CLARK ATLANTA UNIVERSITY
Center for Cancer Research and Therapeutic Development

ON THE CAMPUS OF
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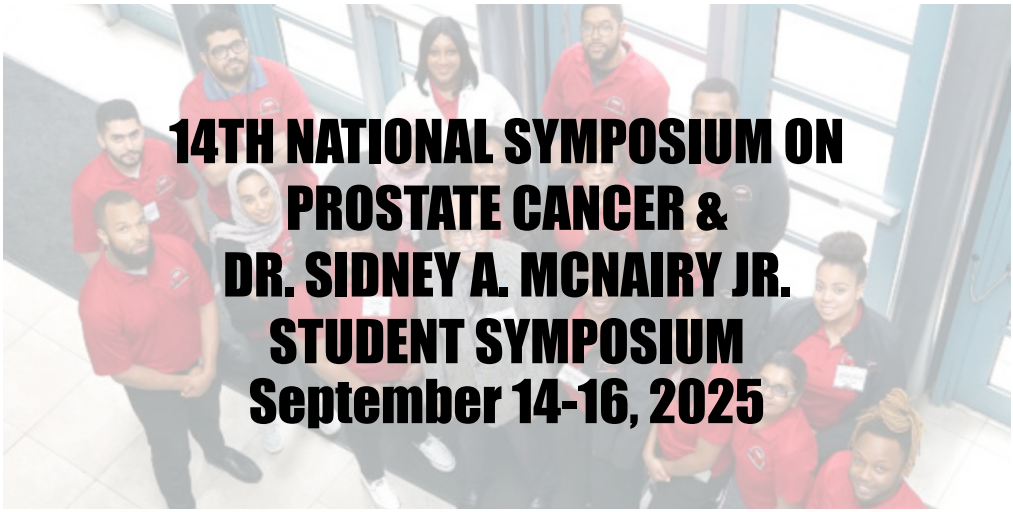
ATLANTA, GA



14TH NATIONAL SYMPOSIUM ON PROSTATE CANCER & DR. SIDNEY A. MCNAIRY JR. STUDENT SYMPOSIUM

September 14-16, 2025

KEYNOTE SPEAKERS:

WILLIAM DOUGLAS FIGG, SR.
&
RICK KITTLES

LWHK CHUNG MEMORIAL LECTURE:

DOUGLAS STRAND

CLARK ATLANTA UNIVERSITY

GEORGE T. FRENCH, JR., PH.D.

PRESIDENT

September 12, 2025

Dear Symposium Guests:

Welcome to the 14th National Symposium of the Center for Cancer Research and Therapeutic Development (CCRTD) at Clark Atlanta University (CAU). At Clark Atlanta University, we believe that research and education must go hand in hand. The research and scholarly work conducted at CAU are vital for providing our students with a top-tier educational experience and delivering valuable insights to the world. This biennial prostate cancer symposium, attended by world-renowned prostate cancer researchers, clinicians, investigators, and industry leaders, demonstrates the thriving academic environment and vibrant innovative community that define CCRTD.

CCRTD continues its mission of serving the African-American community by providing opportunities for high-caliber basic and translational research; training scientists in cancer research; and providing an educational environment for community outreach, prevention, early detection, and treatment of prostate cancer, which disproportionately affects African-American men.

This remarkable symposium unites the diverse talents and passions of investigators and clinicians from around the world dedicated to tackling prostate cancer from research to treatment. We aim to promote discussion among scientists, researchers, physicians, and other stakeholders involved in the fight against prostate cancer and to help address the disparities faced by Black men living with this devastating disease.

The work, training, and education being done at CCRTD and in each of your labs and institutions are critical to moving the needle forward in cancer research, innovation, and care. As you share your work, you plant seeds for a bright future that will reduce health disparity, encourage a more diverse cancer workforce, and lead the way in prostate cancer research.

Sincerely,

George T. French, Jr.

George T. French, Jr.
President

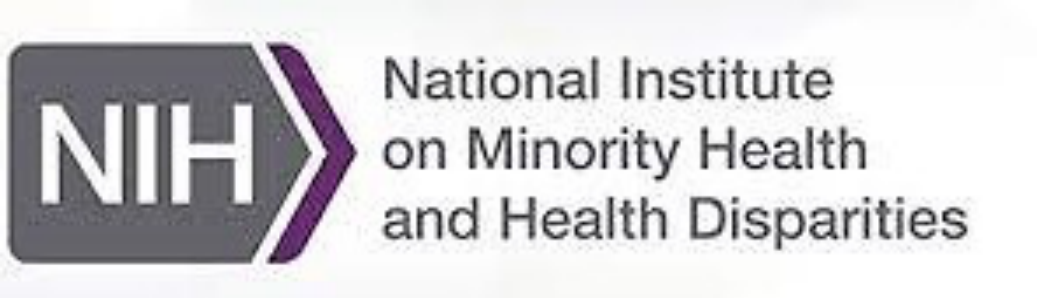
Sidney A. McNairy, Jr., Ph.D., D.Sc., L.H.D

Former Member of the Senior Executive Service
Associate Director, NCRR and
Director, Capacity Building Branch, NIGMS
National Institutes of Health

Dr. Sidney A. McNairy, Jr. is an award-winning academician and senior-level federal grants administrator. He received a B.S. degree in Chemistry/Mathematics from LeMoyne-Owen College; he received both the Master's and Doctorate degrees in biochemistry with minors in both organic chemistry and human physiology from Purdue University. During his graduate studies his research focused on the isolation and chemical/biological characterization of tri-terpenoid glycosides. He has done further studies at Columbia University and the Harvard Kennedy School of Government.

He was a Professor of Chemistry at Southern University in Baton Rouge and Director of the Health Research Center. While there, he was a visiting scientist at Charles Pfizer, Eli Lilly, Standard Oil of California, Centers for Disease Control, and the George C. Marshall Space Flight Space Center. Thereafter, he spent over 25 years at the National Institutes of Health as a member of the Senior Executive Service developing and managing programs that focused on the support for both basic and clinical research. Among his numerous awards are nine honorary doctorate degrees including Clark Atlanta University.

Sponsors and Donors



14th National Symposium on Prostate Cancer
and
Dr. Sidney A. McNairy Jr. Student Symposium

Clark Atlanta University

September 14-16, 2025

Sunday, September 14th, 2025

Opening Reception: 6:00 – 8:00 pm

6:00 – 8:00 pm	***Hotel: Hyatt Regency Atlanta Downtown*** ***Hotel Address: 265 Peachtree Street NE, Atlanta, GA. 30303***

Monday, September 15th, 2025
Thomas W. Cole, Jr. Center for Science and Technology, Aldridge Auditorium

7:45 – 8:00 am	***Gather and depart from Hotel Lobby for transport to CAU***
8:00 – 8:30 am	***Breakfast*** ***Cole Boardroom***

Opening Ceremony & Keynote Address: 8:30 – 10:00 am

Chair: Jaideep Chaudhary, PhD
Co-Chair: Shafiq A. Khan, PhD

8:30 – 8:40 am	***Jaideep Chaudhary, PhD*** *Director, Center for Cancer Research and Therapeutic Development*
8:40 – 8:45 am	***Shafiq A. Khan, PhD*** *Professor and Georgia Research Alliance Eminent Scholar* *Center for Cancer Research and Therapeutic Developments*
8:45 – 8:55 am	***Charlene D. Gilbert, PhD*** *Provost*
8:55 – 9:00 am	***George T. French, Jr., JD, PhD*** *President*

9:00 – 9:45 am	***Keynote Address*** ***William Douglas Figg Sr., Pharm.D., M.B.A.*** *National Institutes of Health* *Presentation Title: High-Throughput Drug Screening to Identify Novel Synergistic Combinations for mCRPC*
9:45 – 9:50 am	**Q&A**
9:50 – 10:00 am	***Coffee Break*** ***Cole Boardroom***

Monday Morning Session I: 10:00 am – 12:05 pm
Castration Resistant and Neuroendocrine Prostate Cancer

Chair: Bekir Cinar, PhD
Co-Chair: Crystal Byrd

10:00 – 10:20 am	***Adam Sowalsky, PhD*** *National Institutes of Health* *Presentation Title: Intrinsic mediators of persistent AR-positive Prostate Tumors*
10:20 – 10:25 am	***Q&A***
10:25 – 10:45 am	***Amina Zoubeidi, PhD*** *University of British Columbia* *Presentation Title: Lineage Plasticity in Prostate Cancer: From Molecular Insights to Clinical Strategies*
10:45 – 10:50 am	***Q&A***

New and Emerging Prostate Cancer Therapeutics

10:50 – 11:10 am	***Ramesh Narayanan, PhD*** *University of Tennessee Health Science Center* *Presentation Title: Novel Inhibitors of AR, AR-splice Variants, and GR for the Treatment of Refractory Castration-Resistant Prostate Cancer*
11:10 – 11:15 am	***Q&A***
11:15 – 11:35 am	***Daqing Wu, PhD*** *Clark Atlanta University*

Presentation Title: Overcoming Prostate Cancer Chemoresistance through Ferroptosis-Targeted Drug Repurposing

11:35 – 11:40 am ***Q&A***

11:40 – 12:00 pm ***Ravi Madan, MD***
National Institutes of Health
Presentation Title: Charting a New Path for Immunotherapy in Prostate Cancer

12:00 – 12:05 pm ***Q&A***

Lunch: 12:05 – 1:45 pm

12:05 - 1:45 pm ***Lunch***
2nd Floor Exhibition Hall

Thomas W. Cole, Jr. Center for Science and Technology, Aldridge Auditorium
LWHK Chung Memorial Lecture: 1:45 – 2:45 pm

1:45 - 2:00 pm ***Introduction to Dr. Leland Chung***
Shafiq Khan, PhD
Introduction to Dr. Douglas Strand
Nathan Bowen, PhD

2:00 – 2:40 pm ***Douglas Strand, PhD***
University of Texas Southwestern Medical Center
LWHK Chung Memorial Lecture
Presentation Title: A Cellular Anatomy of the Healthy Human Lower Urinary Tract

2:40 – 2:45 pm ***Q&A***

Monday Afternoon Session II: 2:45 – 4:00 pm
Prostate Cancer Metastasis

Chair: Geou-Yarh (Stancy) Liou, PhD
Co-Chair: Nicholas Cook

2:45 – 3:05 pm ***Shafiq A. Khan, PhD***
Clark Atlanta University
Presentation Title: Targeting Gαi2 Protein to Block Migration and Invasion in Prostate Cancer Cells

3:05 – 3:10 pm ***Q&A***

3:10 –3:30 pm ***Jiaoti Huang, PhD***
Duke university
Presentation Title: Discovering Novel Therapeutic Targets for Advanced Prostate Cancer

3:30 – 3:35 pm ***Q&A***

3:35 –3:55 pm ***Bal Lokeshwar, PhD***
Augusta University
Presentation Title: Increased Inflammatory Milieu is a Strong Driver for Cadmium-induced Carcinogenesis in Prostate Epithelial Cells.

3:55 – 4:00 pm ***Q&A***

Monday Afternoon Session III: 4:00 – 5:00 pm
Cancer Genomics

Chair: Nathan Bowen, PhD
Co-Chair: Kofi Khamit-Kush

4:00 – 4:20 pm ***Joseph Lachance, PhD***
Georgia Institute of Technology
Presentation Title: Heterogeneous Genetic Architectures of Prostate Cancer Susceptibility in Sub-Saharan Africa

4:20 – 4:25 pm ***Q&A***

4:25 – 4:45 pm ***Anna Woloszynska, PhD***
Roswell Park Comprehensive Cancer Center
Presentation Title: Identifying Biomarkers and Potential Treatment Targets in Prostate Cancer Health Disparities

4:45 – 4:50 pm ***Q&A***

4:50 – 5:00 pm ***Coffee Break***
Cole Boardroom

Poster Session: 5:00 – 6:15 pm

Chair: Professor Elycia Daniel
Co-Chair: Betelihem Beshir

5:00 – 6:00 pm ***Poster Session***
2nd Floor Hallway

6:00 – 6:15 pm ***Networking with Speakers and Students***
Cole Boardroom (Interested individuals can remain if they would like to connect with the speakers)

6:15 – 7:30 pm ***Dinner***
2nd Floor Exhibition Hall
Prostate Cancer Survivor: *Mr. David Moffett*
Spouse: *Mrs. Marjané Moffett*
Prostate Cancer Survivor: *Mr. Tony Williams*
Spouse: *Mrs. CeCe Williams*

Musician: *Mr. Cameron Barnes*

7:30– 7:45 pm ***Gather and depart from CAU for Transport to Hotel***

<u>Tuesday, September 16th, 2025</u>
Thomas W. Cole, Jr. Center for Science and Technology, Aldridge Auditorium

7:45 – 8:00 am ***Gather and depart from Hotel Lobby for transport to CAU***

8:00 – 8:30 am ***Breakfast***
Cole Boardroom

Tuesday Morning Session IV & Keynote Address: 8:30 – 11:55 am
Biological and Social Determinants of Health

Chair: Daqing Wu, PhD
Co-Chair: Kezhan Khazaw

8:30 – 9:15 am ***Keynote Address***
Rick Kittles, PhD
Morehouse School of Medicine
Presentation Title: Re-imagining Prostate Cancer Disparities in the Era of Precision Medicine

9:15 – 9:20 am ***Q&A***

9:20 – 9:40 am ***Jong Park, PhD***

	Moffitt Cancer Center *Presentation Title: Differentially Methylated Genes in Aggressive Prostate Cancer in African American and Puerto Rican Men*
9:40 – 9:45 am	***Q&A***
9:45 – 10:05 am	***King Jordan, PhD*** *Georgia Institute of Technology* *Presentation Title: Epigenetics and Cancer Disparities: When Nature Might be Nurture*
10:05 – 10:55 am	***Q&A***
10:55 – 11:15 am	***Kathryn Barry, PhD, MPH*** *University of Maryland School of Medicine* *Presentation Title: Neighborhood Disadvantage, Prostate Tumor RNA Expression of Stress-related Genes, and Aggressive Prostate Cancer*
11:15 –11:20 am	***Q&A***
11:20 – 11:40 am	***Vanessa Marshall, PhD*** *National Institutes of Health* *Presentation Title: Engaging Communities in Science to Improve Population Health and Reduce Health Disparities*
11:40 – 11:45 am	***Q&A***
11:45 – 11:55 am	***Coffee Break*** ***Cole Boardroom***

Tuesday Morning/Midday Session V: 11:55 am – 12:30 pm

	Chair: Shivani Chandel, PhD
11:55 – 12:05 pm	***Student Presentation #1 (15 mins)—Mr. Nicholas Cook***
12:05 – 12:10 pm	***Q&A for Student Presentation #1***
12:10 – 12:25 pm	***Student Presentation #2 (15 mins)—Ms. Kezhan Khazaw***
12:25 – 12:30 pm	***Q&A for Student Presentation #2***

Closing Ceremony & Student Awards: 12:30 – 1:45 pm

12:30 – 1:25 pm	***Announcement for Poster Winners (awards, photos, presentations, etc.)***
1:25 – 1:45 pm	***Closing Ceremony***
1:45 pm	***Boxed Lunch, Networking and Departure Cole Boardroom***

Speakers' Biographical Sketches and ***Abstracts***

Dr. William D. Figg

William D. Figg
National Cancer Institute, National Institutes of Health

Dr. Figg received a BS in Pharmacy from Samford University and a Doctor of Pharmacy from Auburn University. He completed a clinical internship at UAB Hospital and a fellowship in Drug Development at UNC Chapel Hill. He holds honorary degrees from Georgetown College and the Philadelphia College of Osteopathic Medicine. Dr. Figg joined the NCI in 1992. He is Acting Chief of the GU Malignancy Branch and Director of the Clinical Pharmacology Program. He is also co-director of the Office of Translational Resources and Associate Director of the Center for Cancer Research. Dr. Figg has >900 peer-reviewed publications.

The title of presentation: High-Throughput Drug Screening to Identify Novel Synergistic Combinations for mCRPC

Dr. Rick A. Kittles

Rick Kittles, Ph.D., is Professor in the Department of Community Health and Preventive Medicine and the Senior Vice President for Research at Morehouse School of Medicine. Dr. Kittles was previously Professor and founding Director of the Division of Health Equities within the Department of Population Sciences at the City of Hope (COH) and Associate Director of Health Equities of COH Comprehensive Cancer Center. Dr. Kittles is also co-founder and Scientific Director of African Ancestry, Inc. Dr. Kittles serves on many national and international steering committees and advisory boards. He has been a member of the Board of Scientific Counselors (BSC) for the National Human Genome Research Institute (NHGRI/NIH) and is Past Council Chair of the Minorities in Cancer Research (MICR) of the American Association for Cancer Research (AACR). Dr. Kittles served on the National Academies of Sciences, Engineering, and Medicine Committee on the "Use of Race, Ethnicity, and Ancestry as Population Descriptors in Genomics Research." Dr. Kittles' research has focused on understanding the complex issues surrounding race, genetic ancestry, and health disparities. He has been at the forefront of the development of genetic markers for ancestry and how genetic ancestry can be used in genetic studies on disease risk and outcomes, showing the impact of genetic variation across populations. He has published over 260 research articles and is well known for his research on prostate cancer and health disparities among African Americans. In March of 2012 Dr. Kittles presented the Keynote Address to the United Nations General Assembly, "International Day of Remembrance of Victims of Slavery and the Transatlantic Slave Trade."

Re-imagining Prostate Cancer Disparities in the Era of Precision Medicine

Rick Kittles

Morehouse School of Medicine

Prostate cancer remains one of the most significant health disparities affecting African American men, who continue to experience higher incidence, earlier onset, and disproportionate mortality compared to other populations. Despite decades of research, these inequities persist, underscoring the need for innovative approaches that move beyond traditional paradigms. The emergence of precision medicine offers unprecedented opportunities to reframe how we understand and address prostate cancer disparities. By integrating genetic ancestry, molecular profiling, environmental exposures, and social determinants of health, we can uncover the complex interplay of biology and context that

drives disease disparities. This talk will highlight recent advances in molecular epidemiology, biomarker discovery, and genomic medicine that are reshaping our capacity to predict risk, tailor interventions, and personalize treatment strategies. Equally important, it will emphasize the role of community engagement and equitable access in ensuring that precision medicine fulfills its promise for all populations. Re-imagining prostate cancer disparities through this lens requires bridging laboratory discoveries with real-world contexts, centering health equity as both a scientific and moral imperative.

Douglas Strand, PhD

UT Southwestern Medical Center
Department of Urology

Title of Presentation: A Cellular Anatomy of the Healthy Human Lower Urinary Tract

Abstract: Androgen deprivation induces lineage plasticity in both non-malignant and malignant prostate luminal epithelia. Recent efforts to define the molecular identity of various induced plastic states rely on a deep understanding of normal cellular identities. To better understand the molecular control of normal cellular differentiation, we created a multiomic cellular atlas of the healthy human lower urinary tract using single cell, single nuclear, and spatial transcriptomic platforms. These data provide a healthy reference for understanding prostate and other lower urinary tract diseases.

Short Bio: Douglas Strand, PhD received his PhD in Molecular and Cellular Biology at Baylor College of Medicine and went on to the urology department at Vanderbilt University for a postdoc. He started his lab in the urology department at UT Southwestern in 2014 to focus on the cellular pathogenesis of lower urinary tract dysfunction. His laboratory has developed a comprehensive human tissue repository that includes young and aged organ donor specimens as well as specimens from clinically annotated patients undergoing surgery. His laboratory uses single cell and spatial transcriptomic technologies in the human and mouse lower urinary tract and discovered several new cell types involved in homeostasis and disease. He is an active member of the NIH GUDMAP and HuBMAP consortia, as well as the international Human Cell Atlas.

Dr. Kathryn Barry

Title: Neighborhood disadvantage, prostate tumor RNA expression of stress-related genes, and aggressive prostate cancer

Abstract: African American men are more than 1.5 times as likely to develop prostate cancer and more than 2 times as likely to die from prostate cancer when compared with European American men. This is thought to be due to a variety of factors, including differences in access to care and use of screening. African American men are also more likely to develop aggressive forms of the disease, but the reasons are not well understood. It is critical to determine factors driving aggressive prostate cancer and underlying mechanisms to reduce the burden of aggressive disease and related disparities by race. There are growing data that living in disadvantaged neighborhoods, which more commonly affects African American individuals, is linked with a higher chance of developing aggressive prostate cancer. However, research gaps remain, including the neighborhood factors that are most important in risk, whether they interact with race, and underlying mechanisms. Chronic stress and its biological effects, such as increased inflammation, may play a role. We hypothesized that neighborhood disadvantage is associated with altered expression of stress-related genes, contributing to a higher risk of aggressive disease. Under this hypothesis, we used data from the University of Maryland Medical Center to evaluate 1) associations for neighborhood disadvantage metrics (including neighborhood deprivation, racial segregation, and historical redlining) and aggressive vs. non-aggressive prostate cancer – overall and by race, and 2) associations for the same metrics and prostate tumor RNA expression of stress-related genes. The present talk will also discuss future directions in this line of research.

Bio: Dr. Kathryn Barry is an Assistant Professor and cancer epidemiologist at the University of Maryland School of Medicine and University of Maryland Greenebaum Comprehensive Cancer Center. She received her PhD from Yale through the T32 Yale/NCI Partnership Doctoral Training Program in Cancer Epidemiology and received additional postdoctoral training in this area at the NCI (Occupational and Environmental Epidemiology Branch in the Division of Cancer and Epidemiology and Genetics). Her research aims to investigate environmental and occupational risk factors for cancer, gene-environment interactions, and

biomarkers of cancer risk and prognosis. Much of her current work focuses on cancer health disparities.

Dr. I. King Jordan

Title: Epigenetics and cancer disparities: when nature might be nurture.

Abstract: Despite an overall decrease in cancer mortality, survival disparities between racial and ethnic groups stubbornly persist. Black patients have the highest all-cancer mortality rate in the US, including common breast, colon, lung, and prostate cancer types, even though Whites show the highest overall rate of new cancer cases. We conducted an analysis of survival disparities of 33 cancers for 9,818 patients using the NCI Cancer Genome Atlas. We identified four cancer types with significant cancer survival disparities between racial and ethnic groups – breast invasive carcinoma, head and neck squamous cell carcinoma, kidney renal clear cell carcinoma, and skin cutaneous carcinoma – along with seven cancer-related genes that interact with genetic ancestry to contribute to the observed disparities. In this talk, I highlight the implications of our results pointing to epigenetic mechanisms, DNA methylation in particular, as a link between genetic (nature) and environmental (nurture) contributions to cancer health disparities. We found that genetic differences between groups could not explain the disparities we observed. There were, however, clear differences in group-specific patterns of DNA methylation and gene expression that were associated with cancer survival disparities. For example, hypomethylation of the PAQR6 gene promoter region in African ancestry patients is associated with higher gene expression and a greater risk of breast cancer mortality compared to European ancestry patients. These results suggest that changes in gene expression mediated by epigenetic mechanisms have a greater contribution to cancer survival disparities than group-specific genetic variants, consistent with a previously proposed role for epigenetics in health disparities.

Dr. I. King Jordan is Professor in the School of Biological Sciences and Director of the Bioinformatics Graduate Program at the Georgia Institute of Technology. Members of Dr. Jordan's laboratory at Georgia Tech conduct genetic epidemiology research with an emphasis on human population genomics and genetic ancestry inference in support of health equity. His group also develops bioinformatics software for large-scale genome sequence and functional genomic analyses. He established the Applied Bioinformatics Laboratory (ABiL) to provide bioinformatics data analysis and workforce development services to partners in the academic, non-profit, and industry sectors. In addition to his research and development efforts, Dr. Jordan is actively engaged in bioinformatics and

genomics capacity building, with a focus on global health in Africa and Latin America. He was named a Fulbright Scholar to Colombia, and he is the Co-Founder and Director of the PanAmerican Bioinformatics Network.

Dr. Vanessa Marshall

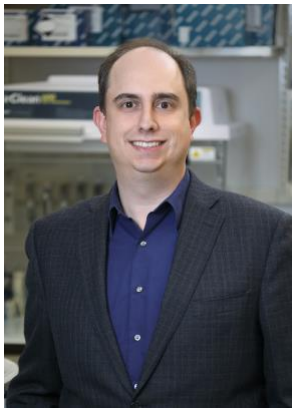
Title: Engaging Communities in Science to Improve Population Health and Reduce Health Disparities

Abstract: Social risks and social needs can contribute to poorer health outcomes and barriers across the cancer care and control continuum. By understanding the impact of social determinants of health on prostate cancer while implementing prevention and intervention studies, communities can improve outcomes among populations that experience health disparities. The presentation will focus on the National Institute on Minority Health and Health Disparities (NIMHD) programs, resources, funding, and training opportunities to address population sciences, social determinants of health, and community engagement to reduce health disparities.

Bio:

Dr. Vanessa Marshall is a Social and Behavioral Scientist Administrator (Program Officer) in the Division of Community Health and Population Science at the National Institute of Minority Health and Health Disparities (NIMHD). Her research focuses on improving health outcomes and promoting research to understand and address the multilevel determinants of factors that play a role in health disparities. She provides expertise in key research areas including minority health, health disparities, health services research, community-engaged research, clinical trials, public health, interventions, quality improvement, implementation, and evaluation. She has fostered innovative collaborations and partnerships to promote and support evidence-based research to inform clinical practice and policy. Dr. Marshall's experiences have allowed her to work at local, regional, national, and international levels to investigate risk and protective factors among various populations to address health disparities.

Dr. Adam Sowalsky



Title of Presentation: Intrinsic mediators of persistent AR-positive prostate tumors

Abstract: Androgen receptor (AR) expression is nearly universal in localized high-risk prostate cancer, yet a subset of tumors persist or recur following definitive local therapy, including surgery or radiation with androgen deprivation therapy (ADT). Emerging evidence from transcriptomic profiling of pretreatment biopsies reveals that this resistance is driven by intrinsic, context-dependent programs that exist prior to therapy; standard clinicopathologic risk stratification is insufficient to predict therapeutic sensitivity. Specific biologic subtypes within AR-positive tumors are intrinsically predisposed to resist standard therapy: in tumors treated with neoadjuvant ADT and AR-targeted agents, resistance is characterized by a low-AR activity, HER2-high transcriptional state that persists despite hormonal suppression and is selectively targetable with HER2 inhibition. By contrast, tumors destined to recur after radiotherapy plus ADT display elevated TGF-β activity at baseline, correlating with adverse genomic features, increased MRI-visible tumor volume, and long-term risk of metastasis and mortality. Importantly, these subtypes can be detected at diagnosis through gene expression profiling of routine biopsy material, enabling early identification of patients who may not benefit from conventional treatment alone. Together, these data highlight the need to incorporate molecular diagnostics into the management of localized high-risk prostate cancer to better define tumor biology, anticipate treatment failure, and guide rational therapeutic intensification or redirection.

Bio: Adam Sowalsky, Ph.D., is a Tenured Senior Investigator in the Genitourinary Malignancies Branch at the National Cancer Institute. His research centers on uncovering mechanisms of treatment resistance in aggressive prostate cancer and translating molecular insights into clinical strategies. Dr. Sowalsky leads a multidisciplinary laboratory that integrates genomics with clinical investigation and has provided leadership across multiple national scientific review panels, mentoring programs, and advisory committees, including the NIH Liquid Biopsy Scientific Interest Group, the Department of Defense Prostate Cancer Research Program, and the NCI's Center for Cancer Research Science Board.

Dr. Bal Lokeshwar

Title: Increased inflammatory milieu is a strong driver for cadmium-induced carcinogenesis in prostate epithelial cells.

Bal L. Lokeshwar1* Kunj Bihari Gupta1, and Truett Taylor1,

1Georgia Cancer Center, Augusta University, Augusta, GA 30912

* Presenter (BLOKESHWAR@augusta.edu)

The mechanism by which chronic exposure to some toxic environmental pollutants, such as the heavy metal Cadmium (Cd), causes prostate cancer or its progression is unclear at present. The countermeasures to prevent are even less known. We tested the hypothesis that chronic cadmium exposure induces upregulation of proinflammatory cytokines (IL-1β, IL-6, and IL-8), which individually or combined promote Cd-induced carcinogenesis and cancer progression. We tested this hypothesis on two non-transformed prostate epithelial cell lines, IL-8 expressing NHPrE1 and IL-8-negative-RWPE-1. These cells were continuously exposed to 10 μM Cadmium Chloride ($CdCl_2$) for 52-weeks and monitored for neoplastic transformation, tumor-associated phenotype, gene and protein expression patterns. Cells exposed to $CdCl_2$ showed increased cell proliferation and clonogenic growth and highly increased expression of IL-8, its receptors (CXCR1 and CXCR2), IL-1β, and other cytokines & related genes. While unexposed RWPE-1 cells did not express IL-8, it was the first cytokine to increase when exposed to $CdCl_2$, followed by activation of NF-κB (p65-rel), VEGF-A & B, and other protumor genes. Constitutively IL-8 expressing cells were also susceptible to transformation; gene silencing of IL-8 suppressed motility, invasion, and survival activity. Inhibition of IL-8 signaling activity suppressed Cd-exposure induced toxicity and pro-carcinogenic activity. The addition of ROS scavenger, N-Acetyl Cysteine, completely abolished Cd-induced changes and increased the inflammatory secretome in these cells. These studies suggest that the ROS-generated inflammatory response might be a significant factor, and inhibition of inflammatory-cytokine signaling, or ROS scavengers, is a countermeasure to prevent Cd-induced toxicity. Funding: GCC, MCGDF, & DOD-TERP Grant No. TX220325.

Biographical Sketch:

Dr. Bal Lokeshwar is a cancer biologist. For the last 30+ years, his academic activities have focused on investigating prostate, bladder, and breast cancers to understand them better and develop new treatments for these malignant diseases. His research has led to the identification of new therapeutics and tumor biomarkers to treat and monitor prostate and bladder cancers. Some of the novel anticancer compounds include a polyphenol called Ericifolin from allspice (Pimenta dioica), Ursolic Acid, and Oleanolic Acid, along with their molecular hybrids. He has published over 100 peer-reviewed articles and reviews, as well as eight book chapters. He has trained about 35 students and trainee clinicians. Currently, he is Dr. J. Harold Harrison, MD, a Distinguished University Professor at Georgia Cancer Center, Augusta University. His work is funded by endowments and peer-reviewed grants from DOD-Army Medical Research, the NIH and VA Medical Research. Title

Dr. Anna Woloszynska

Seminar Title:

Identifying Biomarkers and Potential Treatment Targets in Prostate Cancer Health Disparities

Anna Woloszynska, PhD, is an Associate Professor of Oncology in the Department of Pharmacology and Therapeutics at Roswell Park Comprehensive Cancer Center and a graduate faculty member at the State University of New York at Buffalo. She is actively involved in the NCI Cancer Center Support Grant (CCSG) Developmental Therapeutics (DT) program. Dr. Woloszynska has developed a functional genomics research program that focuses on the genetics and epigenetics of bladder and prostate cancer, aiming to uncover new tumor vulnerabilities and develop effective treatments. Her bladder cancer research centers on the cohesin complex member STAG2 and chromatin modifiers. In prostate cancer, she addresses racial disparities, with a particular focus on early-onset cases in African American men.

Abstract

Prostate cancer affects African American men at higher rates and with worse outcomes than European American men, but the biological reasons for this are not fully understood. This study compared tumor and nearby normal tissue from both groups using DNA methylation and gene expression analyses. The results showed clear ancestry-related differences in how genes are regulated in prostate tumors. Tumors from African American men had distinct patterns of DNA changes and lower activity of several genes involved in androgen signaling. Computer-based modeling predicted that tumors in African American men may respond differently to hormone therapy, particularly through changes in cell growth and immune-related pathways. In addition, certain gene activity patterns were linked to how long patients remained free from disease after treatment, with differences seen by ancestry. Overall, the findings suggest that prostate tumors in African American men may follow different biological paths and respond differently to standard treatments. Understanding these differences may help guide more personalized and effective treatment approaches.

Dr. Jiaoti Huang

Dr. Huang earned MD from Anhui Medical University and PhD from NYU. He did his residency at NYU and fellowship at Memorial Sloan-Kettering cancer center. He was on faculty at the University of Rochester and UCLA. He is currently Distinguished University Professor of Pathology, Pharmacology and Cancer Biology, Johnston and West Endowed Chair, and Pathology Department Chair at Duke.

Dr. Huang is an internationally renowned surgical pathologist and prostate cancer researcher. He has published more than 300 papers and his research lab has been continuously funded by federal agencies and private foundations for over 20 years.

The title of presentation: Discovering novel therapeutic targets for advanced prostate cancer

Daqing Wu presentation at the 14th National Symposium on Prostate Cancer

Title: Overcoming Prostate Cancer Chemoresistance through Ferroptosis-Targeted Drug Repurposing

Abstract

Chemotherapy, often combined with androgen-deprivation therapy, is usually the final treatment option for patients with metastatic prostate cancer. Despite initial responses, many patients eventually develop chemoresistance, leading to disease progression and poor clinical outcomes. Ferroptosis, a recently discovered form of cell death caused by iron-driven damage to cell membranes, offers a promising strategy to treat therapy-resistant cancer. However, the lack of clinically viable ferroptosis inducers remains a major barrier to bringing this approach to patients.

We established a high-throughput screening platform and tested many existing drugs for their ability to kill chemoresistant prostate cancer cells. Unexpectedly, we found that loratadine (brand name Claritin®), a safe, inexpensive, and widely used allergy medication, was highly effective. In mouse models, including tumors derived from patient samples, loratadine slowed tumor growth and enhanced the effects of chemotherapy. Mechanistic studies revealed that chemoresistant prostate cancer cells become hypersensitive to ferroptosis induction, a molecular vulnerability that can be therapeutically exploited. We further discovered that loratadine directly targets SLC7A11, a key protein cancer cells use to defend against ferroptosis. Blocking SLC7A11 disrupts the cells' antioxidant defenses, allowing iron to accumulate and trigger ferroptotic death. Interestingly, this effect appears to be independent of loratadine's antihistamine activity. These findings uncover a completely new anticancer mechanism for loratadine and suggest it could be rapidly repurposed as a treatment for drug-resistant prostate cancer.

In this presentation, I will share our discovery of loratadine's anticancer action, explain its cellular mechanism, and discuss its translational potential to quickly bring new hope to patients impacted by this aggressive form of prostate cancer.

Biographical Sketch:

Dr. Daqing Wu earned his Ph.D. in Biochemistry from Peking University, Beijing, China. He is a Professor of Biological Sciences at Clark Atlanta University and a faculty member of the Center for Cancer Research and Therapeutic Development. His research focuses on understanding how cancer spreads, how it develops drug resistance, and on discovering new treatments for advanced cancers. Dr. Wu's team investigates small-molecule drugs, natural products, and novel uses for existing medicines to accelerate the translation of discoveries into patient care. He is also the founder of MetCure Therapeutics, a company dedicated to transforming laboratory research into real-world therapies. His work is currently funded by the National Cancer Institute and the National Institute on Minority Health and Health Disparities.

Dr. Jong Y. Park

Dr. Jong Y. Park is a cancer epidemiologist at Moffitt Cancer Center, where he has worked since 1999. He earned degrees in Biology (BS), Microbiology (MS), Public Health (MPH), and Epidemiology (PhD) at University of Alabama at Birmingham, with postdoctoral training at Temple University. His research focuses on prostate cancer risk factors and health disparities. Dr. Park has published over 240 peer-reviewed articles and led 27 cancer research projects as Principal Investigator, with funding from NCI, DoD, Florida State, and industry.

Differentially methylated genes in aggressive prostate cancer in African American and Puerto Rican men.

This study aimed to identify differentially methylated genes between tumor vs. adjacent normal tissues, and aggressive vs. indolent prostate cancer (PCa). DNA methylation patterns in biological samples from African American (AA) and Puerto Rican men were assessed using the human Illumina Methylation EPIC array.

When comparing normal vs. tumor obtained from 120 African American patients, around 5,139 differentially methylated CpG sites were identified. We compared our results with those from the European American cohort from TCGA. 261 differentially methylated genes were identified only in AA patients. In addition, 2,061 differentially methylated CpG sites were identified when the grade group (GG)1 was compared with GG4/5.

With prostate tissues from 50 Hispanic Latino men from Puerto Rico (PR), we assessed DNA methylation patterns between aggressive and indolent PCa along with ancestry analysis. We identified 8,293 differentially methylated CpG sites in prostate tumor tissues compared with normal tissues. 366 differentially methylated genes were identified only in Puerto Rican patients. Regarding PCa aggressiveness, 141 differentially methylated CpG genes were identified.

The identification of DNA methylation profiles associated with PCa aggressiveness in AA and PR men will shed light on potential mechanisms contributing to PCa disparities.

Dr. Joseph Lachance

Biographical sketch:

Dr. Lachance is a Doc Blanchard Associate Professor in the School of Biological Sciences at Georgia Institute of Technology. He is the director of a GAANN training grant and chair of the MADCaP Network's genomics working group. Highlights of his population genetics research include the first pan-African GWAS of prostate cancer, detecting signatures of archaic introgression in African genomes, and the first application of polygenic risk scores to ancient humans. His lab has also explored why genetic predictions of disease risk generalize poorly across populations, with one key cause being ascertainment bias.

Joseph Lachance

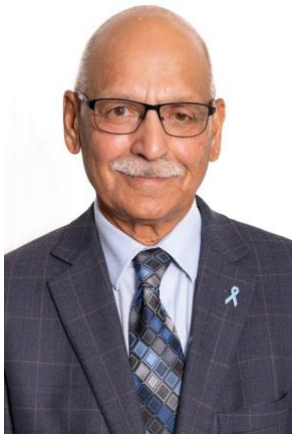
Title:

Heterogeneous genetic architectures of prostate cancer susceptibility in sub-Saharan Africa

Abstract:

Men of African descent have the highest prostate cancer incidence and mortality rates, yet the genetic basis of prostate cancer in African men has been understudied. We used genomic data from 3,963 cases and 3,509 controls from Ghana, Nigeria, Senegal, South Africa, and Uganda to infer ancestry-specific genetic architectures and fine-map disease associations. Fifteen independent associations at 8q24.21, 6q22.1 and 11q13.3 reached genome-wide significance, including four new associations. Intriguingly, multiple lead associations are private alleles, a pattern arising from recent mutations and the out-of-Africa bottleneck. These African-specific alleles contribute to haplotypes with odds ratios above 2.4. We found that the genetic architecture of prostate cancer differs across Africa, with effect size differences contributing more to this heterogeneity than allele frequency differences. Population genetic analyses reveal that African prostate cancer associations are largely governed by neutral evolution. Collectively, our findings emphasize the utility of conducting genetic studies that use diverse populations.

Dr. Shafiq A. Khan



Shafiq A. Khan, Ph.D.
Dr. Shafiq Khan is Georgia Research Alliance Eminent Scholar in Cancer Cell Biology and Professor in the Department of Biological Sciences. He served as the Director of the Center for Cancer Research and Therapeutic Development (CCRTD) from January 2004 to December 2021. He serves as the Principal Investigator of the NIH/NIMHD/RCMI program at Clark Atlanta University. Dr. Khan earned his Master's degree in Biological Sciences in 1976 from Quaid-i-Azam University in Islamabad, Pakistan, and his Doctorate in Reproductive Endocrinology in 1985 from the Karolinska Institute in Stockholm, Sweden. He was an Associate Professor in the Department of Cell Biology and Biochemistry at Texas Tech University Health Sciences Center, where he also served as the Director of Basic Research at the Southwest Cancer Center. Earlier, he was affiliated with the University of Muenster in Germany, the University of Toronto and the University of Kansas Medical Center.

Targeting Gαi2 Protein to Block Migration and Invasion in Prostate Cancer Cells

ABSTRACT

Tumor cell motility is the initial step during the process of migration, invasion and metastasis formation, and it is an essential component of dissemination of tumor cells from the primary tumor to local and distant sites. Numerous growth factors, chemokines, and hormones are responsible to bind to different types of membrane receptors, and to induce migratory and invasive behavior in prostate cancer cells. Several of these ligands bind and activate G-protein coupled receptors (GPCRs), which signal via heterotrimeric G-proteins in the form of activated Gα-GTP and Gβγ subunits. Previously we have shown that Gαi2 is highly expressed, and represents the predominant Gαi isoform in prostate cancer cells. We have also shown that knockdown of endogenous Gαi2 protein led to inhibition of in vitro cancer cell migration and invasion in prostate cancer cells, in response to different stimuli, including TGFβ, SDF-1α, and EGF acting through diverse membrane receptors. Gαi2 acts at two distinct sites to regulate cell migration and invasion: one is dependent on GPCR activation, and the second is independent of GPCR. This GPCR independent action, required

for EGF-induced cell migration, is independent or down-stream of PI3-kinase/AKT/mTOR/Rac1 activation. We have developed first-generation small molecule inhibitors, which specifically interfere with the activation of Gαi2 protein and block cell migration and invasion. Chemotherapy is normally the first line of treatment in cancer patients. Recent studies have shown that chemotherapy can be responsible of inducing the cancer cells to escape from death and migrate to distant sites to form metastases. Our recent studies showed that anti-androgens, docetaxel and HDAC inhibitors induce cell migration in prostate cancer cell models and simultaneous treatment with Gαi2 inhibitors blocks the migratory behavior induced by chemotherapy.

Dr. Ramesh Narayanan

Title Novel Inhibitors of AR, AR-splice variants, and GR for the Treatment of Refractory Castration-Resistant Prostate Cancer

Abstract Castration resistant prostate cancer (CRPC) that relapses from existing treatments is lethal and accounts for majority of the over 30,000 annual deaths in the U.S. Some of the mechanisms attributed to the development of CRPC and drug-resistant CRPC are over-expression of androgen receptor (AR), AR splice variants (AR-SVs), and glucocorticoid receptor (GR). Patients with tumors expressing AR-SV and GR have no effective treatment options. We have discovered molecules that bind to the AR N-terminus domain and induce degradation of the AR and AR-SVs by ubiquitination and proteasome pathways (Selective AR Degraders (SARDs). The lead molecule UT-34 (ONCT-534) in a phase 1 clinical trial reduced serum PSA, AR target genes, and tumor burden of patients who are refractory to current treatment options. Structural modifications of the SARDs led to covalently binding N-terminus domain (Selective AR Irreversible Covalent Antagonists (SARICAs) inhibitors that served as valuable tools to understand AR N-terminus domain structural details. SARICAs changed confirmation of AR and AR-SV and dissolved the liquid liquid phase separated condensates. Additionally, we have discovered triple inhibitors that degrade AR and AR-SV and inhibit GR, exhibiting outstanding activity in CRPC models expressing the three proteins. Collectively, these molecules and drug represent promising strategy to treat CRPC that are refractory to current treatment options.

Funding: NCI R01CA229164, NINDS R01 NS131106, UTHSC Center for Cancer Research, Muirhead Chair of Excellence

Photograph Attached

Biograph Dr. Ramesh Narayanan is a Professor and the Muirhead Chair of excellence in the College of Medicine at the University of Tennessee Health Science Center (UTHSC), Memphis, TN. Dr. Narayanan also serves as the Deputy Director of UTHSC Center for Cancer Research and as the interim Associate Dean of Research in the College of Medicine. He received his Ph.D. from the University of Madras, MBA from the University of Memphis, and did his post-doctoral fellowship at Baylor College of Medicine. Prior to joining UTHSC, Dr.

Narayanan served the biotech industry for nine years, where he contributed to multiple drug discovery programs to treat cancer and musculoskeletal diseases. The SARDs program that was out-licensed to biotech was coinvented in Dr. Narayanan's laboratory at UTHSC. He has published over 75 peer reviewed journal articles, co-authored several book chapters, and is an inventor in over 125 patents. His laboratory is funded by various federal agencies, including NCI, NINDS, DOD, and private industries.

Amina Zoubeidi, Ph.D.
Canada Research Chair (Tier 1) in Cancer Therapy Resistance
Professor, Department of Urologic Sciences, University of British Columbia
Senior Research Scientist, Vancouver Prostate Centre
Michael Smith Scholar

Title: Lineage Plasticity in Prostate Cancer: From Molecular Insights to Clinical Strategies

Abstract: Advances in the standard of care for prostate cancer have significantly improved treatment outcomes but have also driven the emergence of aggressive tumors characterized by lineage plasticity. This phenomenon, defined by the ability of cancer cells to switch identities, often arises early in response to therapy, as demonstrated by clinical studies showing the rapid adaptation of tumors under selective pressure. At the core of this plasticity lies a metastable transition state, an intermediate cellular phase that presents a critical window for therapeutic intervention. Dr. Zoubeidi will discuss how alterations in the epigenome and metabolome play a central role in driving this adaptability, with lineage plasticity largely governed by dynamic and reversible epigenetic dysregulation. She will present how changes in chromatin architecture create an environment that enables transcription factors to undergo “reprogramming,” ultimately facilitating tumor cell plasticity and therapy resistance. Given this inherent epigenetic and metabolomic plasticity, she will highlight how therapeutic strategies targeting these dynamic alterations offer a promising avenue for reversing resistance phenotypes.

Bio: Dr. Zoubeidi is a Tier 1 Canada Research Chair in Cancer Therapy Resistance. Her research program aims to uncover how standard care of prostate cancer therapies induce treatment resistance and controls phenotypic plasticity in prostate cancer. She published 130 peer reviewed manuscripts and received funding from national and agencies. She is currently the leader/PI of the Terry Fox New Frontiers Program Project on understanding lineage plasticity in treatment resistance. She is a Michael Smith Scholar received awards from UBC including Faculty of Medicine Distinguished Achievement Award in Overall, University of British Columbia, Award for excellence in mentoring Early Career Award, multiple Research Teaching Award for Excellence in Basic Science.

Charting a New Path for Immunotherapy in Prostate Cancer

Ravi A. Madan, MD
Senior Clinician
National Cancer Institute

Abstract

Historically, prostate cancer has been considered a “cold tumor,” but this oversimplified concept dismisses the potential for immunotherapeutics in prostate cancer. The first modern immunotherapeutic (beyond cytokines) approved by the FDA was sipuleucel-T in prostate cancer. Although it has faced marketing challenges, it demonstrated the potential of immunotherapy in prostate cancer to improve outcomes. Extensive studies with immune checkpoint inhibitors alone and in combination have not worked in prostate cancer, beyond a small subset of patients where immune checkpoints have tumor agnostic indications. In recent years bispecific T-cell engagers have demonstrated promising early data and are now in late stage clinical trials in prostate cancer. At the National Cancer Institute there is an ongoing effort to better understand the potential of immunocytokines as strategy to broadly impact the pleiotropic tumor microenvironment beyond T-cells and improve clinical outcomes.

Biography

Dr. Madan is a Senior Clinician at the National Cancer Institute (NCI), conducting clinical research in prostate cancer with a focus on early recurrence, PSMA imaging and immunocytokines. Dr. Madan received his M.D. from Rutgers Medical School in 2001 and completed his internal medicine residency at Rutgers University Hospital in June 2004. He joined the NCI Medical Oncology Branch as an oncology/hematology fellow in 2005. In 2009 he was appointed to the position of Assistant Clinical Investigator. In 2014 he was made the Clinical Director of the Genitourinary Malignancies Branch and in 2018 he was appointed to the role of Senior Clinician. Dr. Madan serves on review panels for the Veterans Association, Prostate Cancer UK, Prostate Cancer Foundation and the FDA’s Oncologic Drug Advisory Committee. His research program is looking to inform current standards of care while defining new strategies for therapeutic development in prostate cancer.

Poster Abstracts

STRESS-MEDIATED INFLAMMATORY PATHWAYS ARE DOWNREGULATED BY AN 8-WEEK BREATHWORK INTERVENTION: EVIDENCE FROM MULTI-OMICS PROFILING

Vinitha Ganesan,[1] Venugopalareddy Mekala,[1] Anita Chaudhary,[2] Sung Jung,[3] Priyam Singh,[1] Lauren A. Fowler,[1] Haiyan Qu,[1] Sahil Bajaj,[4] Snehaa Krishnan,[1] Navneet Baidwan,[1] Tapan Mehta,[1] Timiya Nolan,[1] Vandana Kalia,[2,5] Surojit Sarkar,[2,5] Nusrat Jahan,[1] Ritu Aneja.[1]

[1]The University of Alabama at Birmingham, [2]Seattle Children's Research Institute, [3]Baylor College of Medicine, [4]The University of Texas MD Anderson Cancer Center, [5]University of Washington School of Medicine.

Chronic stress negatively impacts cardiovascular and immune health. Accessible behavioral interventions are critically needed to reverse the effects of chronic stress. ORBIT model emphasizes Phase-I/II studies are required to ensure feasibility/preliminary effectiveness before advancing behavioral interventions to efficacy trials. To that end, we conducted an 8-week randomized controlled Phase II study of Sudarshan Kriya Yoga (SKY) which involves rhythmic, cyclic breathwork, in **stress-exposed healthy individuals**: 45 participants were either randomly assigned to SKY (n=30) or waitlisted control (n=15) groups. Outcomes included: primarily - feasibility (adherence, data collection, study completion), heart rate variability (HRV); secondarily - self-reported mental health, psychological thriving; exploratorily (cytokine profiling) measured in blood samples. We observed: (a) strong study completion (95.6%) and multi-source data collection (84.4%) rates in both groups, with high adherence to breathwork practice (4.27 times/week) within SKY group, **demonstrating feasibility of intervention and study processes**; (b) via linear mixed effects models, large effect sizes in SKY group (hedge's *g*) for improved HRV (*g*=-0.83), anxiety reduction (*g*=1.37), improved social connection (*g*=-0.98), and IL-6 reduction (*g*=0.93) at week-8, **demonstrating preliminary effectiveness of SKY**; (c) multi-omics (DNA-methylation, transcriptomics, proteomics) revealing **downregulation of key (NFkB-mediated inflammation, Th17, and NK cell-mediated cytotoxicity) pathways**, and flow cytometry validating reduction of Th17 and NK cell proportions in PBMC of SKY group at week-8. These signal SKY's potential to reverse chronic stress effects at multiple (psycho-neuro-immune) levels, laying the foundation for future well-powered confirmatory trials. We will examine multi-level effects of breathwork in other highly stressed populations such as **cancer survivors**.

HER2 AS AN ANCESTRY-ASSOCIATED THERAPEUTIC TARGET IN PROSTATE CANCER

Nicole Mavingire, Ph.D.[1], Abdulrahman M. Dwead, Ph.D.[1], Joy Solomon[2], Janelle Moore, M.S.[1], Moyinoluwa Adeniyi[2], Odunayo Oluokun[2], Jabril R. Johnson, Ph.D.[1], Mya Walker, Ph.D.[3], Isaiah Sailors[3], Serene Dowiri[3], Greisha L. Ortiz-Hernandez, Ph.D.[3], Jillian C. McDonough[3], Diana LeVasseur, M.S.[1], Jazlyn Farlough[1], Justin Tran[3], Frank Myers, M.D.[3], Fornati Bedell, M.D.[3], Zhirong Yin, M.D.[3], Rachel Martini, Ph.D.[1], Melissa B. Davis, Ph.D.[1], Clayton C. Yates, Ph.D.[4], K. Sean Kimbro, Ph.D.[1], Rick A. Kittles, Ph.D.[1], Tanya Dorff, M.D.[3], Cristina Magi-Galluzzi, M.D., Ph.D.[5], Soroush Rais-Bahrami, M.D.[6], Firas Kobeissy, Ph.D.[1], Yehia Mechref, Ph.D.[2], **Leanne Woods-Burnham, Ph.D.[1]**

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Human epidermal growth factor receptor 2 (HER2) overexpression in prostate cancer (PC) correlates with worse prognosis. We hypothesize that that unique ancestry-associated multi-omic molecular signatures may confer improved tumor response to anti-HER2 drug targeting. We detected moderate correlation with *HER2/ERBB2* and West African genetic ancestry (WAA) in prostate tissue through genotyping. We quantified HER2 expression by immunohistochemistry and detected HER2+ scores >2+ in 70% of primary PC tissue in our cohort of Black men (n=10). *HER2/ERBB2* was detected in PC cells by qPCR. In PC cells treated with anti-HER2 drug, we observed significantly reduced viability only in PC cells from Black patients. We observed a significant reduction in tumor volume in PDXs treated with anti-HER2 drugs. Systems biology revealed differential pathway and network expression in Black compared to white PC cells treated with anti-HER2 drug. By leveraging the unique characteristics of HER2+ tumors in Black men, an additional oncogenic pathway can be therapeutically targeted which will have a major impact in reducing progression to metastasis and reducing mortality for high-risk PC patients.

Acknowledgements: This study has been supported by NIH (KL2TR002381), NIH (UL1TR002378), PCF (20YOUN04), DoD Prostate Cancer Research Program (W81XWH2110038), NIH (TL1TR002382), NIH/NCI (U54CA118638), and NIH/NIMHD (2U54MD007602).

VITAMIN D INCREASES SENSITIVITY TO RADIOTHERAPY IN ADVANCED METASTATIC AFRICAN AMERICAN PROSTATE CANCER CELL LINES

LaKendria K. Brown, Ph.D.[1]; Jabril Johnson, Ph.D.[2]; Rick Kittles, Ph.D.[1]

[1] Morehouse School of Medicine; Atlanta, GA 30310; Department of Community Health and Preventive Medicine

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Background/Significance: Prostate cancer (PCa) disproportionately affects African-American (AA) men, with higher incidence and mortality rates than European-American (EA) men. Differences in radiation therapy responsiveness may impact outcomes, as studies suggest AA men may have a lower response to radiotherapy than EA men, potentially affecting therapeutic efficacy. Vitamin D, a steroid hormone with anti-proliferative effects on PCa cells, may play a role since AA men are more likely to be deficient, potentially contributing to reduced radiotherapy sensitivity. The active form of vitamin D, $1\alpha,25(OH)_2D_3$, shows promise in enhancing radiosensitivity. Our study investigates how vitamin D modulates transcriptome profiles in advanced-metastatic PCa cell-lines derived from AA men. **We hypothesize that vitamin D will modulate signaling pathways involved in radiotherapy differentially in AA advanced-metastatic PCa cell-lines than EA advanced-metastatic PCa cell-lines.**

Methods: Whole-transcriptome analysis was performed using RNA sequencing (RNAseq) on MDA-PCa-2b, an AA cell-line, and 22RV1, an EA cell-line. Both were treated with $1\alpha,25(OH)_2D_3$. Bioinformatic and pathway-enrichment analysis were conducted from RNAseq data to identify differential gene expressions between treated and untreated samples, pinpointing significant biological pathways that are impacted.

Results: RNAseq revealed significant gene expression changes in response to treatment in 22RV1 (FDR p value < 0.05). Hierarchical clustering heatmaps demonstrated clear separations between treated and untreated groups, indicating significant treatment effects on gene expression in both cell-lines. Principal Component Analysis (PCA) confirmed the distinct transcriptomic changes. Volcano plots identified TPD52L1/MOSMO in MDA-PCa-2b and TMPRSS2/CYP24A1in 22RV1. Venn analysis showed 245 overlapping DEGs, with 4,370 unique to MDA-PCa-2b and 234 to 22RV1. Pathway enrichment analysis identified IGF1R, PARP1, XRCC6, ATM, and XRCC5 as central mediators in radiosensitivity, with repression of IGF1R and ATM showing potential contributions to increased radiosensitivity in MDA-PCa-2b cells.

Conclusions and Implications: In conclusion, we highlight differential radiosensitivity response of AA cell-lines to $1\alpha,25(OH)_2D_3$ treatment, with a distinctive gene network. Our study highlights potential treatment with $1\alpha,25(OH)_2D_3$ and therapeutics targets to improve radiosensitivity in high-risk populations, thereby addressing and alleviating PCa disparities in AA men.

Acknowledgment of Funding: This research is supported in part by the Robert Smith Foundation and the U54CA118638 and U54MD007602 grants from the NIH/NCI.

Conflict of Interest: The authors declare no conflicts of interest.

PALMITOYL ACYTRANSFERASE ZDHHC7 STABILIZES NRF2 TO SUPRESS PROSTATE CANCER

Que Thanh Thanh Nguyen, PhD[1], Zhuoyuan Lin [2], Shivani Agarwal [2], Jindan Yu, MD, PhD[1,2,3,4]

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[2]Division of Hematology/Oncology, Department of Medicine, Northwestern University, Chicago, IL, USA.

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Background: Protein palmitoylation, the covalent attachment of a palmitate fatty acid to substrate proteins, is catalyzed by the ZDHHC family of 23 palmitoyl transferases. Our previous study identified ZDHHC7 as a key inhibitor of androgen signaling and a suppressor of prostate cancer (PCa) progression. In addition, we found that ZDHHC7 is down-regulated in the late-stage neuroendocrine PCa and its induction kills these cells. However, the role of ZDHHC7 in NEPC has not been well studied.

Methods: ZDHHC7 and its catalytically inactive mutant, ZDHHC7^{C160S}, was overexpressed in PCa cell lines by infection with lentivirus expressing either ZDHHC7 or ZDHHC7^{C160S}, followed by RNA sequencing (RNA-seq). Chromatin immunoprecipitation-qPCR (ChIP-qPCR) assay was performed in 22RV1 cells overexpressing ZDHHC7 or ZDHHC7^{C160S}, using an NRF2 antibody to enrich target-bound DNA regions, which were then quantified by qPCR. Western blot (WB) was used to assess protein expression levels.

Results: We found that overexpression of ZDHHC7, but not ZDHHC7^{C160S}, upregulated the expression of NRF2-target genes in 22RV1 and LNCaP cells, including *AKR1C1 (~7-fold), HMOX1 (~6-fold), SQSTM1 (~3-fold),* and *NQO1 (~2-fold)*. Mechanistically, this is likely due to increased NRF2 binding to those antioxidant response gene promoters following ZDHHC7 overexpression. Accordingly, we showed that NRF2 protein level was induced by ZDHHC7. Surprisingly, there were no apparent increase of NRF2 mRNA levels, suggesting that ZDHHC7 induces NRF2 protein stability. Indeed, WB analyses revealed that KEAP1, a key suppressor of NRF2 protein stability, was reduced by ZDHHC7.

Conclusions: Our results suggest that ZDHHC7 induced NRF2 protein level and transcriptional activity in PCa, likely by decreasing KEAP1 and thus increasing NRF2 protein stability.

Future directions: Future studies will focus on determining how ZDHHC7 stabilizes NRF2 protein, potentially by palmitoylating KEAP1 to promote KEAP1 degradation or induce

structural changes in palmitoylated KEAP1 to release NRF2. Additionally, a more comprehensive understanding of ZDHHC7's function in NEPC will be conducted.

LORATADINE OVERCOMES PROSTATE CANCER CHEMORESISTANCE VIA SLC7A11/XCT INHIBITION AND FERROPTOSIS INDUCTION

Xin Li [1, 2], Alira Danaher [1], Jedidiah Zhu [1], Nicholas Cook [1], Nathan J. Bowen [1], Jason M. Wu [3], Min Qui [3], Yuhong Du [3], Haian Fu [3], Adeboye O. Osunkoya [3], Mehmet A. Bilen [3] Omer Kucuk [3], Daqing Wu [1, 2, 3, 4]

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Background: Chemoresistance remains a major clinical challenge in the treatment of prostate cancer (PCa). Ferroptosis, an iron-dependent form of regulated cell death driven by lipid peroxidation, has emerged as a promising strategy to overcome chemoresistance. However, clinically viable ferroptosis inducers are not yet available.
Methods: Phenotypic screening identified Loratadine as a specific inhibitor of chemoresistant PCa cells. Molecular and cellular approaches were conducted to elucidate the mechanism of action of Loratadine. Its *in vivo* efficacy was evaluated in clinically relevant xenograft models.
Results: Loratadine, a widely used FDA-approved antihistamine, selectively killed chemoresistant PCa cells by directly targeting the cystine/glutamate antiporter SLC7A11, thereby inducing ferroptosis. These effects appeared to be independent of Loratadine's antihistamine function. *In vivo*, Loratadine significantly inhibited tumor growth and synergized with docetaxel across multiple PCa models, including patient-derived xenografts.
Conclusion: This study reveals a previously unrecognized mechanism of action for Loratadine and provides compelling preclinical rationale for its repurposing as a clinically viable ferroptosis inducer. With its established safety profile, low cost, and broad availability, Loratadine offers a unique and rapidly translatable strategy to overcome chemoresistance in PCa.

Acknowledgements: National Institutes of Health grants R01CA256058, R42CA217491, and U54MD007590 (Project 1).

EFFECTS OF CURCUMIN ON AIP-MEDIATED AHR SIGNALING IN CASTRATION-RESISTANT PROSTATE CANCER

Nana-Ama Acquah & Dr. Joann Powell**,** Cancer Center for Research and Therapeutic Development, Clark Atlanta University, Atlanta, GA

Castration-resistant prostate cancer (CRPC) is a major clinical challenge, often sustained by abnormal aryl hydrocarbon receptor (AhR) signaling. AhR stability relies on the AhR-interacting protein (AIP), which protects AhR from degradation and maintains its activity. Persistent AhR signaling supports tumor survival and progression. Curcumin, a natural polyphenol with anti-cancer properties, has been shown to modulate AhR activity. We hypothesize that curcumin interferes with AIP-mediated stabilization of AhR, thereby reducing its signaling capacity in CRPC cells. CRPC cell lines were treated with increasing concentrations of curcumin. AhR–AIP interactions were examined by co-immunoprecipitation, and protein levels of AhR and AIP were analyzed by Western blotting. Beta-actin was used as a loading control. We anticipate that curcumin treatment will weaken AhR–AIP binding and decrease AhR protein stability, as evidenced by reduced protein expression on Western blots. These changes are expected to correlate with decreased CRPC proliferation in a dose-dependent manner, with the most pronounced effects occurring at intermediate curcumin concentrations. Curcumin may act as a natural inhibitor of AIP-dependent AhR signaling. By destabilizing the AhR–AIP complex, curcumin could reduce tumor cell survival and highlight AIP as a novel therapeutic target in advanced prostate cancer. These findings support further evaluation of curcumin and related compounds as adjuncts to conventional therapies.

Acknowledgements: This research was supported by Title III

Word Count: 245

ADVANCING TROP-2 DIRECTED THERANOSTICS

Camryn Carter**,** Christopher Massey1, Shiqin Liu1, Qing-Qing Yin2, Alifiani Bonita Hartono1, Hyeonji Hwang1, Bhavya Pandey1, Trinity Tran1, Anna Wu1, 3, Owen Witte1,4, Peter Clark1, Tanya Stoyanova1,5

We aimed to develop a new and more precise way to treat aggressive forms of cancer, such as castration-resistant prostate cancer (CRPC) and triple-negative breast cancer (TNBC). These cancers are difficult to treat and often express high levels of a protein called Trop2. Trop2 is overexpressed in many epithelial cancers that occur at high levels. This makes it a promising target, but past treatments targeting Trop2 haven't been specific enough, leading to harmful side effects.We used a theranostic approach — a method that combines therapy and diagnostics — using a human antibody designed to bind to Trop2. The diagnostic version is tagged with a radioisotope called zirconium-89, allowing us to visualize how much Trop2 is present in the tumors and where the antibody travels in the body. This helps identify which patients might benefit most from the treatment.Once the antibody pharmacokinetics have been validated, we move to the therapeutic version, which is labeled lutetium-177. This delivers therapeutic radiation to the cancer cells, reducing damage to healthy cells. We tested these antibodies in mice implanted with human cancer cells that either express high or low levels of Trop2.Our goal is to create a smarter, more personalized treatment option for cancers that currently lack safe and effective therapies. This research lays the groundwork for using Trop2-targeted theranostics to both detect and destroy cancer with greater accuracy and fewer side effects

Acknowledgements:these studies were supported by the NIH/NCI grant and NIH/NIMHD RCMI grant.

LORATADINE AS A POTENTIAL FERROPTOSIS INDUCER AGAINST CHEMORESISTANCE CANCER

Nicholas Cook[1], Xin Li[1], Nathan Bowen[1], Alira Danaher[1], MaKayla Thomas[1], Saani Alhassan[1], Briah Arms[1], Daqing Wu[1]

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Cancer is the second leading cause of death in the United States. Chemotherapy is a frontline treatment for metastatic cancers. Despite the success of chemotherapy treatment drugs, many patients eventually develop resistance to chemotherapy, leading to disease relapse and poor survival outcomes without a cure. Understanding the mechanism of chemoresistance and identifying new therapeutic strategies are therefore critical. Ferroptosis is a form of regulated cell death that is driven by the accumulation of iron, lipid peroxidation, and plasma membrane rupture, and its induction has been shown to overcome chemoresistance in preclinical settings. Nonetheless, clinically viable ferroptosis inducers are not yet available. Loratadine, an antihistamine antagonist of H1R and commonly used for treatment of allergies, has demonstrated selective and high potency in chemoresistance in KB cell lines. Loratadine showed similar effects compared to erastin, a known inducer of ferroptosis. Loratadine treatment in drug resistant KB cells increased the accumulation of iron, lipid peroxidation, and depleted intracellular glutathione. RNA-seq analysis revealed that loratadine treatment rapidly increased the expression of ferroptotic and oxidative stress genes. *In vivo*, loratadine treatment reduced tumor size in athymic nude mice as monotherapy and in combination with paclitaxel. These results support the repurposing of loratadine as a novel therapeutic strategy to overcome chemoresistance and improve clinical outcomes in cancer patients.

Acknowledgements: National Institutes of Health grants R01CA256058, R42CA217491, and U54MD007590 (Project 1)

SIMULATED MICROGRAVITY ENRICHES AGGRESSIVE PROSTATE CANCER STEM CELL CLUSTERS: A NOVEL IN VITRO MODEL

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Prostate cancer is the most common and leading cause of cancer-related mortality among men, particularly within African American populations. The cellular and molecular mechanisms underlying the deadly disease remain insufficiently characterized. This presents a significant obstacle to developing effective treatments for metastatic prostate cancer. Anchorage-independent growth of circulating prostate tumor cell clusters is crucial for metastasis. However, there is no reliable in vitro model to comprehensively investigate the molecular and functional properties of these tumor cell clusters in the laboratory. To address this limitation, we established LNCaP cell clusters, designated LN-G1, LN-G2, and LN-G3, by sequentially culturing LNCaP cells in a simulated microgravity bioreactor called Slow Turning Lateral Vessel (STLV). Quantitative PCR analysis demonstrated that LN-G1, LN-G2, and LN-G3 cell clusters expressed higher levels of cancer stem cell markers CD44, CXCR4, and ALDH1A than the parental LNCaP cell, and exhibited a reduced expression of embryonic stem cell markers OCT4, NANOG, and SOX2. Functional assays indicate that, unlike LNCaP cells, LN-G1, LN-G2, and LN-G3 cells possessed enhanced and distinct growth and clonogenic abilities in both monolayer and nonadherent conditions. These findings suggest that cancer stem cell-like clusters conferring unique molecular and functional heterogeneity constitute a specific, aggressive form of stemness distinct from the pluripotent state of embryonic cells and may contribute to metastatic castration-resistant prostate cancer. Moreover, the STLV bioreactor represents a promising tool for enriching cancer stem cell populations and elucidating the mechanisms underlying lethal prostate cancer, which may facilitate the therapeutic strategies to improve patient outcomes.

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INFILTRATING B CELLS PROMOTE CELL MIRGRATION OF HUMAN PROSTATE CANCER CELLS

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Prostate cancer survival rate diminishes drastically once the cancer metastasizes. One of the factors that plays a role in the progression of cancer cells to metastasis is the signaling within the tumor microenvironment. The interaction of the components within this environment, such as infiltrating B cells with cancer cells, can lead to dual roles in the progression of cancer development. B cells are known for their role in antibody production during immune response in the body. However, it remains largely unknown of the different roles that B cells may play within the tumor microenvironment. Our preliminary data showed that infiltrating B cells were at a higher density near cancer cells in human prostate cancer tissues, as compared to the benign tissue regions. We hypothesized that infiltrating B cells would promote the progression of prostate cancer cells. In this study, we utilized Transwell® assays to evaluate the migration of human prostate cancer cells in the presence or absence of B cells. We also evaluated if the secreted factors of the B cells mediate prostate cancer cell migration using the Transwell® assay of human prostate cancer cells. Our findings showed an increase in migration of human prostate cancer cells when they were co-cultured with B cells and with the recombinant proteins identified to be secreted by B cells. Altogether, these results revealed that infiltrating B cells can play a critical role in prostate cancer migration/metastasis, through their secretion factors.
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EVALUATION OF ANCESTRY-ASSOCIATED HER2 AND ANDROGEN RECEPTOR EXPRESSION IN MEN AT RISK FOR PROSTATE CANCER USING LIQUID BIOPSY

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Black men suffer an increased incidence and mortality rate of prostate cancer (PC) in comparison to white men at a rate that is nearly doubled. After controlling for socioeconomic factors, the health disparity of incidence and mortality still exists among Black men, suggesting that biological determinants could be a key driver of differing outcomes. Androgen receptor (AR) and human epidermal growth factor 2 (HER2) are known to promote advanced disease. Significant evidence exists for the role that HER2 plays in breast cancer; however, the function of HER2 in PC remains obscure. Preliminary data from our group and others suggest a higher prevalence of HER2 expression in PC tumors from Black men compared to non-Black men. We hypothesize that there is a positive correlation between HER2 expression and WAA in Black men. Blood sera were collected from men at multiple study sites. We observed associations between HER2, AR, and WAA using enzyme-linked immunosorbent assay (ELISA) and ancestry genotyping. Our findings contribute evidence towards our investigator-initiated protocol to target HER2 in a clinical trial using a precision medicine approach.

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COMBINING LORATADINE AND BROMOCRIPTINE TO OVERCOME CHEMORESISTANCE IN PROSTATE CANCER – A DRUG REPURPOSING APPROACH

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Prostate cancer is the second leading cause of cancer-related deaths in men in the United States, with over 35,770 deaths projected in 2025. While androgen deprivation therapy, surgery, and chemotherapy are effective initial treatments, many cases eventually develop chemoresistance, limiting treatment options. To address this challenge, we investigated the repurposing of loratadine (Claritin) and bromocriptine, two existing FDA-approved drugs, for their efficacy against chemoresistant prostate cancer cells.

In our study, we tested these compounds individually and in combination on C4-2BtaxR100 and CABR cells, both resistant to taxane-based chemotherapy. Cell viability was assessed using the CCK-8 assay after 72 hours of drug exposure. Bromocriptine alone had an IC50 of 0.30 μM, while loratadine's IC50 was 1.36 μM in CABR cells. Notably, when combined, the IC50 of loratadine at a constant 0.5 μM bromocriptine was reduced to 0.09 μM, and bromocriptine's IC50 at a constant 0.5 μM loratadine was lowered to 0.079 μM, demonstrating a synergistic effect.

These findings suggest that loratadine and bromocriptine significantly enhance cytotoxicity when used together, offering a promising strategy for treating chemoresistant prostate cancer. Given their established safety profiles and widespread clinical use, repurposing these drugs could accelerate the development of novel treatment options for patients with limited alternatives.

Building on these findings, our research is now focused on AB43549, a loratadine analog developed in our lab. Preliminary results indicate that AB43549 is more potent than loratadine, effectively killing chemoresistant prostate cancer cells at lower concentrations. We are currently evaluating its efficacy in combination with cabazitaxel to further explore its potential as a therapeutic agent.

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CONSTITUTIVE ARYL HYDROCARBON RECEPTOR ACTIVITY REDUCES SENSITIVITY TO BORTEZOMIB IN PROSTATE CANCER CELLS, AN EFFECT AMPLIFIED BY LEPTIN EXPOSURE.

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The aryl hydrocarbon receptor (AhR) plays a role in regulating diet-induced obesity and is a nuclear transcription factor involved in the transcriptional regulation of target genes, such as enzymes of xenobiotic metabolism (CYPs, UGTs) or genes involved in the transcriptional regulation. Induced enzymes facilitate the metabolism of various endogenous compounds and drugs, such as bortezomib. We report that in an *in* vitro model for prostate cancer, the constitutive activity of AhR promotes bortezomib resistance which is exacerbated by leptin signaling. Leptin is known to contribute to cancer progression and has an emerging role in chemoresistance. Of note, both leptin receptor and aryl hydrocarbon receptor are overexpressed in prostate cancer cell lines. Additionally, our lab previously reported an integrative bioinformatics analysis using TCGA data (n=498) which revealed a positive correlation in AHR and LEPR mRNA expression in prostate cancer tissue. Previously published data from our lab demonstrated that AhR activity leads to androgen independence, increased metastatic potential and disease progression. AhR has been shown to be an integral driver of prostate cancer progression and further investigation is needed into AhR potential drug target for the treatment of advanced prostate cancer.

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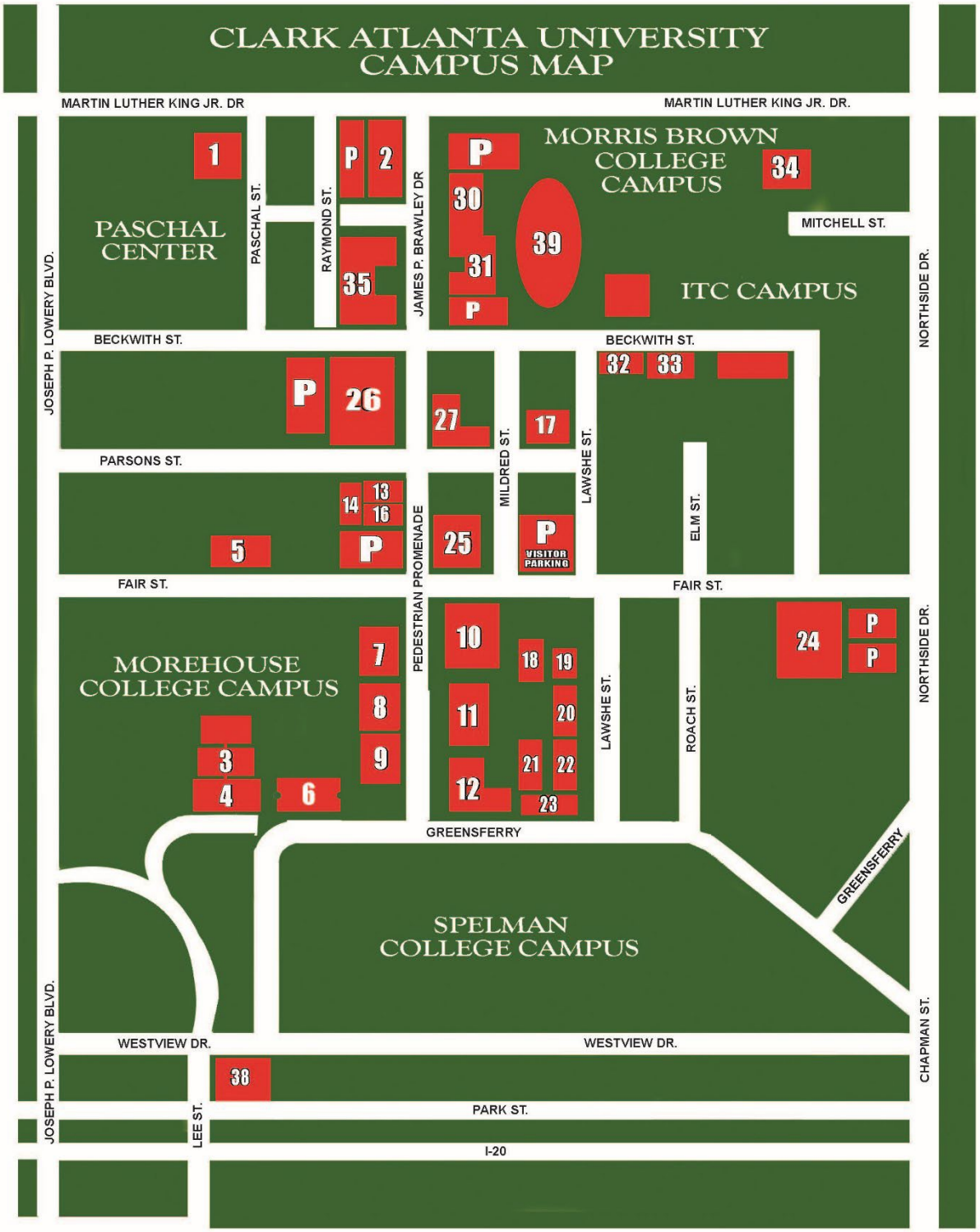

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