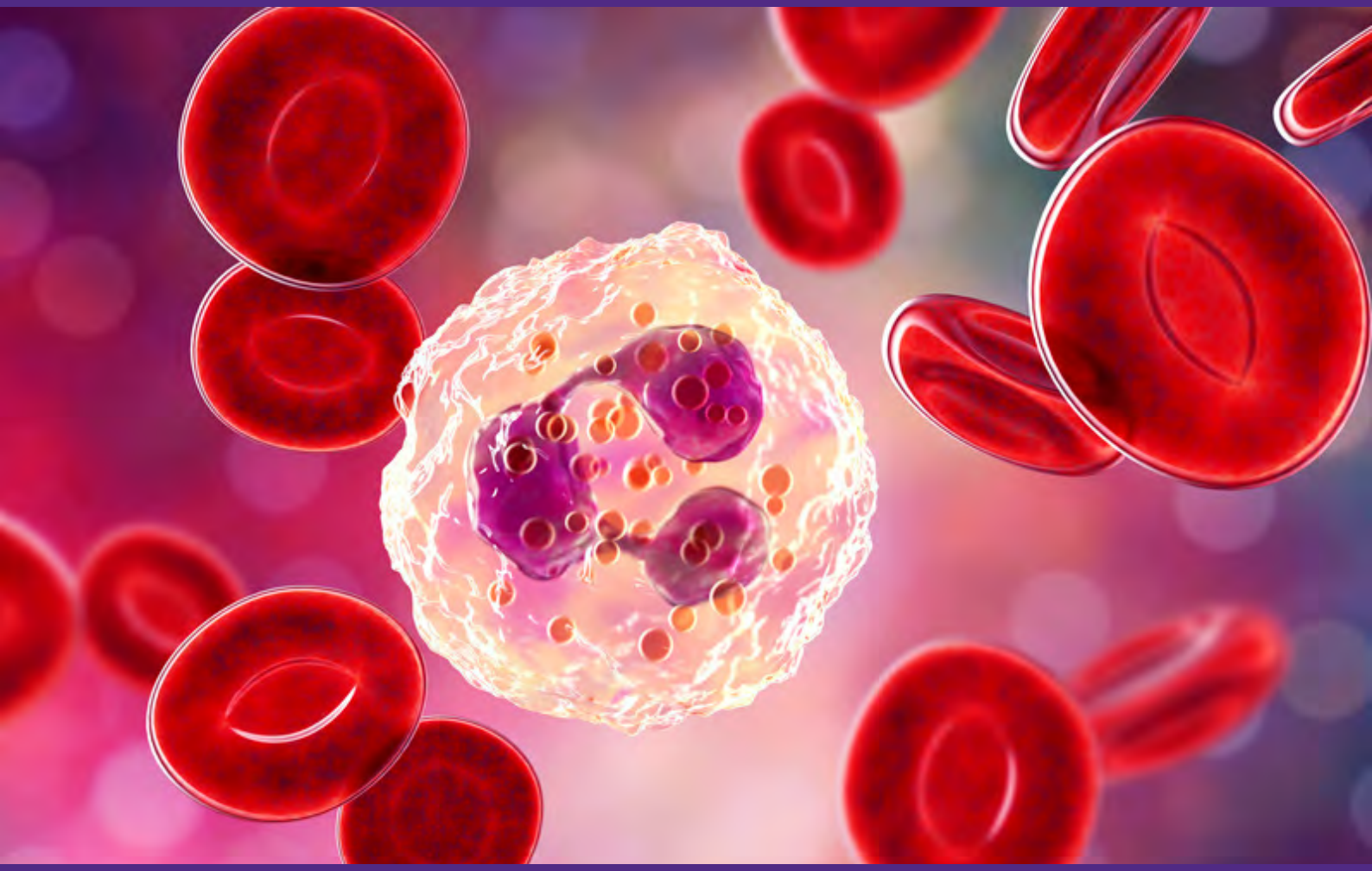
4TH ANNUAL SPEECH REGIONAL CANCER HEALTH DISPARITY CONFERENCE



Hosted By:

Hunter College of The City University of New York

Thursday, May 12, 2022
9:00 am - 1:15 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

TABLE OF CONTENTS

AGENDA

9:00 am - 10:00 am

General Session

Welcome Remarks:

Jennifer Raab, JD
Jason Wingard, PhD

Overview of TUFCCC/HC Partnership:

Olorunseun O. Ogunwobi, MD, PhD
Grace X. Ma, PhD

NCI Welcome:

Sandra L. San Miguel, MS

Keynote Speaker:

Joseph R. Osborne, MD, PhD

10:00 am - 11:30 am

Poster Presentations

Session 1: 10:00 am - 10:15 am - *Basic Science*

Session 2: 10:20 am - 10:35 am - *Population Science*

Session 3: 10:40 am - 10:55 am - *Basic & Clinical Science*

Session 4: 11:00 am - 11:15 am - *Population & Clinical Science*

Session 5: 11:15 am - 11:30 am - *Basic Science*

AGENDA

11:30 am - 12:30 pm

Oral Presentations:

Mandë Holford, PhD
Hiroshi Matsui, PhD
Aisha Bhimla, PhD, MPH
Jayashri Ghosh, PhD

12:30 pm - 1:00 pm

Lunch

1:00 pm - 1:15 pm

Presentation Awards Ceremony and Conference Adjourns

1:15 pm - 3:15 pm

Mentor Training Workshop

Virginia Valian, PhD
Distinguished Professor of Psychology,
Department of Psychology,
Hunter College of The City University of New York

This workshop includes a discussion of:

a) what we know about the value of mentoring (surprisingly little, in part because of a lack of consistent definitions of mentoring and a lack of rigorous experimentation); b) the value of multiple mentors, including peers (sometimes called a composite mentor, or mentor network, or circle of advisors); c) the role of workshops; d) mentoring across differences; and e) creating inclusive environments. Components of mentoring include providing psychosocial support, career and goal advice, sponsorship or advocacy, and necessary skills and knowledge. No single person can fulfill all of those functions. Thus, our job as "mentors" is to help people find the support, information, and skills they need to fulfill their aspirations.

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 4th Annual SPEECH Regional Cancer Health Disparity Conference.

The U54-funded partnership between Temple University/Fox Chase Cancer Center and Hunter College (TUFCCC/HC) was established with the purpose of reducing cancer health disparities among minority populations in the Pennsylvania, New Jersey, and New York City region, while encouraging diversity in the field of cancer research by training and mentoring students from underrepresented backgrounds.

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

Our goal is to offer a professional space for students, investigators, and researchers to learn about ongoing work and opportunities in cancer research. The conference includes a general opening session with keynote speaker, poster presentations, oral presentations and a mentor training workshop featuring Hunter College Distinguished Professor of Psychology Virginia Valian, PhD.

We hope this event provides valuable opportunities in academic and career development for all conference attendees.

Thank you for your participation.

Sincerely,

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

Grace X. Ma, PhD
Director of the Center for Asian Health
Laura H. Carnell Professor and Professor of Clinical Sciences
Lewis Katz School of Medicine, Temple University
SPEECH Contact Principal Investigator

ABOUT SPEECH

The Synergistic Partnership for Enhancing Equity in Cancer Health (SPEECH) is a comprehensive regional cancer health disparity partnership between Temple University/Fox Chase Cancer Center and Hunter College (TUFCCC/HC), the U54 grant funded by the National Cancer Institute. TUFCCC/HC Cancer Health Disparity Partnership was formed as a collaborative effort to develop a regional comprehensive cancer health equity research infrastructure in the Pennsylvania, New Jersey, and New York City Regions. Our goal is to establish rigorous and sustainable research, education and outreach programs at all institutions in order 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 Cancer Partnership is to reduce cancer health disparities among underserved health disparity populations 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 Grace X. Ma, PhD, Jean-Pierre Issa, MD, Olorunseun O. Ogunwobi, MD, PhD, and Joel Erblich, PhD, MPH
2) Research Education Core, led by Carolyn Y. Fang, PhD and Olorunseun O. Ogunwobi, MD,
3) Planning and Evaluation Core, led by Marsha Zibalese-Crawford, PhD, MSW and Sarah-Jane Dodd, PhD
4) Community Outreach Core, led by Yin Tan, MD, MPH, Ming-Chin Yeh, PhD, Marilyn A. Fraser, MD and Evelyn González, MA
5) Biostatistics and Bioinformatics Core, led by Eric Ross, PhD and Konstantinos Krampis, PhD

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

The TUFCCC/HC Health Disparity Partnership added three new research projects for years 4 and 5:
1) Attitudes Towards Somatic and Germline Genetic Testing Among Cancer Patients: Examining the Role of Medical Mistrust in Genetic Testing Disparities led by Michael J. Hall, MD, MS, Sarah Bauerle Bass, PhD, Tracey A. Revenson, PhD
2) Large-scale Generation of Immunotherapeutic Exosomes Stimulated by Self-assembling Peptides in Donor Dendritic Cells for Prostate Cancer Treatment led by Hiroshi Matsui, PhD, Olorunseun O. Ogunwobi, MD, PhD and Vincent Tan, MD
3)Characterizing the Activity and Function of Transient Receptor Potential Channels in Liver Tumor Cells Using New Terebrid Snail Venom Peptides led byMadee Holford, PhD, Joan Font-Burgada, PhD and Roland L. Dunbrack, Jr, PhD

Visit Our Website At:
http://www.speechregionalpartnership.org/

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GENERAL SESSION MODERATOR

Joel Erblich, PhD, MPH (MPI, HC)

Professor, Department of Psychology

Hunter College of The City University of New York

Joel Erblich, Ph.D., MPH (MPI, HC), Professor in the Department of Psychology. 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 HC 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. Dr. Erblich is currently the Hunter College MPI on the TUFCCC/HC U54 project, as well as the Project Co-Leader for the Lung Cancer project.

GENERAL SESSSION SPEAKERS

Jennifer J. Raab

President, Hunter College of The City University of New York

Jennifer J. Raab marked her 20th anniversary as President of Hunter College, the largest college in the City University of New York system, with more than 24,000 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; a townhouse on E 67th St to house Hunter's theater department and the $25 million restoration of the historic 1908 Roosevelt House.

President Raab was earlier a litigator 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 sat on the Board of Directors of Compuware Corporation and is currently a member of the Council on Foreign Relations, serves on the Steering Committee of the Association for a Better New York, Advisory Committee for WOMEN.NYC and sits on the boards of directors of 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.

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 Princeton in 1979, and a JD cum laude from Harvard Law School in 1985. In 2016, President Raab's achievements were recognized by her election to the American Academy of Arts and Sciences.

GENERAL SESSSION SPEAKERS

Jason Wingard, PhD

President, Temple University

Dr. Jason Wingard is a leading academic and executive specializing in the areas of organizational strategy, leadership development, and the future of work. He currently serves as President of Temple University and holds a dual appointment as Professor of Policy, Organization, and Leadership; and Professor of Human Resource Management. Temple is a public research university, consisting of 17 schools across eight campuses and a regional health system. The university enrolls approximately 37,000 undergraduate, graduate, and professional students and is among the nation's largest providers of professional education in the combined fields of dentistry, engineering, law, medicine, podiatry, and pharmacy. Temple University Health System is a $2.2 billion health system consisting of more than 1,000 physicians supporting excellence in medical education, research, and patient care. Dr. Wingard is also currently Founding Partner and Chairman of The Education Board, Inc., a boutique management consulting firm specializing in executive coaching and corporate advisory services.

Prior to Temple, Dr. Wingard served as Dean and Professor of the School of Professional Studies (SPS) at Columbia University. The graduate school offers 16 interdisciplinary master's degrees covering domains including analytics, management, communication, and science. Before Columbia, he served as Managing Director and Chief Learning Officer at Goldman Sachs, a multinational investment bank. At Goldman Sachs, he oversaw the acclaimed Pine Street Leadership Development Group and Goldman Sachs University and was responsible for the strategy and implementation of leadership development solutions for the firm's partners, global workforce, and clients.

Previously, he served as Vice Dean of the Wharton School, University of Pennsylvania, where he was the head of Executive Education and oversaw one of the world's largest providers of leadership and management development. He also served as Senior Vice President of ePals, Inc. and President & CEO of the ePals Foundation. ePals, Inc. (now Cricket Media) is the world's leading provider of interactive/collaborative learning products. Prior to joining ePals, Dr. Wingard was Executive Director of the Stanford Educational Leadership Institute, at Stanford University, where he led the engagement of executive coaching and applied research practicums for school leaders across the United States. He has also served in a variety of cross-functional executive and consulting roles for organizations including the Aspen Institute, the Vanguard Group, and Silicon Graphics, Inc. (SGI).

Dr. Wingard is a frequent keynote speaker. Recent engagements include Google, National Football League (NFL), PwßC, Procter and Gamble (P&G), CNBC, National Public Radio (NPR), the Wall Street Journal, and the Atlantic. Dr. Wingard serves as a member of the Board of Directors of Kroll, the world's premier provider of services and digital products related to governance, risk and transparency.

As a public servant, he currently serves on the Boards of Directors of JUST Capital, Roundabout Theater Company, and the Education Board Foundation. He previously served on the Boards of Directors for Tides, Building 21, United Cerebral Palsy of Philadelphia, the National Center for Fathering, and Philadelphia Futures.

Dr. Wingard holds a BA in Sociology (Organizational Behavior & Social Psychology), with honors, from Stanford University where he was a member of the varsity football and track teams. He also holds a MA in Education (Professional Development) from Emory University, a EdM in Technology in Education from Harvard University, and a PhD in Education, Culture, and Society (Corporate Education) from the University of Pennsylvania.

He enjoys classic jazz, cycling, and spending time with his wife and their children.

GENERAL SESSSION SPEAKERS

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.

His laboratory has established novel circulating tumor cell models that are being used progressively to elucidate molecular mechanisms underlying the role of circulating tumor cells in cancer metastasis. 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.

The Ogunwobi laboratory is also now working on epitranscriptomics, and utilization of genome engineering of the 3'untranslated region of the mRNA of known oncogenes as a novel therapeutic approach for lethal 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 U54 grants (CA221704 and CA221705), Contact Principal Investigator of 3U54CA221704-03S1 and Co-Investigator on R01 grant CA239603 from the National Cancer Institute. An author of 68 peer-reviewed journal articles and 2 book chapters, Dr. Ogunwobi has been issued 5 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.

In 2022, Dr. Ogunwobi became a recipient of the Hunter College Presidential Award for Excellence in Scholarship or Creative Activity.

GENERAL SESSSION SPEAKERS

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, Temple University
Primary Member at Fox Chase Cancer Center, Temple University Health System

Dr. Ma is a nationally recognized behavioral health scientist, leader and pioneer in cancer and health disparities research among underserved and vulnerable racial/ethnic minority populations. In her role as Associate Dean for Health Disparities, Dr. Ma has provided critical and effective leadership in building robust research infrastructure and leading multidisciplinary teams across Temple schools/colleges and multiple institutions in conducting independent and collaborative, highly competitive and innovative cancer health disparities research and education/training a pipeline of diverse investigators in population, translational and clinical sciences. Built on two decades of her leadership, Dr. Ma and her research team established successful partnerships with over 400 community organizations to engage Asian-Pacific American, Black/African American, Hispanic/Latino populations in health disparity research and interventions. During her career, Dr. Ma has received continuous awards in research grants from National Institutes of Health (NIH), federal agencies and other funders.

Built on two decades of her leadership, Dr. Ma and her research team established successful partnerships with over 400 community organizations to engage Asian-Pacific American, Black/African American, Hispanic/Latino populations in health disparity research and interventions. During her career, Dr. Ma has received continuous awards in research grants from National Institutes of Health (NIH), federal agencies and other funders. She has made seminal contributions in improving health equity and reducing health disparities. Over the past 22 years, Dr. Ma as Principal Investigator has directed 4 cycles of large-scale cancer health disparities research centers/networks funded by NCI/NIH including the recent U54 "TUFCCC/HC Regional Comprehensive Cancer Health Disparities Partnership." Dr. Ma's expertise spans a broad range of health disparities disciplines. Her community-based participatory research (CBPR) and patient-centered outcome research (PCOR) have focused on improving early detection, patient navigation, cancer prevention and control (Hepatitis-related liver cancer, cervical, breast, lung and colorectal cancers), smoking cessation, and access/quality of healthcare in underserved and racial/ethnic minorities. Dr. Ma has directed more than 100 intervention or observational longitudinal research studies, including large-scale cluster randomized intervention trials, implementation and dissemination studies at worksites, community health centers, primary care clinics, community-based organizations and churches (NIH funded R01s, U01s, R24s).

She also conducted a number of studies focusing on multilevel risk factors and viral related diseases (e.g. HBV, HCV, HPV and HIV), evidence-based interventions for improving screening, vaccination, disease management, medication adherence, quality of life and continuum of care in underserved Asian Pacific Americans and Black/African American populations. Dr. Ma mentored over 260 minority junior faculty, post-doctoral fellows, doctoral and master students that created a pipeline of diverse workforce of researchers in health disparities.

Dr. Ma authored 5 books, over 210 scientific journal articles, and delivered over 680 professional presentations. Dr. Ma has served on more than 40 scientific advisory boards in health disparities research, including NIH national Health Disparity Science Vision Advisory Panel and NIH study sections. Currently, Dr. Ma Co-Chairs Asian American, Native Hawaiian & Other Pacific Islander (AA NHOPI) Interest Group for NIH Community Engagement Alliance (CEAL) Against COVID-19 Disparities.

GENERAL SESSSION SPEAKERS

Sandra L. San Miguel, MS

Program Director, CRCHD, National Cancer Institute/National Institute of Health

Sandra L. San Miguel, MS, DrPH(c) is a Program Director in the Integrated Networks Branch of the National Cancer Institute's Center to Reduce Cancer Health Disparities (CRCHD). In this role, she provides technical and scientific expertise to the Comprehensive Partnerships to Advance Cancer Health Equity (CAPACHE-U54) by managing various partnerships across the nation. The CPACHE funds equal partnerships between institutions serving underserved health disparity populations and underrepresented students (ISUPS) with NCI-designated cancer centers across the nation.

San Miguel co-developed and is responsible for managing the Connecting the Underrepresented Populations to Clinical Trials (CUSP2CT), a U.S. White House Cancer Moonshot Initiative.

CUSP2CT is a UO1 national program that will implement and evaluate multilevel and culturally tailored outreach and education interventions with the primary goal of increasing referral and ultimately, accrual of underrepresented racial/ethnic minority populations, to NCI-supported clinical trials San Miguel is also responsible for managing a portfolio of Administrative Supplements (P30s) to the Cancer Center Support Grants at NCI-designated cancer centers. These administrative supplements constitute the National Outreach Network (NON) and play a critical role increasing accrual into cancer clinical trials, implementing the Screen to Save: NCI Colorectal Cancer Outreach and Screening initiative, and the HPV Health Education and Outreach initiative among racially/ethnically diverse, and rural, communities across the nation.

For the past five years, San Miguel has been leading global cancer control and prevention efforts working with the Pan American Health Organization/World Health Organization (PAHO/WHO) and various global NGOs. Through a groundbreaking collaboration among NCI, PAHO/WHO, and University of Texas MD Anderson Cancer Center, San Miguel is leading the development, implementation, and evaluation of Extension for Community Health Outcomes Latin America - ECHO ELA – collaborating with ministries of health, policy health makers, researchers, and key cancer stakeholders to promote WHO's cervical cancer elimination goals to prevent and treat cervical cancer.

Sandra San Miguel possesses over 25 years of experience in public health, is a published author of multiple peer-reviewed articles, manuals and booklets and has received numerous accolades and recognitions for her work. Her expertise lies in population health - developing/adapting, implementing, and evaluating evidence-based, culturally sensitive, multilingual behavioral cancer interventions to decrease health disparities among racially/ethnically diverse populations within the U.S. and among underserved populations globally. Prior to joining NCI, San Miguel served in academia, holding faculty positions at the Dept. of Medicine - Epidemiology & Biostatistics at UT Health San Antonio and at the Dept. of Biology and International Studies at Trinity University.

San Miguel is a doctoral candidate in Public Health at the University of Illinois at Chicago (UIC). She received her MS in Psychology from Our Lady of the Lake University in San Antonio, Texas, and BA in Psychology from the University of the Incarnate Word.

KEYNOTE SPEAKER

Joseph R. Osborne, MD, PhD

Chief of Molecular Imaging and Therapeutics and
Professor of Radiology at Weill Cornell Medicine
Attending Radiologist at New York-Presbyterian/Weill Cornell Medicine

Dr. Joseph Osborne is the Chief of Molecular Imaging and Therapeutics, Professor of Radiology at Weill Cornell Medicine and Attending Radiologist at NewYork-Presbyterian/Weill Cornell Medicine. Dr. Osborne is the Head of the Rad Health Equity Laboratory. The lab endeavors to move past advocacy into the practical implementation of projects and partnerships to reduce radiologic health disparities. He was the also the principle investigator on an NIH Academic Industrial Partnership RO1 grant "A new technique to make 68Ga-labeled pharmaceuticals widely available for clinical use" and the Dean's Health Disparity Research Award "Prostate Cancer Health Impact Program (pCHIP)".

MENTOR TRAINING WORKSHOP

Virginia Valian, PhD

Distinguished Professor of Psychology

Department of Psychology, Hunter College of The City University of New York

Virginia Valian is Distinguished Professor of Psychology at Hunter College - CUNY and is a member of the doctoral faculties of Psychology, Linguistics, and Speech-Language-Hearing Sciences at the CUNY Graduate Center. She directs the Language Acquisition Research Center (LARC) and the Gender Equity Project (GEP) at Hunter College.

Valian works on gender equity and on the psychology of language. In gender equity Valian performs research on the reasons behind women's slow advancement in the professions and proposes remedies for individuals and institutions. She is the author of Why so slow? The advancement of women, and co-author, with Abigail Stewart, of An inclusive academy: Achieving diversity and excellence. Valian consults with institutions and organizations to improve gender equity.

In the psychology of language Valian conducts research on young children's acquisition of syntax and on the relation between bilingualism and executive function in adults. Her aim is to develop a model of acquisition that specifies what is innate, how input is used by the child, and how the child's syntactic knowledge interacts with knowledge in other linguistic and extra-linguistic domains. She uses a variety of methods, including computer-assisted corpus analysis, comprehension experiments, elicited imitation experiments, and elicited production experiments.

ORAL PRESENTATION

Hiroshi Matsui, PhD

Professor, Department of Chemistry
Hunter College of The City University of New York

Professor Hiroshi Matsui teaches courses in biophysical chemistry and physical chemistry at Hunter College. After engineering a variety of peptide/protein assemblies (Chem. Soc. Rev., (2010) 39, 3499-3509), biomimetic autonomous motors (Nature Mater., (2012) 11, 1081-1085), and enzyme-mimicking peptides (J. Am. Chem. Soc., (2014) 136, 15893-15896), Dr. Hiroshi Matsui's nanotechnology labs have developed various nanoparticles including inorganic nanocages (Nature Commun., (2014) 5, 3870). Currently his lab is focused on medical applications of nanotechnology, including nanoparticle-based drug delivery systems, medical imaging, and lab-on-a-chip cancer diagnostic devices.

ORAL PRESENTATION

Mandë Holford, PhD

Associate Professor, Department of Chemistry

Hunter College of The City University of New York

Dr. Mandë Holford is a tenured Associate Professor in Chemistry at Hunter College and CUNY-Graduate Center, with scientific appointments at The American Museum of Natural History and Weill Cornell Medicine. Her research combines biological and chemical techniques to examine venoms and venomous animals. She is particularly interested in using venom peptides to study rapidly evolving genes and to examine the cellular physiology of malfunctioning signals in pain and cancer. Dr. Holford's lab applied an evolutionary venomics approach, integrating phylogenetic, transcriptomics, and proteomics, to investigate the evolution of venom in terebrid snails and to characterize their venom peptides. She was first to reconstruct the molecular phylogeny of the group and demonstrate the analgesic and antitumor activity of terebrid peptides. The Holford has recently focused on developing invertebrate venom gland model systems that can be genetically manipulated to study the molecular innovation of venom. She is active in science education, advancing the public understanding of science, and science diplomacy. She co-founded Killer Snails, LLC, an award winning EdTech company that uses tabletop, digital, and XR games about nature as a conduit to advance scientific learning in K-12 classrooms. Her honors include being named: a Sustainability Pioneer and Champion Scientist by the World Economic Forum, Breakthrough Women in Science by the Howard Hughes Medical Institute and NPR's Science Friday, a Wings WorldQuest Women of Discovery Fellow, a Camille Dreyfus Teacher-Scholar, an NSF CAREER awardee, a AAAS Science & Technology Policy Fellow, and Fellow of the California Academy of Sciences. She received her PhD from The Rockefeller University.

ORAL PRESENTATION

Jayashri Ghosh, PhD

Assistant Professor (Research)

Fels Cancer Institute of Personalized Medicine, Temple University

Dr. Jayashri Ghosh started her career in the field of epigenetics as a Post-Doctoral Fellow in Dr. Carmen Sapienza's laboratory at the Fels Cancer Institute for Personalized Medicine, Temple University. She has been associated with Sapienza lab for more than 8 years now and has been recently promoted to the rank of Assistant Professor (Research). Dr. Ghosh initial works have focused on looking at epigenetic differences between two or more groups and interpreting the data into a clinically relevant aspect of Assisted Reproductive Technology (ART). One of the most interesting finds of her ART research has been the identification of epigenetic outliers or "Outlier Methylation Phenotype" (OMP). Such outliers have abnormal methylation patterns throughout the genome. The concept of epigenetic outliers is not restricted to ART and the team has expanded the study on outliers to the cancer field (secondary analyses of TCGA datasets). Furthermore, their recent work on racial disparity in colon cancer patients shows that methylation differences exists between patients of different races and African Americans patients are more likely to be OMPs than Caucasians (TUFCCC/HC U54 Colon Pilot Study).

ORAL PRESENTATION

Aisha Bhimla, PhD, MPH

Postdoctoral Fellow, Center for Asian Health

Temple University Lewis Katz School of Medicine

Aisha Bhimla is a postdoctoral fellow at the Center for Asian Health at Temple University Lewis Katz School of Medicine. In this position, she provides support to federally funded projects that address health disparities among Asian American, Black/African American, and Latinx communities. Her involvement in health disparities research has included a broad range of community-based interventions and assessments related to chronic disease and cancer, physical activity, mental health, cognitive functioning, and teen pregnancy/STI prevention. She is specifically passionate about using epidemiological methods to understand how social determinants of health, such as the neighborhood environment affects health behaviors and risk factors associated with colorectal and liver cancers among racial and ethnic minority populations. She holds a Ph.D. in Kinesiology from Temple University and an MPH in Epidemiology and Global Health from the University of South Florida.

Session 1

Presenter 1 (10:00 am - 10:15 am)
Abstract #1: Erin Toussaint Jacques
A Content Analysis of Colorectal Cancer News Coverage: Disparities in Health Communication

Presenter 2 (10:20 am - 10:35 am)
Abstract #2: Cicely K. Johnson
Exploring the impact of mental health and trauma as predictors for CRC screening among Blacks in New York City

Presenter 3 (10:40 am - 10:55 am)
Abstract #3: Alvaro García
PINCH translational modifications affect chemotherapy susceptibility on GBM cells depending on p53 status

Presenter 4 (11:00 am - 11:15)
Abstract #4: Ra'Ann Merceir
Raising Awareness of STI Prevention: An Evidence-Based Sexual Health Education Program for High-Risk Youth

Presenter 5 (11:15 am - 11:30 am)
Abstract #5: Chidiebere Awah
Engineered destabilized AU rich elements on the 3'UTR of HER2 degrades HER2, inhibits proliferation, and induces apoptosis in HER2+ trastuzumab resistant breast cancer cells

Session 2

Presenter 1 (10:00 am - 10:15 am)
Abstract #6: George Kwakye Annor
Contributions of Mutant p53 Oligomerization and C-terminal Domains to Tumorigenic Gain-of-Function Activities

Presenter 2 (10:20 am- 10:35 am)
Abstract #7: Sakib Adnan
Trends in Lung Cancer Screening Practices: The Impact of the COVID-19 Pandemic

POSTER PRESENTATION

Presenter 3 (10:40 am - 10:55 am)
Abstract #8: Favour Achimba
Characterizing the Activity and Function of Transient Receptor Potential Channels in Liver Tumor Cells Using New Terebrid Snail Venom Peptide.

Presenter 4 (11:00 am - 11:15 am)
Abstract #9: Ellen Kim
Increasing Knowledge of Colorectal Cancer Risk Factors and Screening Through A Community-Based Education Initiative

Presenter 5 (11:15 am - 11:30 am)
Abstract #10: Jorge Canar
Impact of somatic STAT2 mutations on the antitumor type I interferon response in colon cancer

Session 3

Presenter 1 (10:00 am - 10:15 am)
Abstract #11: Nazia Nayeem
Preclinical Evaluation of a Potential Ruthenium-Based Chemotherapeutic Agent for the Treatment of Triple Negative Breast Cancer of Mutant p53 Oligomerization and C-terminal Domains to Tumorigenic Gain-of-Function Activities

Presenter 2 (10:20 am - 10:35 am)
Abstract #12: Sarah Shalan
Clinical implications of opioid use to treat cancer related pain

Presenter 3 (10:40 am - 10:55 am)
Abstract #13: Jonathan Zirkiev
Using HepG2 cells as a Hepatocyte Model to Study APOL1 Secretion onto TLF Complexes the Activity and Function of Transient Receptor Potential Channels in Liver Tumor Cells Using New Terebrid Snail Venom Peptide

Presenter 4 (11:00 am - 11:15 am)
Abstract #14: Tamar Tertulien
Healthy Eating for A Health Liver: Changes in Dietary Behaviors in Three Racial/Ethnic Minority Populations Through A Community-Based Liver Cancer Education Initiative

POSTER PRESENTATION

Presenter 5 (11:15 am - 11:30 am)
Abstract #15: Daniel Lopes
Characterization of TbCatL Recombinant protein in a CHO-S mammalian model

Session 4

Presenter 1 (10:00 am - 10:15 am)
Abstract #16: Avrosina Kamel
KRAS inhibitor treatment disparities in non-small cell lung cancer patients: increasing equity through precision medicine

Presenter 2 (10:20 am - 10:35 am)
Abstract #17: Tyrell Mann-Barnes
Anxiety and Depression Were Significant Moderators for the Association between Adverse Childhood Experiences and Quality of Life Among HIV Positive Men Who Have Sex with Men

Presenter 3 (10:40 am - 10:55 am)
Abstract #18: Dennis Huang
Understanding the mechanisms of gliomagenesis in oligodendrocyte progenitors, driven by Trp53 loss and Idh1 mutation.

Presenter 4 (11:00 am -11:15 am)
Abstract #19: Di Zhu
Are mHealth interventions on Hepatitis B Virus screening among Asian Americans Effective? A Scoping Review

Presenter 5 (11:15 am - 11:30 am)
Abstract #20: Jade Truehart
A Preliminary Look at Second Year Evaluation Data of a Sexual Health Intervention Program in Philadelphia

POSTER PRESENTATION

Session 5

Presenter 1 (10:00 am -10:15 am)
Abstract #21: Rusia Lee
Examining MDMX / MDM2 Signaling Pathways in Breast Cancers Expressing Mutant p53

Presenter 2 (10:20 am - 10:35 am)
Abstract #22: Kristine Chin
Building A Library of Patient Stories To Connect Communities To Lung Cancer Care And Screening

Presenter 3(10:40 am - 10:55 am)
Abstract #23: Priyanka Ghosh
Intracellular interaction of downstream molecular mediators of miR-1207-3p in prostate cancer cells

Presenter 4 (11:00 am - 11:15 am)
Abstract #24: Safa Ibrahim
A Community-Based screening intervention to Improve Colorectal Cancer Education for underserved African American, Asian American, and Hispanic American Communities in New York City

Presenter 5 (11:15 am - 11:30 am)
Abstract #25: Yassah Nupolu Lavelah
A Review of Existing Literature on Non-Alcoholic Fatty Liver Disease (NAFLD) among Asian Americans

Session 6

Presenter 1 (10:00 am - 10:15 am)
Abstract #26: Amy Yu
Common mRNA targets of Estrogen Receptor Alpha (ERα) and PARN in Breast Cancer Cells

Presenter 2 (10:20 am - 10:35 am)
Abstract #27: Samantha Boudeau
African ancestry-informative markers and the identification of population-specific disease loci in cancer

POSTER PRESENTATION

Presenter 3 (10:40 am - 10:55 am)
Abstract #28: Yuliya Severynenko
Understanding how Stanniocalcin-1 (STC1) affects Apolipoprotein-1 (APOL1) mediated cellular cytotoxicity

Presenter 4 (11:00 am - 11:15 am)
Abstract #29: Veronica Gomes
Ethnic Enclaves and Colon Cancer Stage at Diagnosis Among New Jersey Hispanics

Presenter 5 (11:15 - 11:30 am)
Abstract #30: Devorah Mincha Natelson
The RNA binding protein HuR mutation K313R suppresses ubiquitination by BRCA1/BARD1 and increases HuR accumulation

Session 7

Presenter 1 (10:00 am - 10:15 am)
Abstract #31: Anthony Ramadei
Regulation of p21 expression in the DNA damage response by calreticulin, CUGBP1, and a long non-coding RNA generated by alternative polyadenylation.

Presenter 2 (10:20 am - 10:35 am)
Abstract #32: Wenyue Lu
Depression Significantly Predicted Poor Medication Adherence Among Asian Americans Living with Chronic Hepatitis B: A Longitudinal Study

Presenter 3 (10:40 am - 10:55 am)
Abstract #33: Jessica Magarinos
Single-Encounter Telemedicine (SET) Lung Cancer Screening Reveals Lower Rates Of Screening But Preserves Access to Minorities

Presenter 4 (11:00 am - 11:15 am)
Abstract #34: Zachary Wilmer Reichenbach
Investigating the cellular and molecular factors contributing to health disparities in esophageal cancer

POSTER PRESENTATION

Session 8

Presenter 1 (10:00 am - 10:15 am)
Abstract #35: Jason S. Wasserman
FAM122A is a substrate-competitive inhibitor of the B55α/PP2A phosphatase required for timely progression through the cell cycle interphase

Presenter 2 (10:20 am - 10:35 am)
Abstract #36: Julia N. Trout
Measuring prevalence of psychosocial-related factors in predicting depressive symptoms of cancer patients and their primary caregivers

Presenter 3 (10:40 am - 10:55 am)
Abstract #37: Zobaida Maria
The Relationship Between Medical Mistrust and Compliancy with Follow-Up Care in

Presenter 4 (11:00 am - 11:15 am)
Abstract #38: Amy Alvarado
STAT2 Signaling Reprograms Lipid Metabolism in Colorectal Cancer

Presenter 5 (11:15 am - 11:30 am)
Abstract #39: Fayola Levine
Investigating the clinical relevance in prostate cancer of the serum biomarkers PVT1 exons 4A, 4B and 9 across risk levels and ethnicity/race

Session 9

Presenter 1 (10:00 am - 10:15 am)
Abstract #40: Nora Kimbrough-Perry
Graph convolutional neural networks applications to cancer cellular network analysis

Presenter 2 (10:40 am - 10:55 am)
Abstract #41: Lynde Lutzow
Lung Cancer Screening in the COVID-19 Era: Understanding Program-Level Impact

Presenter 3 (11:00 am - 11:15 am)
Abstract #42: Kenneth Bonett
Opti-Health: A Phone Application for Lung Cancer Screening

Presenter 4 (11:15 am -11:30 am)
Abstract #43: Chelsea Yu
Cancer prognosis with tissue birefringence measured by label-free quantitative microscopy

4th Annual SPEECH Regional Cancer Health Disparity conference Abstracts

ACKNOWLEDGEMENTS

The SPEECH Conference Planning Team wishes to thank the many individuals and groups who assisted in the efforts to organize this year's regional conference.

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The SPEECH Conference is supported by the Temple University, Fox Chase Cancer Center and Hunter College U54 Regional Comprehensive Cancer Health Disparity Partnership (1U54CA221704 and 1U54CA221705), funded by NIH/NCI.

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Abstract#1

A Content Analysis of Colorectal Cancer News Coverage: Disparities in Health Communication

Erin T. Jacques, Ed.D., Ph.D.[1], Grace C. Hillyer, Ed.D.[2], Corey H. Basch, Ed.D., MPH[3]

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[3]Department of Public Health, William Paterson University, Wayne, NJ 07470

To address an uptick in colorectal cancer (CRC) morbidity and mortality rates, the United States Preventive Services Task Force (USPSTF) lowered the recommended ages for colorectal cancer (CRC) screening from 50 to 45. Nonetheless, research on how CRC is represented in the news is lacking. Thus, the aim of this study was to describe the content of Google news stories related to CRC. Using the term colorectal cancer, researchers coded 100 most recent relevant Google news stories published between June 2021 to March 2022. Researchers used a deductive coding process to capture the dichotomous categories of risk factors and the mention of screening, disparities, mortality, severity, fear of screening, insurance/costs, colonoscopy, the spread of cancer, treatment, and research. An inductive approach elucidated the source of the news stories to include the internet, traditional, health, consumer, education, non-health organization, academic journal, and the government. Of the 100 videos reviewed, nearly half (49%) were created by health news organizations and 27% by traditional news services. Only 18% discussed CRC disparities. In contrast, most videos mentioned CRC screenings (61%). Themes that predominated among videos that mentioned CRC screening more often talked about risks for developing CRC, including the age at onset (75.4% vs. 33.3%, $p<0.001$), CRC mortality (73.8% vs. 30.8%, $p<0.001$), and severity of disease (60.7% vs. 33.3%, $p=0.008$). Google news articles did not adequately highlight disparities in CRC morbidity and mortality. The results of this study support a need to communicate information on screening and early-onset CRC, particularly among communities of color.

Abstract #2

Effective Recruitment Strategies Utilized to Examine Dietary Practices of Blacks In New York City in the Midst of a COVID-19 Pandemic

Cicely K. Johnson, PhD,[1] Grace X. Ma, PhD[4], May May Leung, PhD[3] , Olorunseun O. Ogunwobi, MD, PhD[2]

[1]Hunter College Center for Cancer Health Disparities Research, Hunter College of the City University of New York
[2]Department of Biological Sciences, Hunter College of the City University of New York
[3]Nutrition Program, School of Urban Public Health, Hunter College of the City University of New York
[4]Center for Asian Health, Temple University, Pennsylvania

African Americans have the highest colorectal cancer incidence and mortality rates of all racial groups in the U.S. Factors that may be contributing to these high rates, however, remain poorly understood. Specifically, this study assessed the relationship between dietary habits and colorectal cancer screening behavior and intent among individuals who identify as Black, with their ethnicity as African-American, Caribbean, and African. There are many factors that influence dietary habits, and a salient factor is culture. Many studies have experienced challenges recruiting in communities of color for various reasons. The purpose of this presentation is to describe recruitment methods utilized for this study in the midst of the COVID-19 pandemic, and to discuss challenges, strategies that were implemented and lessons learned that can inform and improve future recruitment efforts. Effective recruitment strategies included partnership, consistent engagement, and meeting people in places where they frequent and felt comfortable. Utilizing faith-based locations, barbershops, hair salons, and pivotal community locations allowed individuals to trust the researchers, and also eliminated the need to retain study subjects over a period of time, due to on-site data collection. Though our findings are limited to Black families in predominantly minority neighborhoods, we have identified successful strategies for this specific high-risk population and potentially similar others.

Abstract #3

PINCH translational modifications affect chemotherapy susceptibility on GBM cells depending on p53 status

Garcia-Blanco A.[1] Rahman A., Imbert F[1]., Tice C., Kalimuthusamy N., Langford D.[1]

[1]Department of Neural Sciences, Lewis Katz School of Medicine, Temple University, Philadelphia, PA

Gliomas are the most common adult CNS tumors and despite treatment efforts including surgery, radiation, and chemotherapy, essentially all low-grade gliomas progress to glioblastoma multiforme (GBM). Surgical resection is usually not possible and the recurrence rate of GBM is almost 100% after 9 months with a median survival of approximately 15 months due to its high capacity for invasion and resistance to therapy. Thus, improved therapeutic approaches are needed to decrease recurrence and prevent progression of astrocytoma into GBM. The PINCH protein is expressed in the mature CNS in neuropathological conditions, including Alzheimer's Disease, HIV infection, or glioma. PINCH-mediated signaling involves cell migration, spreading, and survival pathways which are all critical events in cancer progression. In fact, increased PINCH expression is related to poor prognosis in colorectal, pancreatic and breast cancer. Our new data link PINCH expression with the anti-oncogenic gene p53, suggesting that the two proteins work in concert in progression to glioblastoma (GBM). p53 is mutated in approximately 80% of gliomas leading to p53 pathway deregulation that contributes to chemotherapy resistance. Our data show that PINCH is dramatically increased in brain cancers as a function of grade of malignancy. We also observed a PINCH post-translational modification linked to p53 mutation in glioma cell lines. Since PINCH expression levels and post-translational modifications are linked to vulnerability of GBM cells to therapeutic intervention, findings from these studies will provide valuable data for potential adjuvant therapies for GBM and possibly other cancers.

Abstract #4

Raising Awareness of STI Prevention: An Evidence-Based Sexual Health Education Program for High Risk Youth

Ra'Ann Merceir, BS[1]; Jade Truehart, MPH[1]; Aisha Bhimla, PhD[1], MPH; Yin Tan, MD, MPH[1]; Lin Zhu, PhD[1]; Jane Sileo[2]; Min Qi Wang[3], PhD; Sabrina Liao[3]; Julia Trout, BA[1]; Ellen Kim, MS[1]; Aimme Bogan, MA,LPC[4]; Grace X Ma, PhD[1]

[1]Center for Asian Health, Lewis Katz School of Medicine, Temple University, PA

[2] ACT Investments, Inc.

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[4] Maritime Academy Charter High School

Background: HPV, the most common STI, is the major cause of various cancers including cervical, penile, and oropharyngeal cancer. Approximately 25% of Philadelphia's STI cases are among teens aged 15-19. Proud Teens of Philly (PTOP) is a youth sexual health education program to promote healthy choices and ultimately reduce cancer risk. **Methods:** Participants (12-19 year-olds) recruited from schools and CBOs attended 8 synchronous 1-hour sessions delivered virtually. Making Proud Choices, an evidence-based curriculum, aimed to reduce risky behaviors. We administered pre- and post-surveys to 246 youth to understand students' intention to have sex, perceived susceptibility to STIs/HIV, and attitudes toward sex, condom use, and STIs. We used paired samples t-tests to evaluate differences before and after the program.

Results: There were significant increases in positive attitudes towards condom use (pre=26.54, post=27.73, t=-2.18, p=0.03), more negative attitudes towards STIs/HIV (pre=7.62, post=6.65, t=5.22, p<0.001), and increased perceived susceptibility towards contracting STIs/HIV following the intervention (pre=4.41, post=5.39, t=-2.84, p<0.01). There were no significant changes in intentions to have sex in the next three months (pre=2.03, post=2.11, t=-0.66, p=0.51) or attitudes towards having sex (pre=21.58, post=21.20, t=1.09, p=0.28).

Conclusion: Implementing PTOP in high-risk youth can mitigate HPV rates by increasing positive attitudes towards barrier protection. We will discuss the successes and challenges in the implementation and evaluation process, as well as plans for moving forward.

Acknowledgement: This project was supported by the Philadelphia Teen Outreach Project (PTOP) funded by the Department of Health and Human Services (DHHS), Award Number TP1AH000219 (PI: Ma), and partially supported by TUFCCC/HC Regional Comprehensive Cancer Health Disparity Partnership, Award Number U54 CA221704(5) from the National Cancer Institute of National Institutes of Health (NCI/NIH). Its contents are solely the responsibility of the authors and do not necessarily represent the official views of the NCI/NIH or DHHS.

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Abstract #5

Engineered destabilized AU rich elements on the 3'UTR of HER2 degrades HER2, inhibits proliferation, and induces apoptosis in HER2+ trastuzumab resistant breast cancer cells

Chidiebere U. Awah[1,2], Yana Glemaud[1], Fayola Levine[1,3], Leonard Ash[1], Afrin Ansary[1], Olorunseun Ogunwobi[1,2,3]

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Resistance to molecular targeted therapy is a major challenge facing breast cancer patients and unfavorably impacts clinical outcomes, leading to hundreds of thousands of deaths yearly. Resistance to cancer therapies arises from many factors. One of the key factors driving resistance is oncogenes which initiate pro-growth, pro-survival and metastatic programs that enable cancers escape various therapies. In HER2+ breast cancer, the oncogene HER2 is the master driver of this tumor. Trastuzumab effectively targets HER2. While this is generally successful, some patients who have HER2+ breast cancer develop resistance to trastuzumab and there are limited treatment options for them. Moreover, for low HER2+ expressing breast cancer, there are limited treatment options for them. And in African American women who have HER2+ breast cancer, there is disparity in their treatment outcomes. We have discovered that 3'UTR of HER2 is enriched with poly U stabilizing AU rich elements (ARE). We have developed a novel technology wherein we engineered the stabilizing HER2 3'UTR ARE motifs to destabilizing motif in HER2+ and HER2+ trastuzumab resistant breast cancer cells to control HER2 transcript. We achieved complete degradation of HER2 within 4-8 days in HER2+ wildtype breast cancer cells and 9-11 days in HER2+ trastuzumab resistant breast cancer cells. We had loss of cancer cell viability by more than 90%. We also show that control of HER2 transcript downregulated HER2-dependent kinases, transcription factors, and HER2 interactome. Mechanistically, the control of HER2 transcript was achieved by destabilized ARE sequence specificity which triggered the proteins PARN, XRN1 and CNOT1 to degrade HER2 transcript and this led to induction of active caspase 3/7, reduction of cell size, and severe distortion and disruption of the cancer cell membrane leading to cell death. Taken together, we have developed a novel approach to control HER2 transcript expression both on spatial and temporal scale. This novel approach offers applications for targeting HER2+ trastuzumab resistant breast cancer as well as other cancers resistant to HER2 targeted therapy and driven by pervasive oncogenic signals.

Abstract #6

Contributions of Mutant p53 Oligomerization and C-terminal Domains to Tumorigenic Gain-of-Function Activities

G.K. Annor[1,2], G. Xiao[2], S. Maimos[2], DG. Conde[2], and J. Bargonetti[1,2,3]

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[2]The Department of Biological Sciences Hunter College
[3]Weill Cornell Medical College, New York City

To address an uptick in colorectal cancer (CRC) morbidity and mortality rates, the United States Preventive Services Task Force (USPSTF) lowered the recommended ages for colorectal cancer (CRC) screening from 50 to 45. Nonetheless, research on how CRC is represented in the news is lacking. Thus, the aim of this study was to describe the content of Google news stories related to CRC. Using the term colorectal cancer, researchers coded 100 most recent relevant Google news stories published between June 2021 to March 2022. Researchers used a deductive coding process to capture the dichotomous categories of risk factors and the mention of screening, disparities, mortality, severity, fear of screening, insurance/costs, colonoscopy, the spread of cancer, treatment, and research. An inductive approach elucidated the source of the news stories to include the internet, traditional, health, consumer, education, non-health organization, academic journal, and the government. Of the 100 videos reviewed, nearly half (49%) were created by health news organizations and 27% by traditional news services. Only 18% discussed CRC disparities. In contrast, most videos mentioned CRC screenings (61%). Themes that predominated among videos that mentioned CRC screening more often talked about risks for developing CRC, including the age at onset (75.4% vs. 33.3%, $p<0.001$), CRC mortality (73.8% vs. 30.8%, $p<0.001$), and severity of disease (60.7% vs. 33.3%, $p=0.008$). Google news articles did not adequately highlight disparities in CRC morbidity and mortality. The results of this study support a need to communicate information on screening and early-onset CRC, particularly among communities of color.

Abstract #7

Social Determinants of Lung Cancer Screening: Knowledge Gaps and Controversies

[1]**Sakib M. Adnan** MD, [1]Kristine Chin, [2]Grace X. Ma, PhD, [1]Cherie P. Erkmen, MD

[1]Temple University Hospital, Philadelphia, PA; [2]Center for Asian Health, Lewis Katz School of Medicine at Temple University, Philadelphia, PA

Lung cancer is the most prevalent malignancy and most common cause of cancer-related death worldwide. While the utility of routine lung cancer screening (LCS) with low-dose computed tomography (LDCT) had previously been under debate, its mortality benefit is now established after reports from multiple prospective trials. In response, there has been an expansion of LCS programs for asymptomatic, high-risk individuals, with downstream increases in diagnosis of early-stage disease and long-term survival. Specifically, the Center for Medicare & Medicaid Services instituted LCS coverage in February 2015 and expanded screening eligibility in February 2022. However, these reassuring metrics are accompanied by limitations in screening practices, especially when applying LCS recommendations toward marginalized groups. Historically, cancer screening guidelines are drawn from clinical trials that largely underrepresent minorities and women. This has fostered further discussion on screening strategies and protocols, exposing a need for strategic screening practices for marginalized groups and minority populations. The aim of the current clinical practice review is to present these knowledge gaps and controversies, with a focus towards disparities and social determinants associated with lung cancer management.

Abstract #8

Characterizing the Activity and Function of Transient Receptor Potential Channels in Liver Tumor Cells Using New Terebrid Snail Venom Peptide

Favour T. Achimba[1,2], Petr Filipenko[1], Roland Dunbrack[3,4,5], Joan Font- Burgada[4], Mande Holford[1,2,6]

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[2]Department of Biochemistry, Graduate Center, City University of New York
[3]University of Pennsylvania School of Medicine
[4]Fox Chase Cancer Center, Temple University
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The regulation of cellular processes by ion channels has become central to the study of cancer mechanisms. Designing molecules that can target ion channels overexpressed in tumor cells is a promising area for finding selective cancer therapies. Although natural products have played a significant role in drug development, venom peptides are a largely untapped class of compounds. The exquisite specificity and selectivity of venom peptides for modulating ion channels and receptors highlights their promise for drug discovery and development. Hepatocellular carcinoma (HCC) is the most common form of primary liver cancer worldwide accounting for about 90% of liver-cancer related deaths. It is difficult to detect until it has progressed to the advanced stages resulting in high death rates. Sorafenib, a molecular targeted therapeutic, has proven effective in treating advanced HCC, but only improved survival rate by a few months and is cytotoxic to normal cells. Hence, there is a need for targeted therapy with higher specificity and less cytotoxicity to non-tumor bearing cells. Our previous studies showed the potential of terebrid venom peptide (Tv1) as a selective inhibitor for HCC tumors in 1MEA cell lines. These findings led to the working hypothesis that teretoxin peptides sharing a cysteine framework similar to that of Tv1 will have similar antitumor effects and interact in similar ways with TRP channels to exert the antitumor effect. This work will identify and functionally characterize the anti-tumor mechanism of action of Tv1 and other terebrid venom peptides (teretoxins) that target Transient Receptor Potential (TRP) channels overexpressed in HCC. From our library of 1157 teretoxin, we identified peptides with a similar cysteine framework to Tv1 and those with homology to published solved structures. Using trRosetta and AlphaFold we predicted the structures of the selected peptides. Peptides with a structural homology to Tv1 were then docked to various TRPC and TRPV channels overexpressed in HCC. Here we describe our initial results that validate the use of teretoxins to target overexpressed TRP channels in HCC to minimize or prevent cancer progression.

Abstract #9

Increasing Knowledge of Colorectal Cancer Risk Factors and Screening Through A Community-Based Education Initiative

Ellen Kim, BA;[1] Lin Zhu, PhD;[1,2] Wenyue Lu, ML, PhD(c);[1] Safa Ibrahim;[7] Steven Zhu;[3] Nathaly Rubio-Torio, LMSW;[4] Evelyn González, MA;[5] Marilyn A. Fraser, MD;[6] Ming-Chin Yeh, PhD;[7] Grace X. Ma, PhD;[1,2] Olorunseun O. Ogunwobi, MD, PhD;[8] Yin Tan, MD, MPH;[1]

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[5] Office of Community Outreach, Fox Chase Cancer Center, Temple University Health System, Philadelphia, PA
[6] Arthur Ashe Institute for Urban Health, New York City, NY
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[8] Department of Biological Sciences, and Department of Psychology, Hunter College, City University of New York, New York, NY

Introduction: Disparities in incidence and mortality in colorectal cancer (CRC) continue to persist between racial/ethnic minority populations and non-Hispanic whites, despite the provision of widespread screening and improved treatments for CRC. The cause for the disparities in incidence and mortality is multifactorial. One important aspect is the suboptimal knowledge and awareness of risk factors and the lack of access to screening in racial/ethnic minority populations.

Methods: To increase awareness of CRC prevention and screening, we trained 20 community health workers affiliated with our community partners and jointly designed and conducted an educational initiative in African, Asian, and Hispanic American communities in the greater Philadelphia area and New York City. We administered surveys before and after the intervention workshops to assess the impact of the educational sessions.

Results: The analysis sample included 413 participants, among which 388 completed the post-survey. One in ten (10.3%) reported a family history of CRC, and half (50.6%) had never had a colonoscopy. The baseline CRC knowledge score was 9.5 out of 16, indicating a moderately low level of knowledge. Participants scored particularly low on the age of CRC screening initiation, needs for screening even without symptoms, and the impact of physical activity on CRC risk. The knowledge score significantly increased to 10.9 ($p < .001$) at post-survey, indicating a significant impact of the educational workshops.

Conclusion: Culturally-tailored, community-based educational initiatives are effective in raising knowledge of CRC in medically underserved populations. We will discuss the successes and challenges in the implementation process of the initiative.

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Abstract #10

Impact of somatic STAT2 mutations on the antitumor type I interferon response in colon cancer

Jorge Canar[1] and Ana Gamero, PhD[1]

[1]Lewis Katz School of Medicine at Temple University, Department of Medical Genetics and Molecular Biochemistry

Colorectal cancer (CRC) is the third deadliest cancer in the US, with an alarming increase in incidence rates in young adults, especially among African Americans and non-Hispanic Whites. There is an urgent need to better understand the biology of this disease to identify new targets and develop novel therapeutic interventions. Signal transducer and activator of transcription 2 (STAT2) is a positive effector of antitumor type I interferon (IFN-I) signaling. In previous studies, we reported that, paradoxically, STAT2 is tumorigenic in CRC. Our studies now show STAT2 protein is elevated and activated in human colorectal tumors. We analyzed TCGA-CRC datasets and found high STAT2 mRNA levels associated with poor prognosis. We also identified five somatic STAT2 missense mutations of interest that based on their location, could potentially alter IFN-I responses in CRC. Of particular interest was R148>W because it was identified as a lethal homozygous germline mutation in children born with a condition known as type I interferonopathy. To interrogate their functionality, we generated STAT2 deficient colon cancer cell lines and reintroduced different STAT2 mutations individually. Western blot analysis showed STAT2 mutants are expressed to comparable levels as wild-type STAT2. To determine their transcriptional activity in response to IFN-I, we measured luciferase reporter transcriptional activity. Interestingly, STAT2 R148W displayed reduced activity to wild type STAT2, in stark contrast to the expected phenotype of aberrant IFN-I signaling. Similar responses were seen with STAT2 R310C. These novel findings reveal somatic STAT2 mutations in CRC can impair IFN-I signaling and potentially their antitumor effects.

Abstract #11

Preclinical Evaluation of a Potential Ruthenium-Based Chemotherapeutic Agent for the Treatment of Triple Negative Breast Cancer

Nazia Nayeem[a,b,c], Rachele Rameau[a,d], Karen Hubbard[a,d], Maria Contel[a,b,c]

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[b]CUNY Brooklyn College Cancer Center
[c]CUNY Brooklyn College Chemistry Department
[d]CUNY City College Biology Department

Triple negative breast cancer (TNBC) is an aggressive subtype of breast cancer defined by the absence of expression of progesterone, estrogen, and human epidermal growth factor 2 receptors, with a higher incidence rate in pre-menopausal and women of African ancestry. Due to the inability to target a receptor, treatment is limited to nonspecific chemotherapy. A potential ruthenium-based chemotherapeutic agent has been developed, with preclinical evaluation of this complex showing significant anti-angiogenic, anti-invasive, and anti-migratory properties, along with subcellular accumulation in the mitochondria, increased ROS generation, and an apoptotic mechanism of cell death in both European and African-derived TNBC cell lines in vitro. Proteomic and epigenetic analysis has implicated the inhibition of PI3K/Akt pathway as a potential target of this drug, and a future in vivo study will be completed for the purposes of enhanced pharmacokinetic and molecular analysis from an initial study that showed 56% tumor shrinkage in TNBC MDA-MB-231cell line xenograft.

Abstract #12

Clinical implications of opioid use to treat cancer related pain

Sarah Shalan[1], Laura Schienfeldt, PhD[1]

[1]Coriell Institute for Medical Research, Camden, NJ

The opioid epidemic is a serious national crisis that affects both public health and social and economic welfare. For decades, opioids have been prescribed for the treatment of acute and chronic pain, including chronic pain associated with cancer treatment. Long term opioid use for chronic pain treatment continues to be controversial. Concerns related to the effectiveness, safety, and risk of misuse persist. The risk benefit assessment of opioid use for cancer treatment related chronic pain is still unresolved; however, safe and effective cancer treatment related pain management is needed. In this analysis, we assessed two large-scale reports of SEER-medicare data collected from thousands of patients. The first study included tens of thousands of cancer patients (n=69,889) and matched controls (n=125,007), and the second study included over ten thousand (n= 10,773) breast cancer patients. Our literature review found that previous incidence of depression or substance abuse disorder increased the risk of developing opioid use disorder in cancer patients treated with opioids, and longer-term exposure to opioids during cancer treatment dramatically increased the risk of opioid use disorder in cancer patients treated with opioids. In addition, other negative outcomes, including treatment non-compliance must be considered when using opioids to treat cancer treatment related pain.

Abstract #13

Using HepG2 cells as a Hepatocyte Model to Study APOL1 Secretion onto TLF Complexes

Jonathan Zirkiev[1], Jyoti Pant[1], Jayne Raper[1,2]

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[2]. PhD Program in Biochemistry, The Graduate Center of the City University of New York, New York, New York, USA

APOL1 is the lytic component of the high-density lipoprotein called trypanosome lytic factor (TLF). It protects against trypanosomes in humans and other higher-order primates. APOL1 has selectively evolved into variants called G1 and G2 in individuals of African descent. These variants are associated with increased risk of chronic kidney diseases, autophagy, and cancer. APOL1 in plasma is predominantly secreted from the liver. Here we used liver hepatocellular carcinoma cells (HepG2) cells to study the secretion of APOL1 and its assembly into the TLF complex. Our results show that APOL1 is produced and secreted by HepG2 cells. Upon induction with gamma interferon, the intracellular APOL1 production increases (90% to 99.8%). However, the secretion of the protein into the media decreases upon induction with interferon (10% to 0.2%). Size exclusion chromatography shows that secreted APOL1 is distributed among complexes of various sizes, from 63.5 kDa to 711.7 kDa. Based on these results we conclude that HepG2 cells can be used to study the secretion of APOL1 and assembly of TLF complexes (500kDa). In the future we intend to investigate the composition and trypanolytic activity of the secreted APOL1 containing complexes.

Abstract #14

Healthy Eating for A Health Liver: Changes in Dietary Behaviors in Three Racial/Ethnic Minority Populations Through A Community-Based Liver Cancer Education Initiative

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Background: There is an increasing body of literature that suggests a relationship between modifiable dietary behaviors and alcohol use and liver cancer. We designed and implemented a culturally tailored community-based education program to promote liver cancer prevention.
Methods: Using CBPR approach, we implemented an educational initiative to promote liver cancer in the community settings in African, Asian, and Hispanic American communities in the Philadelphia metropolitan area and New York City. In this study, we used data from the pre-education surveys and follow-up assessments at 6 months post-education to assess the changes dietary behaviors and alcohol consumption among participants.
Results: The analysis sample consists of 344 participants recruited through community-based organizations, including 31 African Americans, 174 Asian Americans, and 139 Hispanic Americans. Among African American participants, the consumption scores of fruits (2.170 to 3.581) and poultry (2.613 to 3.677) significantly increased. In Asian Americans, the scores of non-refined cereals (3.362 to 4.282), fruits (3.385 to 4.374), red meat (2.655 to 3.195), poultry (2.420 to 3.736), and dairy products (2.851 to 3.701) significantly increased; consumption of vegetables (3.908 to 3.236) decreased. In Hispanic participants, the consumptions of poultry (2.173 to 3.554), dairy products (2.101 to 3.863), olive oil (3.022 to 3.489) went up significantly.
Conclusion: This community-based educational imitative generated different impacts in the three populations, further highlighting the needs for more targeted, culturally tailored efforts in health promotion among these underprivileged communities.
Acknowledgement: This study supported by TUFCCC/HC Regional Comprehensive Cancer Health Disparity Partnership, Award Number U54 CA221704(5) from the National Cancer Institute of National Institutes of Health (NCI/NIH) and by NIGMS/NIH award # 1SC3GM131949-01 (PI: Ming-Chin Yeh). Its contents are solely the responsibility of the authors and do not necessarily represent the official views of the NCI/NIH or the NIGMS/NIH.

Abstract #15

Characterization of TbCatL Recombinant protein in a CHO-S mammalian model

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Trypanosoma brucei, known to cause African Trypanosomiasis in the sub-Saharan populations of Africa, is a eukaryotic parasite that killed many people. Humans have been able to develop a resistance to this affliction with a trypanolytic lipoprotein, apolipoprotein-1 (APOL1) G0, that forms ion channels in the parasite membranes and causes lysis. However, this never-ending battle continues with the *Trypanosoma brucei* Cathepsin-L (TbCatL), a protease, is hypothesized to degrade APOL1 G0 and thus allows for the evasion of trypanolysis.

To test this hypothesis, pure recombinant TbCatL will be incubated with rAPOL1 embedded in an artificial lipid bilayer, which mimics the membranes of the parasite. The rAPOL1 will be activated, which results in open channel conformation and ion flux. If rTbCatL can degrade APOL1 in the bilayer, we hypothesize that the ion flux will stop. The transformation of Chinese Hamster Ovary suspension cells (CHO-S) expression with a pcDNA/TbCatL vector will provide rTbCatL needed for experimentation, generating high quality and functional protease that can be purified via proteolytic activation. Work to optimize this process to obtain purer rTbCatL is underway.

Using this novel expression system, we will evaluate if rTbCatL can degrade and inactivate rAPOL1 G0 channels in membranes. With the model, further investigations can be made into how rTbCatL interacts with APOL1 G1 and G2 variants, new participants of the escalated immune arms race, that display a resistance to *Trypanosoma brucei* that utilize the protease to evade trypanolysis but to the detriment of renal function and health in human African populations.

Abstract #16

KRAS inhibitor treatment disparities in non-small cell lung cancer patients: increasing equity through precision medicine

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KRAS is a proto-oncogenic GTPase that normally activates pathways involved in cell growth, differentiation, and survival. KRAS is mutated in about 25% of all cancers and 33% of lung cancer. A first-in-class KRAS G12C inhibitor approved in 2021, sotorasib, is dependent on mutant cysteine at the 12th codon for its inhibitory function. Most patients relapsed after ~6 months, indicating development of resistance mechanisms. Therefore, we sought to identify synthetic lethal drug combinations to eliminate possible rerouting mechanisms of KRAS activity. A combinatorial drug screen identified GSK3β as a possible candidate for this interaction. We tested two GSK3β inhibitors – Kenpaullone and SAR502250 – in a combination drug experiment with sotorasib; however, the results were not indicative of synergy. Several possible effector pathways linked KRAS and GSK3β. Immunoblots to test proposed pathway interactions identified changes in inhibitory phosphorylation of GSK3β induced by SAR502250. Disparity trends exist in the population prevalence of the targetable KRAS allele, recruitment in clinical trials, and objective response rates (ORR). G12C mutations were more prevalent in White populations than in Black or Asian populations. In the Phase I/II clinical trials for sotorasib, 102 White, 18 Asian, and 2 Black patients were recruited. The drug's ORR was 18% in Asian populations, 40% in White populations, and 0% in Black populations, indicating a clear treatment disparity. Understanding why some populations tend to display enhanced drug resistance is critical to increasing equity in cancer therapy, emphasizing the need to expand treatment modalities.

Anxiety and Depression Were Significant Moderators for the Association between Adverse Childhood Experiences and Quality of Life Among HIV Positive Men Who Have Sex with Men

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Background: The effects of childhood trauma on quality of life among HIV positive men who have sex with men (MSM) living in the United States are poorly understood. This study investigated the relationship between early childhood trauma and health related quality of life in HIV positive MSM, and whether anxiety and depression moderate that relationship.

Methods: Online surveys were administered to eligible participants who identified as MSM, living with HIV, and resided in Philadelphia or Hawaii (n=214). Participants were recruited through hospital and HIV clinics through referrals, and through community-based organizations. A linear regression model was used to assess if childhood trauma measured by Early Trauma Inventory (ETI) was a predictor of Health Related Quality of Life (HRQOL). Moderation analyses were conducted to determine whether anxiety, measured by the generalized anxiety disorder scale and measured by the Center for Epidemiologic Studies Depression Scale, moderated the relationship between childhood trauma and HRQOL. **Results:** A one unit increase in ETI (β= -0.637, $p<0.001$), was negatively associated with HRQOL. Depression (β= 0.0772, $p<0.001$) and anxiety (β= -0.0443, $p<0.005$) were shown to moderate the association between ETI and HRQOL. **Conclusions**: The results show evidence that anxiety and depression moderate the association between ETI and HRQOL in HIV+ MSM. With higher levels of depression and ETI among participants, the number of unhealthy days reported also increases. With higher levels of anxiety and ETI, participants are observed reporting lower unhealthy days. These findings will help create better trauma informed approaches that increase HRQOL in HIV+ MSM.

Understanding the mechanisms of gliomagenesis in oligodendrocyte progenitors, driven by Trp53 loss and Idh1 mutation

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Gliomas are the most common and lethal tumors of the adult brain. Through the combination of large scale transcriptional and genome-wide associative studies, this cancer has been classified into several subtypes, characterized by distinct mutations and expression signatures. One such subtype of interest is proneural glioma, characterized by increased PDGFRα signaling, loss of TP53, and IDH1 mutation. PDGFRα is a well-established marker of oligodendrocyte progenitors (OPCs), the precursors to myelinating oligodendrocytes of the central nervous system and largest body of proliferating cells in the adult brain. Follow up studies have shown tumors formed from mutated OPCs observed in vivo. The mechanisms which drive tumorigenesis in OPCs are poorly understood but could be elucidated by investigations toward the intrinsic epigenetic landscape of tumorigenic cells. Both the lineage and differentiation process of oligodendrocytes depends on specific changes in the epigenome, mediated by histone and DNA methylation. In addition, previous studies and preliminary results suggest that the mutations found in proneural glioma (TP53 loss, IDH1 mutation) alter repressive histone modifications in different ways. This preliminary finding supports the hypothesis of the epigenome as a driving force of tumorigenesis. This project aims to answer key questions about the epigenetically driven mechanisms in OPCs leading to transformation in a well-established glioma model in mice by utilizing advanced sequencing techniques. ChIP-seq will characterize the genomic distribution of expression regulating histone marks in mutant OPCs harboring Trp53 deletion and Idh1 mutant expression. Single cell multiomics will characterize both chromatin accessibility and gene expression in tumor tissue at single cell resolution. These data may lead to better understanding of the transforming cell population in early proneural glioma and better therapeutic strategies which target genome-wide drivers of gliomagenesis.

Are mHealth interventions on Hepatitis B Virus screening among Asian Americans Effective? A Scoping Review

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Background: Hepatitis B virus (HBV) is one of the major health threats causing liver cancer. Asian Americans are especially vulnerable to HBV infection risk compared to other racial/ethnic groups in America. New mHealth interventions have promising potential to save lives by increasing HBV screening among racial/ethnic minority groups, but its scope, range, and uptake among Asian Americans are not clear. This study aims to scope the literature describing the research of mHealth interventions on HBV screening among Asian Americans.

Methods: A scoping systematic review search is conducted in PubMed and Embase. Eligible studies focus on Asian Americans aged 18 years or older who have participated in an mHealth Hepatitis B screening. All types of mHealth interventions on HBV are relevant (e.g., health apps and text messages). Studies not published in the English language will be excluded.

Results: A review protocol, including systematic search and records screening, is under development. Initial results will be discussed.

Potential impact: The results of this scoping and systematic review could improve our understanding of mHealth HBV screening use and uptake among Asian Americans and inform the development of a future intervention to encourage mHealth HBV screening for this patient population.

A Preliminary Look at Second Year Evaluation Data of a Sexual Health Intervention Program in Philadelphia

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Background: Proud Teens of Philly (PTOP) is a sexual health program aimed at reducing the high teen birth rate and STD cases in Philadelphia. The teen birth rate in Philadelphia is 28.8 per 1,000, and about 25% of its STI cases are among teens 15-19. PTOP provides sexual health education to high-risk youth ages 12-19 in the Greater Philadelphia area. In the first year of the program, PTOP was delivered to 304 youth.
Methods: During the second year of the program, PTOP was implemented virtually and in-person to 411 youth in Philadelphia schools and community-based organizations. The program was implemented using 8 modules of "Making Proud Choices" curriculum. Pre and post surveys were administered to participants at the first and last implementation sessions, respectively. During year 2, 391 pre-surveys and 208 post-surveys were collected.
Results: We will present the results on the changes in teen participants' attitudes on condom use and STD prevention between pre- and post-survey. We will also conduct analysis on the correlates of attitudes and several reported behaviors among the participants.
Conclusion: Implementation of a sexual health program such as PTOP for high-risk youth is effective at strengthening positive attitudes towards condom use and understanding about STDs, including HIV. Interventions such as PTOP can help reduce teen birth rates and STD cases in areas such as Philadelphia.

Acknowledgement: This project was supported by the Philadelphia Teen Outreach Project (PTOP) funded by the Department of Health and Human Services (DHHS), Award Number TP1AH000219 (PI: Ma), and partially supported by TUFCCC/HC Regional Comprehensive Cancer Health Disparity Partnership, Award Number U54 CA221704(5) from the National Cancer Institute of National Institutes of Health (NCI/NIH). Its contents are solely the responsibility of the authors and do not necessarily represent the official views of the NCI/NIH or DHHS.

Examining MDMX / MDM2 Signaling Pathways in Breast Cancers Expressing Mutant p53

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Mouse Double Minute 2 (MDM2) and Mouse Double Minute 4/X (MDM4 also called MDMX) are negative regulators of tumor suppressor protein p53. MDM2 is amplified in approximately ~10% of human cancers, and MDMX is amplified in 10-12% of breast cancers. MDMX forms heterodimer complexes with MDM2 to enhance MDM2 E3 ligase activity for wtp53. Importantly MDM2 and MDMX are often found over-expressed in the context mutant p53. However, the MDMX/MDM2 functions that are independent of targeting wtp53 for degradation remain unclear. There is a need to further study MDM2/MDMX wtp53-independent tumor promoting activities. Using xenograft mouse models, we demonstrated that the MDMX/ MDM2 heterodimeric relationship in the presence of GOF mtp53 R280K plays a role in driving TNBC increased circulating tumor cell (CTC) formation and early metastasis. This pointed to MDM2/MDMX metastasis promotion working through a p53-independent pathway. We identified that MDMX knockdown in primary tumors correlates with a downregulation in CXCR4 and PTGS2 transcripts. Studies show that silencing CXCR4 in BC mouse models reduces metastatic burden, TNBC cell lines, MDA-MB-231-MLP-vector control, MDA-MB-231-MLP-shmdm2, and MDA-MB-231-MLP-shmdmx, were orthotopically injected into xenograft mouse models. TNBC-derived CTC cell lines were established by growing CTCs in culture. We performed an in vitro migration assay to model the metastatic phenotype ex vivo using the MDA-MB-231-MLP and MDA-MB-231-MLP-CTC cells and their knockdown lines. We observed delayed migration with a reduction in MDMX or MDM2 in MDA-MB-231-MLP-CTC lines compared to 231-MLP cells. This suggests that MDMX or MDM2 induces cell migration in vitro in non-CTC (231-MLP) and CTC lines. Through RT-qPCR and immunoblot analysis, we observed a significant reduction in CXCR4 levels, which suggests that the MDMX-mediated early metastasis pathways occur prior to CTC formation.

To identify ubiquitin targets of the MDMX/MDM2 heterodimer complex, we enriched for ubiquitinated proteins through affinity purification. We will report on our preliminary data in this protein-target identification area, with a focus on CXCR4 and histones. Our objective is to identify novel MDMX or MDM2/MDMX heterodimer protein interactions that assist MDMX or MDM2 in driving TNBC metastasis independent of p53.
(n=338)

Abstract #22

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Building A Library of Patient Stories To Connect Communities To Lung Cancer Care And Screening

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Background:

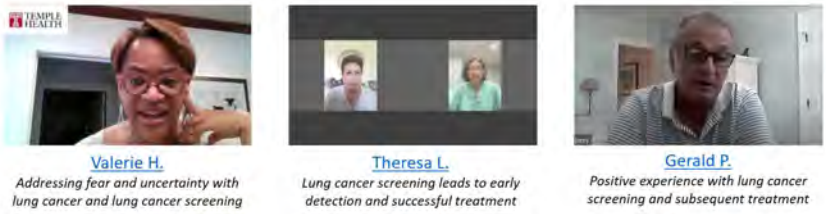
Fear, perceived stigma, mistrust of the healthcare system and lack of awareness of screening are patient-level barriers to screening. Underserved and Black Americans are disproportionally affected by lung cancer and are also less likely to attend lung cancer screening. The purpose of our work is to combat these barriers to screening through providing education and motivation for patients to go to lung cancer screening.

Methods:

Patients were asked to share their lung cancer screening experience for a patient education video. Consenting patients were invited to join a zoom and were recorded answering interview-style questions about their screening experience. Videos were edited and saved in a library of patient story videos.

Results:

We built a library of patient stories to connect communities to lung cancer care and screening. Videos have been used to educate about lung cancer and lung cancer screening, decrease fear and uncertainty surround lung cancer and lung cancer screening, and direct patients to get screened. Videos have been used for patient engagement and community outreach on websites, at patient engagement events, for commercials, by nonprofits, and in a patient education app.



Conclusion:

Patient testimonial videos can build trust in and decrease fear of lung cancer screening, providing motivation for patients to get screened.

Abstract #23

Intracellular interaction of downstream molecular mediators of miR-1207-3p in prostate cancer cells

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Prostate cancer (PCa) is the second most common cancer diagnosed among men in the United States. Human chromosome 8q24 is the most important PCa susceptibility locus. This region contains the MYC oncogene, which is involved in early PCa initiation. Downstream to MYC is the PVT1 gene, which is often amplified in PCa. PVT1 is a non-protein coding gene that encodes six annotated microRNAs (miRNAs), including miR-1207-3p, which our laboratory has previously demonstrated to be a significant modulator in PCa. We revealed that miR-1207-3p is significantly underexpressed in PCa cells and histologically confirmed PCa tissues, when compared to normal prostatic cells and tissues and that miR-1207-3p has tumor suppressive activity in vivo. Moreover, we discovered that miR-1207-3p directly targets fibronectin type III domain containing 1 (FNDC1), which was found to be overexpressed in PCa cells and concomitant overexpression of (FN1)/androgen receptor (AR)/c-MYC. In an effort to better understand the role of FNDC1/FN1/AR/c-MYC in PCa progression we examined the interaction between FNDC1/FN1/AR/c-MYC through coimmunoprecipitation and found direct physical interaction in tumorigenic cell lines as compared to the non-tumorigenic cell line. We also examined the spatial localization of the FNDC1/FN1/AR/c-MYC pathway by performing immunofluorescence staining in PCa cells. Single staining analysis revealed that FNDC1 and c-MYC localize to the nucleus and cytoplasm while AR localizes to the nucleus and FN1 localizes to the cytoplasm in PCa cells. However, in normal prostate epithelial cells FNDC1, AR and c-MYC localize to the nucleus whereas FN1 localizes to the cytoplasm. These results suggest that miR1207-3p may be affecting the interaction between FNDC1/FN1/AR/c-MYC. However, our findings show that miR-1207-3p has no effect on this interaction. Future studies will assess the role of AR in this interaction. Thus, understanding this molecular interaction can reveal additional insight into the role of AR in PCa progression.

A Community-Based screening intervention to Improve Colorectal Cancer Education for underserved African American, Asian American, and Hispanic American Communities in New York City

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Background: In the United States, colorectal cancer is expected to contribute to approximately 52,580 deaths in 2022. It is known as the third leading cause of cancer-related deaths amongst women and men, and the second most common cause of cancer-related deaths for men and women combined. It is estimated that African Americans are 40% more likely to die from colorectal cancer than most other racial groups. Amongst Asian Americans, colorectal cancer is the second most common cause of cancer in the United States. Hispanic have significant rates of mortality and are disproportionately affected by colorectal cancer. In New York City, it is estimated that 1,100 adults will die from colon cancer and over 3,500 New Yorkers are newly diagnosed.

Methods: Our project focused on collaborating and working closely with community-based organizations (CBOs) in New York City by using a community-based participatory research method (CBPR). The CBOs were involved in material development, staff training, and community co-delivery of education to increase awareness of colorectal cancer prevention and screening in New York City. A total of 291 African American, Hispanic American, and Asian American community members were recruited from collaborating CBOs who participated in the educational workshops. Demographic information and knowledge on colorectal cancer were collected before and after the education to assess the effectiveness of the educational workshops. Chi-square and t-test were used to conduct data analysis.

Results: The mean age of study participants was 72 and 75% was female. African Americans, Asian and Hispanic Americans represented 39%, 29%, and 25% of the sample, respectively. Our findings show that 68% of participants have had CRC screening before and the most common screening method was colonoscopy (73%.) About 27% of participants had colon polyps detected. In addition, participants were more knowledgeable about the correct age to start CRC Screening in the post-intervention survey (74.6%) than in the pre-intervention survey (34%). After the workshops, based on 16 multiple choice and true/false questions, participants demonstrated to be more knowledgeable about CRC risk factors and screening behavior (9.6 vs. 11.1 points).

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Conclusions: Our findings demonstrate that community-engaged education interventions are effective in raising awareness and increasing knowledge of colorectal cancer prevention in underserved African American, Asian American, and Hispanic American Communities Using a CBPR approach that incorporates input from CBOs is important in developing and delivering CRC education successfully. Currently, we are planning 6-month follow up interventions with participants to assess if they had lifestyle behavior changes and to determine if participants did a colorectal cancer screening after the workshop.

Abstract #25

A Review of Existing Literature on Non-Alcoholic Fatty Liver Disease (NAFLD) among Asian Americans

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Background: Non-alcoholic fatty liver disease (NAFLD) affects about 25% of the general population in the US. Although epidemiology of NAFLD is well studied in the US, the scientific understanding of how this disease affect Asian Americans is very limited. Given that NAFLD is strongly increases the risks of liver cancer, a condition that disproportionally affect Asian Americans, it is critical that we better understand the burden of NAFLD in this population.

Methods: In this study we conducted a review of previous studies on NAFLD in Asian Americans and in Asia/Pacific regions. We focused on three aspects: (1) the prevalence and incidence rates of NALFD in the target populations; (2) the risk factors and protective factors of NAFLD; (3) the knowledge/awareness of and attitude towards NAFLD and its prevention, diagnosis, and treatment. We also identified limitations in previous studies with regards to study design and methodology.

Findings and Conclusion: We have found that so far there has been there is paucity of data for NAFLD burdens in Asian Americans. The few existing studies estimated the prevalence of NAFLD to be between 18% to 30% in Asian Americans, with higher rates in men than women. Despite inconsistent findings on how prevalence rates compare between Asian Americans and non-Hispanic white, increased evidence points to an elevated risks for advanced NAFLD and worsen outcomes in Asian Americans. We will discuss the implications for the prevention in Asian Americans, as well as linkage to care for early diagnosis and treatment for Asian Americans with NAFLD.

Abstract #26

Common mRNA targets of Estrogen Receptor Alpha (ERα) and PARN in Breast Cancer Cells

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Breast cancer (BC) is one of the most common women cancers in the United States, approximately 1 in 8 women will develop invasive BC in their lifetime. The estrogen signaling pathway regulates the female reproductive system and is primarily mediated by estrogen receptors (ER). ERα is primarily expressed in the mammary glands and its deregulation is involved in promoting BC. While it is well studied how ERα regulates gene expression transcriptionally, the role of ERα in posttranscriptional gene regulation has not been explored. Our preliminary studies indicate that ERα acts as an activator of PARN-mediated deadenylation, through the PARN/p53 interplay in the nucleus but independent of estrogen treatment, affecting gene expression in a transactivation-independent mechanism in BC cells. Inhibiting ERα function and nuclear localization by fulvestrant treatment decreases nuclear deadenylation. As deadenylation is the process in which the poly(A) tails of target mRNAs are shortened signaling mRNA degradation, we tested the functional overlapping of ERα and PARN analyzing by qRT-PCR the upregulation of mRNA targets in samples from different BC cells depleted of ERα or PARN expression. ERa and PARN have common mRNA targets, specifically ID1, KLHL24, and LUM genes, which are involved in cell invasion, metastasis, and angiogenesis in BCs, thus providing a functional connection between mRNA 3' end processing and BC progression. The effect of this regulatory mechanism on cellular transcriptomes might provide

Abstract #27

African ancestry-informative markers and the identification of population-specific disease loci in cancer

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Cancer health disparities remain a significant problem affecting minority populations, especially those of African ancestry. As recently as 2014, African-Americans in the US were found to have the highest rate the cancer-specific mortality rate for malignancies such as colorectal, prostate, lung, and breast[1]. The literature suggests that some of the disparity remains after adjusting for socioeconomic factors. Recent advances in cancer genomics have allowed researchers to generate, share, and access large amount of data creating novel opportunities for significant progress in cancer research. Using data from the 1000 Genome project, we have identified approximately 46,738 African ancestry-informative markers (AIMs) that differentiate African populations from European populations. In this work, we analyze the AIMs for functional and disease annotations to identify cancer-associated AIMs and elucidate their contribution to disease development. Of the 46,738 AIMs, less than 1% were found in coding regions of the genome and 48.54% of them are non-synonymous mutations. Of the non-synonymous SNPs such as rs9830253, rs7645635, rs12186491, rs16891982, rs10238965, rs6601495 have been predicted to be damaging.

Certain chromosomal regions appear to be enriched for AIMs and these regions were also evaluated for disease association using cancer-associated loci identified from the literature. These AIMs were also investigated for their ability to detect population substructure within populations of high degree of African ancestry which would allow us to potentially identify population-specific disease loci. Principal component analysis shows that these AIMs effectively detect substructure within populations of African ancestry and we identified region of high Fst difference between these subpopulations. Overall, Population-specific markers can allow us to better understands population cancer health disparities.

Abstract #28

Understanding how Stanniocalcin-1 (STC1) affects Apolipoprotein-1 (APOL1) mediated cellular cytotoxicity

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Apolipoprotein L-1 (APOL1) is an innate immunity ion-channel forming protein that provides protection against African trypanosomes. Due to a molecular arms race, people of African lineage evolved with two APOL1 variants, namely G1 and G2. These variants, also called renal risk variants, confer a higher risk of chronic kidney diseases in homozygous individuals through increased intracellular *APOL1* expression in podocytes and endothelial cells. APOL1 renal risk variants are also associated with some cancers. This is apparent in the African American demographic, which already faces disproportionate health burdens. Stanniocalcin-1 (STC1) is a glycoprotein hormone, which has been shown to be significantly upregulated in kidney biopsies of patients with G1 and G2 APOL1 compared to G0 genotype APOL1. To understand the function of STC1 in APOL1 renal risk variant cytotoxicity, we overexpressed *STC1* in HEK-293 derived cells with inducible *APOL1* and measured Lactate dehydrogenase (LDH) in the culture medium. Cells overexpressing STC1 with no APOL1 induction were used as control. We found significantly lower LDH release in cells expressing STC1 and APOL1 compared to the control group at 24, but not 42 hours post APOL1 induction.

Our results suggest that STC1 has a protective effect on G2 mediated cytotoxicity. In the future, we plan to further investigate how STC1 reduces APOL1 mediated cellular cytotoxicity.

Abstract #29

Ethnic enclaves and colon cancer stage at diagnosis among New Jersey Hispanics

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Ethnic enclaves are culturally distinct neighborhoods with high concentrations of individuals of the same ethnic origin. Study findings are inconclusive on whether living in ethnic enclaves serves as a risk or protective factor of cancer-related outcomes. This study aims to examine associations between colon cancer stage at diagnosis and living in an ethnic enclave. The study population includes adult Hispanic New Jersey residents with their first, histologically confirmed invasive colon cancer diagnosed between 2006- 2014 (N = 1298). Cases were linked to residential histories and limited to cases with at least 10 years of residential history (N=1072). Sixty-five percent of the cases were late stage (regional + distant) and 26 percent were distant stage. About 63.5 percent of the late-stage cases lived in an enclave at the time of diagnosis, 22.4 percent of the late stage cases lived in an enclave for the 10-year period and 30.5 percent never lived in an enclave. We did not find significant associations between late-stage diagnosis and living in an enclave. However, when we examined the distant stage, we found residence in an enclave for the entire 10-year period resulted in lower odds of distant-stage compared to never living in an enclave (Odds Ratio 0.6 95%CI 0.42-0.85). Among Hispanics in New Jersey, enclave residence was not associated with late stage but was associated with distant stage. Additional research is needed to determine whether the length of residency is protective.

Abstract #30

The RNA binding protein HuR mutation K313R suppresses ubiquitination by BRCA1/BARD1 and increases HuR accumulation

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BRCA1 mutations strongly predispose individuals to breast cancer (BC) and make them more susceptible to developing Triple Negative Breast Cancer (TNBC), a form of BC for which there is a poor prognosis. Identification of new pathways is necessary to improve BC patient outcomes. BRCA1 has roles in DNA repair as well as multiple other cellular processes. Additionally, BRCA1 together with BARD1 acts as an E3 ubiquitin ligase. We have previously shown that BRCA1/BARD1 is able to ubiquitinate the RNA binding protein HuR under non-stress conditions. HuR is a major RNA binding protein which binds to and stabilizes its target transcripts. The mRNA targets of HuR are involved in processes such as carcinogenesis, cell proliferation, and apoptosis. High levels of cytoplasmic HuR in BC correlate with poorer patient outcomes, making HuR a potential target for cancer therapy. Our previous in vitro studies showed that BRCA1/BARD1 ubiquitinates HuR primarily at K313 in the RNA Recognition Motif 3 (RRM3) of HuR. Our preliminary studies show that cells transfected with a K313R HuR mutant have increased levels of ubiquitinated HuR. We hypothesize that this increase may be due to a decrease in degradation of the K313R mutant, which is resistant to ubiquitination by BRCA1/BARD1. Importantly, HuR ubiquitination by BRCA1/BARD1 causes HuR to detach from its mRNA targets, destabilizing these transcripts.These results suggest that BRCA1 mutation in BC patients may be inhibiting normal HuR protein turnover, causing an increase in HuR accumulation and abnormal processing of mRNA transcripts involved in carcinogenesis and tumor suppression.

Abstract #31

Regulation of p21 expression in the DNA damage response by calreticulin, CUGBP1, and a long non-coding RNA generated by alternative polyadenylation

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The cyclin-dependent kinase inhibitor p21 functions in cell-cycle regulation, DNA damage response (DDR), and is at the center of "Therapy-induced senescence" (TIS). While high or low levels of p21 upon chemotherapeutic treatment leads to senescence, moderate levels enable a proliferative fate. Therefore, understanding mechanisms involved in regulating p21 dynamics in TIS is relevant in improving the efficacy of treatments, especially in triple negative breast cancer (TNBC) which undergoes frequent chemoresistance relapse by TIS. There is a current gap in knowledge concerning the early mechanisms regulating expression of p21; a delay in p21 expression following cellular stress has been described despite p53 presence at the CDKN1A promoter, the gene that encodes p21. CDKN1A undergoes alternative polyadenylation (APA), a mechanism that generates alternative transcripts from the same gene. APA in CDKN1A occurs in the first intron after DNA damage generating a long non-coding RNA named APA-CDKN1A. APA-CDKN1A is expressed early in DDR before the induction of p21 expression. Interestingly, APA-CDKN1A depletion does not affect full-length CDKN1A mRNA levels, increases cell proliferation, and significantly decreases p21 protein levels, suggesting a translational regulatory role of APA-CDKN1A. Furthermore, the RNA-binding proteins (RNA-BP) and translational regulators of p21, calreticulin and CUGBP1, compete for binding to the same sequence in APA-CDKN1A and CDKN1A mRNA. My results indicate that changes in CDKN1A isoforms interaction to these RNA-BPs during DDR and in different BC will affect p21 expression levels and cellular functions, and this can be exploited for conditions where p21 levels are relevant, such as in TIS.

Abstract #32

Depression Significantly Predicted Poor Medication Adherence Among Asian Americans Living with Chronic Hepatitis B: A Longitudinal Study

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Introduction: Asian Americans is a Hepatitis B (HBV) disparity population who only account for 6% of the US population but experience a 60% burden of having HBV, which is associated with 75% of hepatocellular carcinoma (HCC). Adherence to HBV medication is a practical approach to prevent liver cancer. However, limited studies have been conducted on the impacts of depression on HBV medication adherence among underserved Asian American HBV patients. **Methods:** This study utilized 12-m follow up data from a randomized controlled clinical trial aimed at improving long-term adherence to HBV medication adherence. Eligible Asian American HBV patients were recruited from the Greater Philadelphia Area and New York City. HBV medication adherence was assessed using the Morisky 8-Item Medication Adherence Scale (MMAS-8), and depression was measured with Patient Health Questionnaire-9 (PHQ-9).
Results: Among 154 participants (118 Chinese and 36 Vietnamese), 43.57% were female, and 56.49% were male. Nearly all the participants reported having health insurance (92.21%) and having a physician to visit regularly (95.21%). Bivariate analysis showed that depression was negatively significantly associated with medication adherence score (r=-0.55, p<0.001). Multivariable analysis revealed that medication adherence score was associated with being in intervention group (Coef. 0.58, 95% CI: 0.10-1.05), Vietnamese ethnicity (Coef. 0.55, 95% CI: -0.02-1.13), and depression severity score (Coef.-0.16, 95% CI: 4.02-6.89), respectively, controlling for demographic covariates.
Conclusion: The findings suggest that depression level has significant impacts on medication taking adherence, implying that targeted interventions addressing psychosocial barriers would be effective in promoting HBV medication adherence among underserved Asian Americans.
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Abstract #33

Single-Encounter Telemedicine (SET) Lung Cancer Screening Reveals Lower Rates Of Screening But Preserves Access to Minorities

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Background: COVID-19 forced a delay of non-essential health services, including lung cancer screening. Our institution developed a single-encounter, telemedicine (SET) lung cancer screening whereby patients receive low-dose CT in-person, but counseling regarding results, coordination of follow-up care and smoking cessation is delivered using telemedicine. This study compares outcomes of SET lung cancer screening to our pre-COVID, single-visit, in-person (SIP) lung cancer screening.

Methods: We recorded gender, race/ethnicity, age, education attainment, and smoking status and dependent variables cancer diagnosis, stage, and treatment between March 2019 -July 2021. We compared outcomes of SIP lung cancer screening before COVID-19 and SET lung cancer screening amid COVID-19.

Results: There was a significant difference in number of patients screened pre- and amid COVID-19. 673 people were screened via SIP, while only 440 were screened via SET. SIP screening consisted of 52.5% African-American patients, which decreased to 37% with SET screening. There was no significant difference in gender, age, or educational attainment. There was also no significant difference in Lung–RADS score between the two methods of screening or diagnostic procedures performed. Ultimately telemedicine based screening diagnosed fewer cancers, 1.6% diagnosed via telemedicine vs 3.3% screened by in person.

Conclusion: We implemented SET lung cancer screening to continue lung cancer screening during a global pandemic. Our study established feasibility of telemedicine-based lung cancer screening among our predominantly African American/Black population, though fewer patients were screened. We found no difference in distribution between age, or educational attainment suggesting other factors discouraging lung cancer screening amid COVID-19.

Abstract #34

Investigating the cellular and molecular factors contributing to health disparities in esophageal cancer

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Esophageal squamous cell carcinoma (ESCC) is more common in African American (AA) males while esophageal adenocarcinoma (EAC) most often occurs in Caucasian American (CA) men. Presently, our understanding of the molecular mechanisms supporting these disparities remains limited. A recent study combined RNA-Seq tissues from AAs and CAs with single cell (sc) RNA-Seq in normal esophageal mucosa to demonstrate that genes upregulated in AAs mapped primarily to two specific cell lineages, supporting the possibility of race-based differences in the cellular heterogeneity of esophageal mucosa. In the current proposal we will directly perform scRNA-Seq on esophageal biopsy specimens from AA and CA subjects with normal esophageal mucosa to explore the innovative ***hypothesis*** *that differential cellular heterogeneity in the esophageal mucosa of African American and Caucasians subjects contributes to the established race-based disparities in the incidence of esophageal cancer subtypes*. To do so, we will compare the cellular and molecular heterogeneity of normal esophageal mucosa in AA and CA individuals using scRNA-Seq. Comprehensive bioinformatics analyses will be performed on scRNA-Seq data to define cell cluster identities, cell fate trajectories, and molecular features in cell types (epithelial, immune, and endothelial cells, fibroblasts) within esophageal mucosa in relation to self-reported race. Identification of cell types and pathways displaying differential representation or expression in AA and CA subjects have great potential to inform our understanding of the biological underpinnings of established racial disparities in esophageal cancer incidence. Additionally, such findings may be used to develop novel strategies for improving diagnosis, monitoring and therapy in esophageal cancer.

Abstract #35

FAM122A is a substrate-competitive inhibitor of the B55α/PP2A phosphatase required for timely progression through the cell cycle interphase

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The heterotrimeric Ser/Thr protein phosphatase 2A (PP2A) is responsible for the dephosphorylation of many regulated phosphoproteins. B regulatory subunits mediate substrate recognition where substrates contain Short Linear Motifs (SLiMs) that mediate docking to holoenzyme subunits. Substrates of B55α/PP2A, the most abundant PP2A holoenzyme, are diverse, and the first consensus SLiM, [RK]-V-x-x-[VI]-R, has only recently been identified (Fowle et al. eLife 2021;10:e63181). Here we report the identification of this SLiM in FAM122A, an inhibitor of B55α/PP2A. This SLiM is necessary for FAM122A binding to B55α in vitro and in cells and the inhibition of the holoenzyme. Computational modeling predicts an interaction consistent with mutational and biophysical data supporting a mechanism whereby FAM122A uses the SLiM to dock to B55α and spatially constrains substrate access by occluding the catalytic subunit. Consistently, FAM122A functions as a competitive inhibitor as it prevents binding of substrates in in vitro competition assays and the dephosphorylation of CDK substrates by B55α/PP2A in cell lysates. Time-lapse and immunofluorescent microscopy show that FAM122A is nuclear in interphase suggesting a role in controlling B55α/PP2A nuclear function. Consistently, knockout of FAM122A in HEK293 and T98G cells results in cell cycle interphase defects. FAM122A-KO cells proliferate slowly, exhibiting an elongated S-phase characterized by severely reduced DNA synthesis. Quiescent FAM122A-KO cells also show delayed pRB phosphorylation kinetics and cyclin expression upon cell cycle re-entry. Overall, these data strongly suggest that FAM122A is a SLiM-dependent, substrate-competitive inhibitor of B55α/PP2A that suppresses its activity to ensure timely progression through the cell cycle interphase.

Abstract #36

Measuring prevalence of psychosocial-related factors in predicting depressive symptoms of cancer patients and their primary caregivers

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Purpose/hypothesis: This project will focus on measuring the relationship between social support and depressive symptoms among cancer patients and their caregivers receiving care at Penn Medicine: Abramson Cancer Center (PMACC). Additionally, an evaluation of the social support resources provided by PMACC will be conducted to examine the social support and mental health needs and expectations of their oncology population.

Number of participants: 23 dyads (cancer patients and caregivers) receiving oncology care through PMACC engaged in the survey. Seven individuals engaged in the optional semi-structured interviews.

Methods/materials: Cancer patients and caregivers will be administered a 40-item survey that measures basic demographics, social support (Multidimensional Scale of Perceived Social Support), and depression (Center for Epidemiologic Studies Depression Scale). The semi-structured interviews contained themes to measure awareness of the social work department and the impact the social support and mental health services had on the patients and caregivers.

Results: In measuring the relationship between total perceived social support score and the total depression score, the two variables were negatively correlated ($r(444)$, -.343, $p=.02$). While there was a lack of initial awareness between the oncology community and the social work department, once they became aware of the department and services, positive experiences and met needs were expressed.

Conclusions: Oncology patients and primary caregivers at PMACC documented a negative association between depressive symptoms and perceived social support. While there was an initial lack of awareness of the social work department, once the population became aware, positive experiences and met needs were expressed.

Abstract #37

The Relationship Between Medical Mistrust and Compliancy with Follow-Up Care in Black and Latinx Breast Cancer Survivors

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Introduction: There are over 633,000 young adult (YA) cancer survivors in the US, a number that is expected to grow exponentially over the next few years. Follow-up care is critical to a successful transition post-treatment as it regulates tests and treatments that survivors will have to undergo to maintain their health. Medical mistrust may be one reason for follow-up non-adherence. By understanding the relationship, researchers and clinicians alike can work to reduce racial disparities in cancer survival rates.

Methods: This sub-analysis stems from a larger study wherein twenty-six female breast cancer survivors, aged 26-41, participated in an online survey assessing family-building practices. Participants included ten Latina women, nine Black women, and seven multiracial women, who all completed the Medical Mistrust Index. Correlation tests were conducted to assess the relationship between medical mistrust and follow-up care.

Results: Most patients reported seeking general follow-up care annually (46.2%) and cancer-specific care when needed (42.3%). Researchers found a significant positive correlation between medical mistrust and general follow-up care ($r(23) = 0.480$, $p = 0.015$), indicating that higher levels of medical mistrust were associated with increased compliance with general follow-up care.

Conclusion: Participants with greater medical mistrust reported greater compliance with general follow-up care. This finding contradicts previous research that has indicated that greater mistrust would result in greater non-compliance. Future studies should explore the source of medical mistrust, any confounding variables, and the race/ethnicity of providers to better understand the impact on survivors of different backgrounds.

Abstract #38

STAT2 Signaling Reprograms Lipid Metabolism in Colorectal Cancer

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Our study aims to elucidate molecular mechanisms by which the transcription factor STAT2 promotes colorectal carcinogenesis. Colorectal cancer (CRC) is the third deadliest cancer affecting both men and women. Health disparities in CRC incidence are attributed to a myriad of factors that range from genetics to unhealthy lifestyle and poor access to medical care in communities of low socio-economic status. Therefore, increased knowledge on the signaling networks involved in disease initiation and progression are imperative to combat CRC. We have collected data employing different animal models of CRC that reproducibly show STAT2 contributes to tumor growth. Transcriptomic profiling of colon tumor xenografts revealed a subset of genes involved in cholesterol metabolism to be downregulated in the absence of STAT2. We identified LIPG as the top hit gene that is involved in lipid uptake and recently shown to promote the growth and metastasis of breast cancer. Lipidomic analysis of tumor xenografts showed that STAT2 reprogrammed the composition of tumor lipids. To determine a relationship between STAT2 and LIPG, we found that treatment with pharmacological inhibitors of LIPG decreased viability of tumor cells expressing STAT2 with mild effect in STAT2 deficient cells. We also found that a deficiency in STAT2 impaired lipid uptake as measured by the accumulation of lipid droplets following incubation with phosphatidylcholine-oleic acid, a LIPG substrate. Altogether, our preliminary findings indicate a novel uncharacterized function of STAT2 that promotes CRC by rewiring lipid metabolism.

Abstract #39

Investigating the clinical relevance in prostate cancer of the serum biomarkers PVT1 exons 4A, 4B and 9 across risk levels and ethnicity/race

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Chromosome 8q24 is of particular importance for cancer susceptibility. Located in this region is the Plasmacytoma Variant Translocation 1 (PVT1) gene, a long noncoding RNA that has been implicated in multiple cancers including prostate cancer. Amplification of the PVT1 gene locus is a common event in many malignant diseases and is associated with poor clinical outcomes. The pioneering role of PVT1, and its alternatively spliced transcripts, as a cancer biomarker is progressively becoming established. We have demonstrated that copy numbers of PVT1 exons 4A, 4B and 9 is quantifiable in cancer cells, tissue, and serum from cancer patients. In this study, we assessed clinically annotated serum samples from 40 prostate cancer patients to investigate the clinical relevance of PVT1 exons 4A, 4B, and 9 as a biomarker across cancer risk levels and ethnicity/race. Explorative data analysis for the development of composite score for prostate cancer was performed using Kruskal-Wallis Rank Sum Test. We observed significantly higher copy numbers of PVT1 exons 4B and 9 across all races (White, Black and Hispanic) and Blacks and Hispanics when compared to the control. Additionally, using a 3-level cancer risk rating assessment in which 0 = healthy, 1 = low risk and 2 = high risk, we observed that PVT1 exon 9 may distinguish between cancerous and noncancerous cases across all races, but may not help distinguish between indolent and aggressive cancer cases. Notably, PVT1 exon 4B may help distinguish between indolent and aggressive cancer cases for Blacks and Hispanics. The results of this study suggest that using PVT1 exon 4B or 9 may identify cancer regardless of ethnicity/race, and that utilization of serum PVT1 exon 4B copy number may help distinguish between indolent and aggressive prostate cancer in Blacks and Hispanics.

Abstract #40

FGraph convolutional neural networks applications to cancer cellular network analysis

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Graph neural networks (GNNs) and graph convolutional neural networks (GCNNs) have the potential to solve and improve solutions to problems in natural language processing, computational chemistry, the computational biology of cancer research, and other fields. A graph is a mathematical object consisting of nodes connected by edges. This type of structure can be used to model, among other things, networks of cells—including protein-protein interactions, gene-protein interactions, and epigenetic interactions. Once nodes are represented in an embedding space, GNNs can begin to make predictions about graphs. Graph-level tasks may be performed; these include graph classification and various types of graph comparison. GCNs and GCNNs may also be used to predict the position of a node in a graph, the label of a node, relationships between nodes, and whole graph labels. GCNs employ key characteristics of ordinary convolutional neural networks such as the creation of feature maps and graph pooling, as well as other graph-specific characteristics. This work draws on data from The Cancer Genome Atlas to expand the scope of what machine learning and GNNs can address, specifically graphs pertaining to the computational biology of cancer and gene networks.

Abstract #41

Lung Cancer Screening in the COVID-19 Era: Understanding Program-Level Impact

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Introduction: Lung Cancer Screening (LCS) reduces lung cancer mortality, yet utilization remains low. COVID-19 impact on LCS program components and uptake is unknown. Understanding program-level barriers in the context of COVID-19 will help guide resource allocation and inform optimization of LCS in the future.

Methods: The GO2 Foundation for Lung Cancer conducts an annual, retrospective survey of United States (US) LCS programs. We partnered with the GO2 Foundation to add additional questions related to delivering LCS in the context of COVID-19. We conducted descriptive statistical analysis of 2021 survey results to understand program demographics and LCS program components most affected by COVID-19.

Results: Ninety-nine programs completed the survey. Nationally, the Southern, Northern, Midwestern, and Western regions represented33%, 28%, 25%, and 13% of respondents, respectively. Community programs represented 67% of respondents while academic centers represented 10%. Programs screened a median of 868 patients (Range 0 – 7,930; SD 1267) in 2020. Program components most significantly compromised by the COVID-19 pandemic were patient recruitment (85%), in-person consultation (79%), patient education (71%), access to radiology services (67%), and smoking cessation (60%). Sixty-two percent of respondents reported improved use of telemedicine.

Conclusion: Our findings suggest some of the most critical LCS components were most vulnerable to compromise. These findings underscore the importance of telemedicine in the delivery of LCS within the context of COVID-19. The importance of this survey effort cannot be overstated as it establishes understanding of real-world LCS challenges and helps guide targeted solutions to optimize the future of LCS.

Abstract #42

Developing and Utilizing a Smart Phone Based App to Educate and Engage At Risk Patient Population in Lung Cancer Screening

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In the United States, there are immense disparities in Lung Cancer outcomes among different ethnic and racial groups. These disparities are primarily due to differences in social determinants of health and patient education between groups. One of the biggest factors that have the potential to ameliorate these disparities and decrease deaths from Lung Cancer is Lung Cancer Screening (LCS) by using Low Dose Computed Tomography (LDCT). Adherence to LCS among underserved populations with risk factors and/or have been diagnosed with Lung Cancer is low. **Given the importance of LCS, our intervention is focused on increasing accessibility, adherence, and education to this process.**

The intervention in this study is to utilize Opti-Health, a customizable phone application which has the potential to increase patient adherence to their LCS appointments and educate patients on Lung Cancer and LCS. Knowledge gaps that have been found in this field of research include how medical compliance and appointment attendance increases when patients receive reminders via phone and how patient education resources are above the average patient's comprehension level of medical topics. **The impact of this intervention has the potential to be long lasting as patients can trust in their health care providers to give them optimal care through engagement and education by this innovative mode of connection.**

Abstract #43

Cancer prognosis with tissue birefringence measured by label-free quantitative microscopy

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The interactions between light and cells, including scattering and absorption, are the foundation for modern quantitative microscopy techniques to quantify their biochemical, functional, and morphological properties. Cancer diagnosis and prognosis with the inherent structural alterations have recently emerged as one promising approach toward more accurate histopathology. The disorganization and the degradation of the collagen fibers have been shown to be correlated with cancer progression and metastasis. We hypothesize that the differences in the microscopic collagen organization measured by quantitative microscopy (birefringence and angular dispersion of collagen fibers) can be used to distinguish aggressive versus non-aggressive cancer.

Triple-negative breast cancer and prostate cancer tissue specimens obtained from Weill Cornell Medical College were imaged using a novel quantitative Differential Phase Contrast (DPC) microscopy at the Biomedical Photonics Laboratory (BPL). Our custom DPC microscope can measure both quantitative phase (cellular mass distribution) and birefringence (structural anisotropy) label-free. The results of the measurements were analyzed using MATLAB to quantify birefringence strengths and their angular dispersion. Statistical analysis was conducted using Excel.

Our findings show a positive correlation between the birefringence and angular dispersion against the recurrent time (the time elapsed until cancer reappeared) for aggressive (recurrent time <5 years) and non-aggressive (>5 years) cancerous tissue. The results reveal that collagen degradation and disorganization increase with the aggressiveness of cancer and highlight the role of collagen organization in cancer aggressiveness assessment.