome composition of colorectal carcinoma tissue samples and to identify race and ethnicity-specific bacterial signatures. We found that bacterial genera Fusobacterium, Prevotella, Sharpea and Dialister were significantly more abundant in African-American colon cancer patients than Asians. Bacterial genera Dialister, Prevotella and Fusobacterium were significantly less abundant in African-Americans than Caucasians. Furthermore, genera Fusobacterium, Clostridium, J2-29, Bacteroides and Alistipes were significantly more abundant in Asians compared to Caucasians. These findings reveal important discrepancies and variations in colorectal cancer microbiota studied across different racial and ethnic groups. We recommend conducting further research with larger samples sizes to accurately determine the validity of race- and ethnicity-specific microbiome-based biomarkers identified here. In summary, the consideration of race and ethnicity in human microbiome research has great potential to further advance targeted treatment as well as diagnostic, and preventive tools for cancer.

Poster #21

Post-transcriptional regulation of mRNA 3' end processing and gene expression by estrogen signaling in MCF7 human breast cancer cells

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According to American Cancer Society, breast cancer (BrCa) is the most commonly diagnosed cancer and leading cause of cancer death among Hispanic women. It is widely known that dysregulation of estrogen signaling pathways leads to development of BrCa. Estrogen signaling is predominantly mediated by estrogen receptors to regulate gene expression. The potential role of estrogen signaling in post-transcriptional regulation of messenger RNAs (mRNAs) has not been extensively documented. mRNA 3' end processing plays an important role in balancing biosynthesis and degradation of mRNAs under different cellular conditions, affecting steady-state levels of cellular mRNAs and contributing to control of gene expression. We have shown that deadenylation, the removal of the poly(A) tail at the mRNA 3' end, is regulated by functional interactions between nuclear poly(A)-specific ribonuclease (PARN) and tumor suppressor factors, p53 and BRCA1/BARD1 under normal and DNA damage conditions in colorectal cancer cells. Our preliminary data indicate that estrogen treatment regulates mRNA 3' end processing and gene expression in MCF7 human BrCa cells. We show that nuclear deadenylation is activated at early time points of estrogen treatment and that ERα can form (a) complex(es) with PARN and p53 in nuclear extracts. Consistent with this, depletion and inhibition of ERα decrease nuclear deadenylation, suggesting ERα may act as an activator of nuclear deadenylation at early time points controlling gene expression in a transactivation-independent manner. These studies will help us to understand unique profiles of different BrCa and identify new molecular targets for treatments.

Poster # 22

Survival prediction of breast cancer patients using Deep Learning analysis.

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Survival models have been explored in clinical settings to investigate the relationships between cancer patients' prognostic covariates, such as genetic and clinical features, and their survival probability. The most common model used to assess prognostic variables in cancer patient survival is Cox proportional hazards, which may not accurately predict cancer patients' outcomes because of the assumption that the outcome is a linear combination of covariates. We present a study of 873 breast cancer patient data collected from The Cancer Proteome Atlas (TCPA) Portal. We applied a Deep Learning method for survival prediction in Breast Cancer (BRCA) patients and validated its predictive accuracy. Survival predictions by means of DeepSurv, a deep feed-forward neural network, was compared with the Cox proportional hazard model and the random survival forest model. We validate that the DeepSurv method performs better than the more common survival models, and its stronger predictive ability with multi-dimensional data. DeepSurv displayed the highest model performance among the three models, with the c-index of the testing sets reaching a perfect score of 1.00, followed by the Cox Proportional Hazard model with 0.94 and Random Survival Forest with 0.91. The predictive and prognostic abilities of DeepSurv, enables clinical researchers to use deep neural networks as a means in predicting the effects of a patient's clinical and genetic features on their risk of death.

Poster #23

Endostatin Regulation of MHC1 as a mechanism of immune evasion in circulating tumor cells

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Metastasis is the leading cause of cancer deaths. Metastasis involves dissociation of cells from the primary tumor, circulation of these cells in the blood, and the formation of secondary tumors. These cells which have separated from the primary tumor and are traveling in the blood are circulating tumor cells (CTCs). These CTCs are a critical step in metastasis. Several molecular changes have to take place for these primary tumor cells to dissociate and circulate in the bloodstream. One characteristic of these CTC's is that they are able to circulate in the bloodstream while successfully evading being destruction by the immune system. We have previously demonstrated that CTCs have significantly lower cell surface expression of an important cell surface protein called major histocompatibility complex class 1 (MHC1) is less expressed in CTCs as compared to primary tumor cells and it also shows a cytokine called endostatin that is less secreted in CTCs as compared to primary tumor cells. The under-expression of MHC1 in CTCs may allow CTCs to evade the immune system. In this study we are hypothesizing that decreased endostatin secretion in CTCs is what causes lower MHC1 cell surface expression via IFN-y signaling and serves as a mechanism of immune evasion for CTCs.

Poster #24

WASP acts downstream of Paxillin during plaque formation

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Each year in the United States, 1.4 million people are diagnosed with cancer. While a localized tumor can be removed surgically, once cells leave the tumor and metastasize, treatment becomes much more difficult. Understanding the migratory behavior of cells will aid in the process of controlling and reducing metastasis. The actin cytoskeleton is important for cell migration. It forms a complex network of filaments that helps regulate migration. While much is known about the actin cytoskeleton, there remain unanswered questions about the biochemical pathways associated with cell motility. Both Paxillin and WASP are proteins that play a crucial role in migration. However, their order of action is unknown. *Dictyostelium discoideum*, a simple eukaryote has orthologs of both Paxillin (PaxB) and WASP (WasA). Both genes appear to regulate migration of *Dictyostelium discoideum* cells. Since both genes are involved in migration, we hypothesize that both PaxB and WasA act in the same linear pathway during plaque formation. Since plaque size will be determined in part by how far a cell can move, small plaques will suggest less movement of cells while large plaques will suggest more movement.

In order to determine the order of action of these genes we carried out epistasis analysis of wasA and paxB using an assay of plaque formation on bacterial lawns. The wild type Ax3, and cells of paxB-,wasA- and wasA-/paxB- genotypes were studied. Plaque sizes were measured 5 days after plating. Results showed that paxB- cells formed large plaques while wasA- cells formed small plaques compared to Ax3. Moreover, wasA-/ paxB- cells also formed small plaque sizes compared to Ax3. By using plaque size to assess epistasis, we concluded that because both wasA- and wasA-/ paxB- cells formed small plaques, that both wasA and paxB act in a linear pathway during plaque formation. In addition, wasA acts downstream to paxB during plaque formation. Since our results show the linear pathway of these genes during plaque formation, then it is possible that the same is true for migration. We next wish to test that possibility by analyzing chemotaxis in these cell lines. Understanding the genes involved in cell motility will allow us to understand cell behavior and can potentially lead us to a better understanding of their roles in cancer metastasis.

Poster #25

Increased STAT2 expression in colorectal cancer cells enhances cell migration and impairs the antitumor effects of type I interferons

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Disparities in colorectal cancer (CRC) incidence and mortality in racial and ethnic minorities are associated with socioeconomic status, genetic, behavioral and environmental factors. The transcription factor STAT2 plays a key role in driving the antitumor effects of type I interferons (IFNα/β), used as adjuvant cancer therapy that has proven ineffective in CRC. Paradoxically, our studies in animal models of CRC have exposed STAT2 as a tumor promoter. Therefore, we analyzed human CRC datasets in TCGA and discovered colorectal tumors had elevated STAT2 expression that was associated with poor survival. African American and Caucasian CRC patients had in common elevated tumor STAT2 expression. The objective of our study was to investigate the biological significance of high STAT2 in CRC which is unknown. We overexpressed STAT2 in the human carcinoma cell line RKO to mimic elevated tumor STAT2 expression and determine its effects on tumorigenicity. We report STAT2 overexpression provided no growth advantage but enhanced cell motility. Furthermore, the antiproliferative effects of IFN-α were drastically impaired. STAT2 overexpression enhanced IFN-α induced activation of STAT2 and tumorigenic STAT3. Our results indicate that enhanced activation of STAT3 when STAT2 is upregulated may represent a mechanism by which tumor cells become resistant to IFN- α.

POPULATION CANCER RESEARCH

Poster #26

Reducing Cancer Health Disparities in Multi-Ethnic Communities in New York City

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Liver cancer is a chronic disease that is prevalent among the Asian American, African American, and Hispanic American groups in the U.S. The prevalence of liver cancer-related hepatitis C virus (HCV) infection is twice as high in African Americans (3.2%) than whites (1.5%). From 2003 to 2011, Hispanic Americans had the highest increase in liver cancer incidence (+35.8%), which has doubled compared to whites. Campaigns designed to raise liver cancer prevention awareness (e.g. hepatitis infection screening, lifestyle changes) were conducted in multi-ethnic communities in NYC. Specifically, handouts, stickers, buttons, and a brief PowerPoint presentation on HBV/HCV related liver cancer risk factors were delivered at organizations, such as YMCAs and senior centers. Participants filled out a survey after the presentation. Presented here are the preliminary descriptive analyses findings. Of those exposed to the cancer campaign (n=106), majority were female (76%), 65 or older (59%), African American (49%) or Hispanic (33%). Surprisingly, nearly two-thirds (62%) of the participants had a family member that was previously diagnosed with some form of cancer. Following the completion of the cancer campaign, majority of the sample said they were interested in learning about ways to prevent liver cancer (89%), likely to discuss liver cancer with their family/ friends (89%) and healthcare providers (86%), and Intended to implement lifestyle changes such as eating healthier and be more active (94%). Community campaigns successfully raised liver cancer awareness in multi-ethnic populations in NYC and encouraged community members to practice healthy eating habits and to engage in physical activities. Campaign strategies may improve liver cancer prevention.

Poster #27

Abnormal Cervical Cytology and Positive Human Papillomavirus Test Result Rates and Rates of Cervical Precancer and Cancer in an Urban Patient Population

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There remain significant discrepancies in rates of cervical cancer among different ethnic and socioeconomic populations. Current guidelines recommend HPV screening and pap smears every 3-5 years. However, our study ascertains the need for more frequent testing by examining rates of abnormal cervical cytology and positive HPV at New York-Presbyterian Brooklyn Methodist Hospital Women's Health Associates (NYPBMH Women's Health Clinic), an urban, racially diverse and underserved community. Using data from NYPBMH clinic patients with cytology and HPV test results in 2016 (N=2,176), we determined the number of patients who had abnormal cervical cytology results and/or positive HPV test results and tracked subsequent rates of cervical pre-cancer or cancer. We then calculated the rates of abnormal results and cancer for this patient population according to demographic data. The prevalence of abnormal cervical cytology and/or positive HPV was 9.3%, which is 24% higher than those reported by a study conducted at Kaiser Permanent Northern California (KPNC) with a predominantly white patient population. Of patients who returned for follow-up, 25% developed cervical pre-cancer or cancer. 6.7% of non-black/non-Hispanic patients had abnormal results. By comparison, abnormal rates for Black and Hispanic patients were 9.9% and 11.4% respectively. Our findings identified a higher rate of abnormal results than those reported at KPNC. This disparity is most likely due to higher risk of incidence associated with Black and Hispanic groups, which represent the majority of patients served in our clinic. Our results suggest that increased testing may promote equity in HPV-related outcomes.

Poster #28

The Development of a Culturally-Tailored Video Intervention to Promote HPV Vaccination among African American Adolescents

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Human Papillomavirus (HPV) is the most common sexually transmitted infection that can lead to long-term cancers. It is a serious problem in the United States affecting about 79 million people. Teenagers are more susceptible to get infected with HPV. Research has found that vaccination is a safe and effective method to prevent HPV. However, there is a massive influx of children that do not get vaccinated. One of the contributing factors to the issue is the lack of educational resources and provided information to parents. The study's objective is to develop a culturally tailored educational video to improve African American parent's beliefs and decision making on HPV vaccination for their children. The intervention will be conducted in health care facilities in Philadelphia. The intervention consists of watching an eight-minute animation educational video to gauge their beliefs and intentions about HPV vaccination. After the video, a health educator will then go over a discussion guide about parent's concerns and questions related to HPV vaccines. In this presentation, we will discuss the process of culturally tailoring educational messages and intervention material development. We will describe how the research team identified the education needs in the targeted population, identified educational topics and focus, and culturally tailored the video scripts. We will also describe the development of the education video.

Poster #29

Barriers and facilitators to the implementation and recruitment of the *mychoice* study

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This project evaluated barriers and facilitators to recruiting patients at the sites as well as factors impacting study retention. *mychoice* is a culturally-developed mHealth tool delivering educational information to patients to enhance informed decision-making about participation in cancer clinical trials to improve the racial diversity of participants. It is currently being tested in a four site RCT. This mixed-methods study utilized a 25-item survey (n=13) and structured interviews (n=4) done with study recruiters at cancer treatment centers to examine *mychoice* recruitment processes. The Consolidated Framework for Implementation Research was used to guide data interpretation. Major themes included trust-building with patients with cancer, maintaining relationships with key site stakeholders, and recruiter comfort with the recruiting process. Barriers involved patient health status, availability, and technology issues that impeded participating in the intervention. Facilitators included having site and physician champions and recruiter-patient engagement. Both barriers and facilitators to recruiting patients with cancer for a technology intervention were found. To address barriers, providing more structured training through scenario practices would help first-time recruiters feel more emotionally prepared to enroll patients with cancer. It is also imperative to gain support from physicians who have access to patients, can provide credibility for the study, improve the screening process and enhance the recruiter-patient relationship. Finally, recruiters should be provided training to enhance skills on how to be supportive if a participant needs assistance with study tasks. Study results can help future researchers identify prospective barriers to implementing tailored communication activities to improve patient participation in clinical trials.

Poster #30

Associations of Neighborhood Ethnic Density and Psychosocial Factors with Colorectal Cancer Screening Behavior Among Asian American Adults

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This study examined the influence of neighborhood ethnic composition on colorectal cancer (CRC) screening behavior in Asian Americans and explored whether associations between psychosocial predictors including knowledge, self-efficacy, and barriers with CRC screening behavior varied by neighborhood ethnic composition. Participants included 1,057 Filipino, Korean, and Vietnamese Americans aged 50 and older. Psychosocial factors associated with CRC screening, CRC screening behavior, and socio-demographics were extracted from participants survey data. Neighborhood ethnic composition was characterized as census-tract level percentage of Asian residents. Participant's addresses were geocoded to the census tract level to determine whether they resided in an ethnically dense neighborhood. The mixed-effects logistic regression model illustrated that residing in an ethnically dense neighborhood was associated with lower odds of CRC screening (OR=0.65, 95% CI=0.49, 0.77; p=0.019) after controlling for age, gender, education, and ethnic group. Furthermore, greater perceived barriers to CRC screening (OR= 0.62; 95% CI=0.55, 0.90; p<0.001) resulted in a lower odds of obtaining a CRC screening, while higher self-efficacy (OR=1.17, 95% CI=1.11, 1.23, p<0.001) was associated with greater odds of CRC screening. Among those living in a high ethnically dense neighborhood, greater barriers to screening was associated with lower odds of having obtained a CRC screening (OR=0.51; 95% CI=0.28, 0.93; p=0.028). The study findings revealed that residing in ethnically dense neighborhoods had negative impacts on CRC screening behavior. Future studies should examine disparities in built environment, socioeconomic, and cultural factors that are characteristic of ethnically dense neighborhoods and their impact on CRC screening participation.

Poster #31

Smoking is a Significant Mediator in the Association Between Metabolic Syndrome and Colorectal Cancer for Adults under the Age of 50

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Age 50 or above is associated with elevated risks of having metabolic syndrome (MetS) or colorectal cancer (CRC). However, recently evidence has indicated the risks to both conditions are increasing among adults under the age of 50. How are MetS and CRC related in this population? How do socioeconomic and modifiable lifestyle behaviors mediate the association? This study seeks to gain insights into these questions. We used data from the 2011–2016 National Health and Nutrition Examination Survey (NHANES) to define a case-control sample to examine the association between MetS and colorectal cancer and potential mediators. We used chi-square test and binary logistic regression to examine the MetS and cancer association. All analyses were conducted in Stata 14. From a total sample of 8,738 adults under the age of 50, we identified 8,730 non-CRC cases, and 8 CRC cases. Regression results showed that MetS was significantly associated with CRC risk (odds ratio = 4.43, p = .049), with age held constant. When we controlled for education, smoking, excessive drinking, and sleep duration, MetS was only marginally significant (odds ratio = 3.86, p = .076). Smoking was significantly associated with an elevated CRC risk (odds ratio = 9.50, p = .036). Smoking is a significant mediator in the MetS-CRC association among adults under the age of 50. Public health education and prevention efforts should target the current smokers in this population. A larger study sample is needed to better explore gender and racial/ethnic disparities of the associations.

Poster #32

Knowledge of Asian American Adults on Risk Factors Associated with Liver Cancer Before a Prevention Program in New York City

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Approximately 70% of liver cancer cases are caused by hepatitis B and C virus (HBV and HCV). Asian Americans (AAs) are disproportionally affected by liver cancer, with a mortality rate 60% higher than that of Caucasians. In recent years the incidence rate of liver cancer in New York City has been 35% higher than the national average. To increase awareness of liver cancer prevention and screening, we partnered with community-based organizations to collaboratively develop and conduct a liver cancer prevention project in New York City. We collected assessment data from Asian American community members who participated in our educational workshops. This presentation will report preliminary findings from 53 participants' knowledge on HBV, HCV, and liver cancer prior to the intervention. Chi-square and t-test were used in data analysis. Around half of the participants reported ever hearing about HBV (48.1%) and HCV (60.4%) from their doctors, and much less of them ever got HBV (37.7%) and HCV (9.4%) blood tests. Although the participants were knowledgeable on HBV/HCV transmission (scored 7.9 out of 10), they lack the knowledge on liver cancer risk factors (scored 1.9 out of 5). Those who were covered with health insurance were more knowledgeable on identifying risk factors of liver cancer than those uncovered (1.94 vs. 1.00, $p<0.001$). The findings reveal there is a pressing need for educational intervention to promote liver cancer interventions among at-risk Asian Americans, especially in New York City.

Poster #33

Increasing Liver Cancer Prevention Knowledge through Community-Based Education Among Asian American Adults in Philadelphia

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Cancer rates as a whole are going down across the U.S., however liver cancer incidence is increasing, especially in Philadelphia. Approximately 70% of liver cancer cases are caused by hepatitis B and C virus (HBV and HCV). Notably, Asian Americans (AAs) are disproportionally affected by liver cancer, with a mortality rate that is 60% higher than that of Caucasians. To increase awareness of liver cancer prevention and screening, we partnered with community-based organizations by co-developing and co-delivering a liver cancer prevention project in Philadelphia. We collected baseline evaluation data from the Asian American community members who participated in the educational workshops. This presentation will report the 152 eligible participants' descriptives and knowledge on HBV, HCV, and liver cancer both before and after the intervention. Chi-square and t-test were used in data analysis. Slightly less than half of the participants rated their health condition as "good" and above (48.95%). With regard to liver cancer prevention awareness, less than one third of the participants ever heard about HBV (31.58%) and HCV (21.05%) from their doctors, and even less of them ever got HBV (21.71%) and HCV (2.63%) blood tests. The knowledge of the participants increased significantly from the pre-survey to the post-intervention survey (7.39 vs. 9.24, $p<0.001$). The findings of our study showed that there is a crucial need for educational interventions to promote liver cancer prevention knowledge among Asian Americans, one of the groups suffering liver cancer disparity in the US.

Poster #34

The Association between Metabolic Syndrome and Cancer: Racial/Ethnic Differences and Underlying Behavioral Mechanisms

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Recent epidemiological studies have suggested a trend of increasing prevalence of metabolic syndrome (MetS) and certain types of cancer among adults under the age of 50. How is MetS associated with cancer in adults under the age of 50? How does the association vary by racial/ethnic groups? What are the potential lifestyle behavioral mechanisms? This study attempts to gain insights into these questions. We used data from the 2011–2016 National Health and Nutrition Examination Survey (NHANES) to define a case-control sample to examine the racial/ethnic disparities in the association of MetS and cancer of any type. We used chi-square test and binary logistic regression to examine the MetS and cancer association. All analyses were conducted in Stata 14. From a total sample of 17,969 cases, we identified 15,463 no-cancer cases, and 1,584 cancer cases. We found that MetS was significantly associated with cancer risk in the total sample (odds ratio = 1.50, p = .005). This association was only found in the Asian subsample with marginal significance (odds ratio = 3.68, p = .06). Smoking was a significant mediator to the MetS-cancer association in the total sample; sleep duration was a marginally significant mediator in the African American subsample. This study findings suggested racial/ethnic differences in how MetS and cancer are associated and the potential mediating effects of lifestyle behavioral factors. Future research with a bigger sample size is needed to fully explore these topics.

Poster #35

Changes in dietary and physical activity among Korean American older adults with a history of colon polyps

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Most colorectal cancers develop from colonic adenomas or polyps, highlighting the importance of studying the high-risk individuals with a history of colon polyps. Given the association between health behaviors and development of CRC, identifying the factors facilitating healthy behaviors is important to prevent CRC among the high-risk individuals. For an ongoing longitudinal pilot study, 18 Korean Americans with a history of colon polyps who were aged 50 years or older have been recruited thus far. Baseline information was used in analysis to identify the characteristics of the participants who reported successful change in health behaviors after detection/removal of colon polyps. Out of 18, 15 participants reported to attempt to change their behaviors, with approximately 50% (n=7) intending to change their diet. Among 15, 8 reported to be successful and 7 reported to be unsuccessful in changing their behaviors. T-test results showed that participants who reported successful change in their health behaviors after detection of colon polyps tended to be older (67.3 vs. 61.1), have better knowledge of health behavior guidelines (2.4 vs. 1.3), engage in greater level of physical activity (1.1 vs. 0.4), and receive greater social support (49.9 vs. 40.4). In our study, half of individuals with a history of colon polyp were unsuccessful in changing their behaviors despite their intention. Our results indicate the importance of developing programs to support the behavior changes by providing knowledge of health behavior guidelines and strengthening the support from social network.

Poster #36

Examining multilevel neighborhood socioeconomic characteristics associated with colorectal cancer screening in Vietnamese Americans residing in Philadelphia County

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Colorectal cancer is one of the most widespread and deadliest cancers. Vietnamese Americans exhibit persistently lower screening rates compared to the general United States population, which is due to cultural, economic, and environmental barriers. The impact of environmental factors in particular is not well known. This study aims to elucidate environmental factors affecting CRC screening rates among Vietnamese Americans. A total of 517 Vietnamese Americans 50 years and older residing in Philadelphia County were included in the study. Surveys were collected to determine CRC screening behavior (colonoscopy and Fecal Immunochemical Test) and sociodemographic characteristics. Individual neighborhood characteristics, which included the Walk Score was obtained based on the participant's address. Neighborhood characteristics were calculated using census-tract level data for the social deprivation index, ethnic composition, and presence of hospitals or federally qualified health centers (FQHC). The generalized linear mixed model revealed that residing in an ethnically dense neighborhood was negatively associated with CRC screening (β=-0.67, SE=0.29, p=0.01), while social deprivation (β=0.30, SE=0.27, p=0.27) and presence of FQHCs or hospitals (β=0.16, SE=0.30, p=0.58) were not significant. Individual neighborhood characteristics including the Walk Score (β=0.21, SE=0.26, p=0.43) was not associated with CRC screening behavior. Neighborhood characteristics, specifically ethnic density is associated with lower uptake of screening in this population. Future interventions should aim to target specific Asian neighborhoods that experience disparities in screening.

Poster #37

Physicians' Perspective About the Mobile Application that Aims to Lower the Risk of Developing Cervical Cancer in HIV Positive Women

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Cervical cancer (CC) is the most common cancer in HIV positive women. Cases are expected to increase by 46% until 2030. The aim was to determine physicians' satisfaction with the mobile application which develops CC algorithms in HIV+ women, provides definitive individual risk and critical preventive education. The study was a cross-sectional design and completed at Escola Bahaina de Medicine Saúde Pública in Brazil as a result of a collaborative project utilizing clinical informaticist expertise. First, CC risk factors were revised and algorithms selected by considering WHO's procedures. Three risk intervals for each algorithm were created by accepted lab results. We classified risk intervals as low, moderate and high. Second, the application was created by using Android Studio with PHP and MYSQL. Third, the physician interface of the application was published which lets physicians calculate CC risk and obtain the education as if they were patient. Last, a survey was created to obtain physicians' opinions. Five physicians associated with the research replied to our survey. 80%(n=4) were satisfied with the application, and 60%(n=3) thought the calculated risk was accurate. 80%(n=4) believed the education section was very helpful to change patients' habits for prevention. 80%(n=4) found the application quite useful to educate patients to understand the correlation between HIV+ and CC. Scholars may be interested in trying other classifications, extraction, or selection of algorithms and having a larger sample which may result in better performance in terms of accuracy sensitivity and specificity.

Poster #38

Nonadherence to Low-Dose Computed Tomography Lung Cancer Screenings: Socioeconomic Profile of High-Risk Patients from a Pilot Study

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Low-dose computed tomography (LDCT) scans have reported a significant reduction in lung cancer mortality among high-risk patients. This descriptive study seeks to examine the socioeconomic profile of patients who did not adherent to the LDCT and examine the social disparities in factors that may influence adherence. A survey was conducted over the phone to patients with initial negative LDCT screenings from 2016-2019 at Temple University Hospital. Surveys were used to evaluate participant's health literacy, beliefs, psychosocial factors, patient-provider relationship, and knowledge of lung cancer screenings. We conducted descriptive analysis and bivariate analysis to examine the socioeconomic profile of 43 patients nonadherent to LDCT. Patients were between ages 55-80, have at least 30 pack-year smoking history, who are current smokers or have quit within the past 15 years. Over two-thirds of the study sample were African Americans (69.77%), followed by 20.93% non-Hispanic white, 4.96% Hispanic, and 4.96% Asian. Bivariable analysis results indicate lung cancer screening knowledge varied across racial groups. Non-Hispanic whites had the highest score of lung cancer knowledge followed by African Americans. Lung cancer screening knowledge also varied among education attainment levels. Those who graduated high school or have higher education had higher scores of lung cancer screening knowledge compared to those who did not graduate high school. Our findings suggested that there is a low socioeconomic standing of LDCT nonadherence patients, and significant racial/ethnic and socioeconomic disparities in lung cancer related knowledge.

Poster #39

Sex differences in the role of social influence on smoking behavior and nicotine dependence among African American smokers

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Accumulating evidence points to an important role of social influence on smoking behavior. Notably, research has demonstrated significant sex differences in motivation to smoke, with women more likely to smoke for pharmacological reasons, and men more likely to smoke for social reasons. Effects, however, have not been thoroughly studied among African Americans (AA), who may have unique social and familial networks that contribute to smoking behaviors. We interviewed a community-based sample of AA smokers (n=30) from clinics and community centers in NYC and Philadelphia as part of a larger study of smoking and lung cancer risk. Measures included demographics, the Fagerstrom Test of Nicotine Dependence (FTND), and items assessing: 1) age at smoking initiation and 2) whether or not the participant has close friends/family in the community who are smokers. Our sample consisted of 20 women and 10 men who smoked an average of 8.9 (SD=5.7) cigarettes/day, from approximately 15.2 (SD=4.31) years of age. Mean FTND was 4.4 (SD=1.3). A factorial ANOVA revealed two interesting interaction effects: having a close friend/family member who smokes was related to higher levels of nicotine dependence [$F(1,26)=4.9$, $p<0.04$] and earlier age of smoking initiation [$F(1,26)=11.4$, $p<0.01$], but only in male smokers. Follow-up longitudinal studies among adolescent male and female AA smokers are warranted to further elucidate the role of social influence on smoking trajectories and risk of nicotine dependence.

Poster #40

Evaluation of a Cancer Wellness Internship: A Pipeline for Promoting Cancer Health Equity Education Among Diverse Pre-Health Students

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Despite biomedical advances in cancer treatment, racial and ethnic disparities persist across the cancer care continuum. Compounding concerns about projected shortages of oncologists, the American Society of Clinical Oncology reports a pressing need to diversify and educate the future oncology workforce towards addressing the sociocultural needs of underserved patients. The Bronx Oncology Living Daily (BOLD) Program of the Montefiore Einstein Center for Cancer Care has partnered with local schools to offer diverse students a cancer wellness internship experience. Interns conduct quality-of-life assessments with underserved cancer patients, assist with wellness workshops, and participate in community cancer education. 20 of 27 (74%) students interning from 3-12 months between 2018-2019 completed anonymous, post-internship evaluations. Respondents indicated increased interest in health careers (90%), health advocacy and equity (95%), and the field of oncology (44%). Interns' Mean self-rating (1-5 scale) about their knowledge of cancer disparities increased more than two-fold, from 2.21 to 4.53. Among valued skills gained were learning about psychosocial needs of underserved cancer patients (65%), communication skills (65%), and social determinants of health (50%). To exemplify impact, one intern stated "*I felt empowered and encouraged to make a difference in a place where I was born and raised. I am grateful to have helped my community in a way that relieves stress upon those who have experienced difficulty with a sick loved one.*" Data indicate the BOLD internship provides a valuable service learning experience and pipeline for diverse students poised to pursue health care careers with an interest in cancer health equity.

Poster #41

Multilevel Factors Related to Colorectal Cancer Screening Among Vietnamese Americans

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Colorectal cancer (CRC) is the fourth most common and deadly cancer in the United States. However, disparities persist among racial/ethnic minorities. Among Asian Americans, CRC is the third most common and deadly cancer. CRC screening (CRCS) is an optimal source of prevention and early detection, but utilization is low among Asian Americans. The goal of this study is to explore the effect of multilevel factors on screening for CRC. We utilized the community-based participatory research approach to assess the impact of multilevel factors on CRCS among a sample of Vietnamese Americans. A total of 801 participants were recruited from 20 community-based organizations in Philadelphia, PA. Multilevel factors assessed included individual (SES, knowledge, negative health belief, self-efficacy, insurance), interpersonal (social support) and community factors (social norms). In total, 194 participants reported previously receiving CRCS. Bivariate analysis found significant differences with the number of years living in the U.S., English proficiency and self-efficacy by screening status. Structural equation model results indicated a significant positive relationship between self-efficacy and CRCS (coefficient=0.092, z=2.98, p<.001). There was a significant negative relationship between self-efficacy and negative health belief (coefficient=-0.03, z=-2.42, p<.05). Additionally, there were significant negative relationships between negative health belief and SES (coefficient=-0.8, z=-3.16, p<.01), social support (coefficient=-0.36, z=-3.33, p<.001) as well as social norms (coefficient=-0.18, z=-2.75, p<.01). Study findings demonstrated the direct and indirect role of multilevel factors on screening behaviors. Targeting multilevel factors could be beneficial in improving CRCS by accounting for interpersonal and community-level factors that could affect individual screening behaviors.

Poster #42

The Development of a Pilot Study on HPV Screening in Men Who Have Sex with Men (MSM)

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Human Papillomavirus (HPV) is one of the most common sexually transmitted infections in the United States. HPV is acknowledged as one of the causes of anal cancer, with a 20-fold increased risk in men who have sex with men (MSM), when compared to age-matched heterosexual men. MSM are at potential high risk of contracting HPV, due to observed prevalence of risk factors such as a high number of sexual partners, rate of partner change, compromised immune system, and age. Vaccinations classified as HPV4 and HPV9 protect the aforementioned high-risk serotypes. Among studies conducted in MSM, few studies have focused on the needs, perceptions, knowledge, and intentions of young-MSM aged 18 to 27 years old pertaining to HPV diagnosis, associated anal cancer risks, and HPV vaccination intentions. To address this significant gap in the literature, in Aim 1 will conduct a study assessing vaccination intentions and background knowledge among young-MSM living in the Greater Philadelphia Area (n=400). In Aim 2 and 3, we will examine the geospatial associations between the accessibility of HPV screening services, HPV diagnosis, and knowledge of anal cancer risks among high -risk black and Hispanic MSM (n=100). The pre-pilot will inform proactive and responsive methods to identify the needs of young-MSM pertaining to HPV vaccination intent, the prevalence of HPV within the population, and associated anal cancer risks to assess disparities among at-risk Y-MSM.

Poster #43

Education and Navigation Intervention for Preventing Liver Cancer in Underserved Asians with HBV

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Asian Americans is a liver cancer disparity population that have the highest incidence and death rates than other racial/ethnic groups in the US. Insufficient Hepatitis B (HBV) management and treatment among Asian Americans are the risk factors of developing liver cancer. The study aims to develop effective intervention strategies to improve HBV management and treatment adherence among Asian Americans with HBV. The study team developed and refined intervention strategies using Community-Based Participatory Research (CBPR) Approach. We invited 9 Patient Centered Advisory Board (PCAB) members, including 3 HBV liver cancer patients, 3 health care providers (community-based physician, nurse and hepatologist), and 3 community leaders, to the entire developmental and test phases. The final intervention consists the following key components: (1) Virtual Patient Education (VPE); (2) Virtual Patient Navigation (VPN). All participants will receive HBV-related text messages, which have been approved to be efficient in improving HBV management. Only those in the intervention group will receive VPE and VPN. Different VPN needs have been identified by the participants: 27.2% of them identified pill-taking need, 42.9% of them identified appointment/transportation need, 3.3% of them reported that they need insurance support, 4.9% of them needed language support, and 37.5% of them identified information-seeking needs. Moreover, all most all of the intervention participants (98.4%-100%) believed that VPE is useful. CBPR approach is critical in developing culturally tailored intervention strategies. We will examine the effectiveness of the intervention at 12-m and 18-m follow up.

Poster #44

Central Obesity was Significantly Associated with Cancer Risk Among Adults under the Age of 50: Evidence from a Nationally Representative Sample

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In the past few decades, there has been an increase in the prevalence of cardiometabolic risk factors such as central obesity (waist circumference) and hypertension, as well as certain types of cancer among adults under the age of 50. This study used data of a nationally representative sample to examine how central obesity and hypertension are associated with cancer in adults under the age of 50. We used data from the 2011–2016 National Health and Nutrition Examination Survey (NHANES) to define a case-control sample to examine the association of central obesity and hypertension and cancer of any types. We used chi-square test and binary logistic regression to examine the associations. All analyses were conducted in Stata 14. In the total study sample of 17,969 adult participants, we identified 15,463 no-cancer cases, and 1,584 cancer cases. First, we found that central obesity was significantly associated with cancer risk in the total sample (odds ratio = 1.62, p = .02), with age, education, smoking, excessive drinking, and sleep duration held constant. However, hypertension was not significantly associated with cancer risks, when we controlled for the covariates in the regression. This study findings suggested a potential elevated cancer risk among those with central obesity. Longitudinal data were needed to decide the causal relationship. Research is also needed to examine any racial/ethnic and gender differences in the association.

Poster #45

Delays in Gastric Cancer Treatment: Observed Disparities by Race and Insurance Status

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Montefiore Medical Center

Time from cancer diagnosis to first treatment (TimeTx) is an important quality of care indicator. Identifying predictors of delayed TimeTx for gastric adenocarcinoma (GC) patients may reveal opportunities to reduce disparities. National Cancer Database identified adults with GC surgically treated 2004-2014. Patients with unknown TimeTx, stage IV GC, or emergency surgery were removed. Descriptive statistics compared those with and without delayed TimeTx (>8 weeks). Logistic regression models identified predictors of delayed TimeTx for the entire cohort, those whose first treatment was surgical (Surgfirst), and those whose first treatment was chemotherapy or radiation followed by surgery (Neoadjuvant). Of 30,945 patients, 19% had delayed TimeTx (22% of Surgfirst, 13% of Neoadjuvant). Predictors of delayed TimeTx for the entire cohort include: Surgfirst (OR 1.8, 95%CI 1.7-2.0), age >75 (OR 1.6, 95%CI 1.4-1.9), Black race (OR 1.3, 95%CI 1.2-1.5), low educational attainment (OR 1.4, 95%CI 1.2-1.6), Medicaid (OR 1.6, 95%CI 1.4-1.8), more comorbidities (OR 1.3, 95%CI 1.1-1.4). Among Surgfirst patients, delay was most associated with academic center treatment (OR 1.6, 95%CI 1.4-1.9). Among Neoadjuvant patients, delay was strongly associated with Black race (1.9, 95%CI 1.5-2.3), Hispanic race (OR 1.8, 95% CI 1.4-2.2), Medicaid (OR 1.7, 95%CI 1.4-2.1). Median time to treatment for non-Hispanic white patients with private insurance was 30 days (IQR: 21-41) compared to 39.5 days (IQR: 22.5-52.5) for Hispanic patients with Medicaid. Racial and insurance disparities are prominently associated with delayed treatment, especially for neoadjuvant therapy, and must be addressed to ensure high quality GC care for all.

Poster #46

Quality of Life After Endometrial Cancer: Which Patients Require Additional Support

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Endometrial cancer is the most common cancer of the female reproductive organs. Almost 60,000 new cases will be diagnosed in 2020 in the United States. Endometrial cancer survivors with overweight or obese body mass index (BMI) are at greater risk of comorbidities and, possibly, cancer recurrence; however, extant literature has not yet evaluated quality of life (QoL) during survivorship among this population. The aim of the current study was to identify patient characteristics associated with QoL after active treatment among endometrial cancer survivors with an overweight/obese BMI. Endometrial cancer survivors with an overweight/obese BMI (N=100) completed a cross- sectional survey after completion of active treatment. Patient-reported measures included demographics; health literacy; protective health behaviors (e.g., physical activity, nutrition); health status and comorbidities; and QoL (physical, social, emotional, functional, and endometrial cancer-specific well-being). Additional medical variables (e.g., grade, stage, BMI) were collected through medical chart review. Multivariable linear regression was completed to identify factors associated with QoL. Comorbidities and greater BMI were negatively associated with QoL (*ps*<.01). Greater BMI was also negatively associated with endometrial cancer-specific QoL (*p*<.05). Additionally, Hispanic/Latinx survivors reported worse endometrial cancer-specific QoL compared to Non-Hispanic White survivors (*p*<.05). Our findings suggest overweight/obese endometrial cancer survivors with higher BMI, more comorbidities, or of Hispanic/Latinx ethnicity report worse QoL during survivorship and may benefit from additional psychosocial management. In future research, it will be important to more finely assess and address QoL in overweight/obese endometrial cancer survivors.

Poster #47

The impact of estrogen metabolism and cigarette smoking on non-small cell lung cancer (NSCLC) risk in African American women: A pilot study in progress

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The incidence of NSCLC is higher in African Americans than other racial groups. This disparity may have biological underpinnings as prior work suggests that African Americans may be more susceptible to smoking-related lung cancer. Although the reasons for this remain unclear, emerging data suggest that estrogen metabolites contribute to NSCLC development. Research by our group indicates the lung can metabolize estrogen to several derivatives, including a putative carcinogen, 4-hydroxyestrogen (4-OHE). Levels of 4-OHE are elevated in NSCLC tumors as compared to paired non-neoplastic tissue and we recently discovered enhanced production of 4-OHE in patients with EGFR-mutated NSCLC, a cancer subtype found primarily in never-smokers. Additionally, our research indicates smoking induces cytochrome 1B1, the enzyme responsible for 4-OHE production, leading to greater levels of this carcinogen. This pilot study explores the mechanism by which African American women may be more susceptible to smoking-related NSCLC and will determine if estrogen metabolism contributes to increased risk. This will be evaluated by analyzing urine specimens from postmenopausal African American and White women (N=100). Levels of 4-OHE and other estrogen species will be quantified using an established UPLC-MS/MS assay and data will be analyzed with respect to smoking (current vs. never), race, and cancer status (NSCLC vs. cancer-free). Urine specimens from cancer-free African American women in the Philadelphia area are awaiting analysis and specimens from NSCLC patients are pending collection from TUH and FCCC clinics. Findings from this study will facilitate the identification of hormone-based biomarkers of cancer risk and inform novel therapeutic interventions.

Poster #48

Disparities in access to psycho-oncological support among Italian head and neck cancer patients

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While studies have focused on cancer health disparities, little is known about health systems that guarantee free access to treatments. Despite the appearance of equitable treatment access, systems can be under-resourced, indirectly creating disparities. Italian head and neck cancer patients receive psycho-oncological support based on their perception and communication of supportive needs. This study assesses the relationship between this criterion and disparities in access to psycho-oncological support. We recruited Italian head and neck cancer patients (N=15) who received psycho-oncological support either before or after their cancer treatment started. We assessed body image at four time points: before treatment and three time points post-treatment. Mixed Effect model analysis was completed to identify the effect of the psycho-oncological support over time. Body dissatisfaction showed different trends among patients who received the psycho-oncological support before the beginning of the cancer treatment and those who received it after (p=.02). Patients who received the support before treatment reported high body dissatisfaction at baseline, improving over time, while those who received support post-treatment demonstrated an opposite trend. Further data will be collected to increase the sample size. However, these preliminary findings suggest that relying on patients' perception and communication of supportive needs increases the risk of disparities in accessing this service. If patients overestimate their resilience and underestimate post-treatments difficulties, their supportive needs might not be communicated, resulting in delay of service and non-receipt of benefits from the treatment. Future research should therefore seek to identify predictors to better screen patients in need of psycho-oncological support.

Poster #49

Gender differences in dietary behavior and urinary gallic acid concentrations in racial minorities in New York City

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Nutrition is important for cancer prevention. To investigate associations between dietary differences based on demographics and risk of cancer, we assessed dietary behavior and the urinary concentrations of gallic acid, an antioxidant found in various fruits and vegetables, in racial minorities in New York City (NYC). Ninety-one (91) participants were recruited from a senior center in East Harlem, NYC, a racially diverse and underserved community. A National Institute of Health (NIH) - validated diet survey questionnaire was used to collect dietary behavior data. Demographic and cancer information were also collected. All 91 participants completed the survey and forty-five (45) participants provided urine samples for gallic acid analysis. Associations between demographic factors and the intake of certain foods were assessed. Gender differences were significantly associated with dietary behavior and urinary gallic acid concentrations (UGAC). Female participants had a higher daily intake of fruits compared to male participants ($p<0.05$). Additionally, female participants had significantly higher UGAC compared to male participants ($p<0.05$). Other associations were also observed. Age was negatively associated with the quantity of french fries/fried potatoes and white potatoes intake ($p<0.05$), while positively associated with frequency of fruit intake ($p<0.05$). Furthermore, Asian race was associated with a higher frequency of fruit intake ($p<0.05$), compared to other races. Among all recruited participants, we collected information on three cancer cases. Compared to non-cancer cases, participants with cancer reported a significantly lower fruit intake quantity ($p<0.05$). In a multivariate analysis, we observed a significant association between fruit intake quantity and UGAC ($p<0.05$) after controlling for income. Our findings suggest that gender differences in dietary behavior and UGAC may explain some of the gender differences in cancer incidence and prevalence observed in racial minority groups.

Poster #50

Distress and Generalized Self-Efficacy during Treatment Decision Making for Localized Prostate Cancer

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Prostate cancer is the second most common cancer among men in the United States. Treatment decision making for prostate cancer poses unique challenges, requiring patients to learn new terminology and manage distress. Individuals with low self-efficacy often struggle with decision making. To improve the treatment decision making process for prostate cancer, this study aims to identify predictors of self-efficacy in prostate cancer patients before treatment that can be integrated into decision making tools.
Localized prostate cancer patients (N=99) were recruited before a treatment decision making appointment to complete a questionnaire assessing demographics, medical comorbidities, health literacy, distress, and generalized self-efficacy. Multivariable linear regression analysis was completed to identify predictors of generalized self-efficacy. Patients were predominantly Non-Hispanic White (74.7%), married (65.7%), had income over $70,000 (48.4%), and had a mean age of 63. Patients had high burden of comorbid disease (M=19.85, SD=0.182) and high levels of health literacy (M=12.8, SD=0.235). Higher levels of distress were associated with lower generalized self-efficacy (p=.011). Health literacy was not significantly associated with generalized self-efficacy (p=.375). A prostate cancer diagnosis poses multiple challenges for patients, which may impact self-efficacy during the treatment decision making process. Our findings demonstrate high levels of distress are negatively associated with self-efficacy. However, we did not find a relationship between health literacy and self-efficacy. This may be due to our study populations' demographics and should be further investigated. Additionally, future research is needed to explore the association between distress and self-efficacy to identify mechanisms for targeted interventions for prostate cancer patients.

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