NIH NATIONAL CANCER INSTITUTE

CENTER TO REDUCE CANCER HEALTH DISPARITIES

CURE CONTINUING UMBRELLA OF RESEARCH EXPERIENCES

A program of the National Institutes of Health

Professional Development Workshop

August 3–4, 2023 | NCI Shady Grove, Rockville MD

Table of Contents

Welcome Letter

August 3, 2023

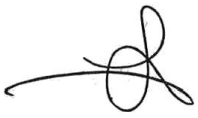
Dear Attendees,

The National Cancer Institute's (NCI) Center to Reduce Cancer Health Disparities (CRCHD) welcomes you to the 2023 Professional Development Workshop (PDW 2023). This year's theme is "Empowering Your Journey to a Sustainable Research Career." Our goals for the Workshop are for you to learn strategies to engage with your peers and your mentorship team, network with fellow grantees and NCI and CRCHD staff, understand NCI and CRCHD opportunities, refine your grant writing skills, and help you define your career path. You will receive information about NCI's current cancer research priorities, techniques and skills to attain an independent research position, and tools that will sustain you as you continue to build resilience during your path forward. This year's Workshop will be held in person to provide the opportunity to meet NCI program staff, meaningfully interact with researchers at different levels, and create scientific and personal networks that will equip and support you going forward. You will gain insight into current CRCHD-sponsored research during the poster session and hear details of select posters during the accompanying flash talk session. Current and past Continuing Umbrella of Research Experiences (CURE) grantee attendees will share their experiences throughout their journey and offer helpful tips, ideas, and do's and do not's. We hope that you leave the two-day PDW 2023 equipped with new skills and empowered to continue your journey to a successful cancer research career.

We are excited to welcome our current cohort of scholars from the Intramural Continuing Umbrella of Research Experiences (iCURE). This program extends CURE into the NCI Intramural Research Program and offers mentored research experiences on the NCI campuses. We are also pleased to welcome Scholars from the Early Investigators Advancement Program (EIAP)—a new NCI initiative that facilitates the advancement of scientists from diverse backgrounds to become independent researchers.

We are aware of the skills, experience, and knowledge that each one of you brings to the Workshop, and we encourage and look forward to your active participation and engagement. We are confident that we will all enjoy a rewarding Workshop that will enhance the network of cancer and cancer health disparities researchers and propel you forward in your career.

Sincerely,



Sanya A. Springfield, Ph.D.
Director, Center to Reduce Cancer Health Disparities

Agenda

NCI Center to Reduce Cancer Health Disparities
Professional Development Workshop
Empowering Your Journey to a Sustainable Research Career

Thursday, August 3, 2023

NCI Shady Grove – Conference Rooms TE 406/408/410 (Lower Level)

7:00–8:00 AM **Registration & Poster Set-Up**

8:00–8:05 AM **Opening Remarks**
Dr. Laritza M. Rodriguez
NCI CRCHD

Welcome Remarks
Dr. Sanya A. Springfield, Director
NCI CRCHD

8:05–8:20 AM **NCI Priorities**
Dr. LeeAnn Bailey, Chief, Integrated Networks Branch
NCI CRCHD

CRCHD Mission
Dr. Behrous Davani, Chief, Diversity Training Branch
NCI CRCHD

8:20–9:20 AM **Plenary Session: The Journey to Success**
Moderator: Dr. LeeAnn Bailey
NCI CRCHD

8:25–8:45 AM Dr. Jelani C. Zarif
Johns Hopkins University School of Medicine

8:45–9:05 AM Dr. Clayton C. Yates
Johns Hopkins University School of Medicine

9:05–9:20 AM **Discussion**

Agenda

9:20–10:50 AM **Plenary Session: Successful Stories: From Career Development Award to Research Independence**
Moderators: Dr. Laritza M. Rodriguez and Dr. Shahrooz Vahedi
NCI CRCHD

9:25–9:45 AM Dr. Yamilé Molina
University of Illinois Cancer Center

9:45–10:05 AM Dr. Jasmine McDonald
Columbia University Herbert Irving Comprehensive Cancer Center

10:05–10:25 AM Dr. Raymond D. Blind
Vanderbilt University School of Medicine

10:25–10:50 AM **Discussion**

10:50–11:05 AM **Break**

11:05 AM–12:05 PM **Plenary Session: The Importance of Mentors and Sponsors in Career Development**
Moderators: Dr. Anil Wali and Dr. Chantel F. Fuqua
NCI CRCHD

The objective of this panel discussion is to learn strategies for building a productive mentor/mentee relationship. Panelists will address topics including how to identify a mentor, building a productive scientific relationship, early identification of issues and how to approach them, what can be done when things go wrong, and establishing expectations.

11:10–11:50 AM **Panelists:**
Dr. Efrén J. Flores
Massachusetts General Hospital

Dr. Elyse R. Park
Massachusetts General Hospital

Dr. Charles R. Rogers
Medical College of Wisconsin

Dr. Derek M. Griffith
Georgetown University

11:50 AM–12:05 PM **Discussion**

Agenda

12:05–12:20 PM **Success Stories: The Impact of CURE**
Moderator: Mr. Brian Davis
NCI CRCHD

Presenters:
Dr. Elva Arredondo
San Diego State University

Dr. Chyke Doubeni
The Ohio State University

Dr. Manuel L. Penichet
University of California, Los Angeles

12:20–1:35 PM **Lunch (on your own)**

1:35–1:50 PM **Networking Session**
Facilitator: Ms. Katelyn Garfinkel
NCI CRCHD

The objectives of this speed networking session are to provide a forum for participants to 1) meet one another and 2) enhance their professional networks.

1:50–2:50 PM **Flash Talks**
Facilitators: Dr. Whitney Barfield Steward, Dr. Shadab Hussain, Ms. Fulera Salami, and Dr. Gregory Adams, Jr.
NCI CRCHD

2:50–4:50 PM **Poster Session I**

4:50–5:00 PM **Evaluation and Adjournment of Day 1**
Dr. Behrous Davani
NCI CRCHD

Agenda

NCI Center to Reduce Cancer Health Disparities
Professional Development Workshop
Empowering Your Journey to a Sustainable Research Career

Friday, August 4, 2023

NCI Shady Grove – Conference Rooms TE 406/408/410 (Lower Level)

7:00–8:00 AM **Registration & Poster Set-Up**

8:00–8:05 AM **Welcome to Day 2 (Recap of Day 1 and Day 2 Preview)**

8:05–9:05 AM **Plenary Interactive Session: Roundtable Discussion to Empower Your Career**
Moderator: Dr. Sylvia E. Long
NCI CRCHD

The objective of this session is to provide interactive engagement among CRCHD Program Directors and attendees discussing different topics and tips for success and networking. Each interactive discussion is scheduled for 20 minutes. Participants will rotate among the tables, participating in up to three discussions.

Roundtable Topics

1. Understanding and Planning for Academic Promotion and Faculty Membership as an Underrepresented Investigator
2. Reviewing Summary Statements and Submitting an Amended Application
3. Building Relationships to Advance Your Career
4. Fostering Your Creative Talents to Advance Science
5. Strategies to Identify the Appropriate NCI Program Staff and Funding Opportunities for Your Research Project(s)
6. Understanding the Business Side of Biomedical Science and Grants Management
7. Your Elevator Pitch - Getting Over Those Awkward Initial Introductions
8. Harnessing Your Emotional Intelligence Against Imposter Syndrome

Agenda

9:05–10:35 AM **Plenary Session: NCI Divisions Presentations**
Moderator: Dr. Tiffany Wallace
NCI CRCHD

9:10–9:15 AM	Dr. Brandy Heckman-Stoddard *NCI Division of Cancer Prevention*
9:15–9:20 AM	Dr. April Oh *NCI Division of Cancer Control and Population Sciences*
9:20–9:25 AM	Dr. Lillian Kuo *NCI Division of Cancer Biology*
9:25–9:30 AM	Dr. Leah Hubbard *NCI Division of Cancer Treatment and Detection*
9:30–9:35 AM	Dr. Tiffany Wallace *NCI Center to Reduce Cancer Health Disparities*

10:35–10:50 AM **9:35–10:35 AM** **Roundtable Discussions**

10:50–11:50 AM **Break**

11:50 AM–1:20 PM **Poster Session II**

1:20–2:20 PM **Lunch (on your own)**

2:20–3:20 PM **Poster Session II contd.**

Plenary Session: Helpful Pointers for Successful Grants Writing
Moderator: Dr. Sangeeta Ghosh
NCI CRCHD

2:25–2:40 PM	Dr. Tiffany Wallace *NCI CRCHD*
2:40–2:55 PM	Dr. Behrous Davani *NCI CRCHD*
2:55–3:10 PM	Dr. Anil Wali *NCI CRCHD*
3:10–3:20 PM	**Discussion**

Agenda

3:20–3:35 PM

Success Stories: Tips on Grant Writing, Resilience, and Mentorship and Being a Mentee
Moderator: Mr. Brian Davis
NCI CRCHD

Presenters:
Dr. Adana A.M. Llanos
Columbia University

Dr. Juan L. Mendoza
University of Chicago

Dr. Patricio I. Meneses
Fordham University

Dr. Lauren E. McCullough
Emory University

3:35–3:55 PM

Poster Award Ceremony
Master of Ceremony: Dr. Whitney Barfield Steward
Award Presenter: Dr. H. Nelson Aguila, Deputy Director
NCI CRCHD

3:55–4:00 PM

Evaluation and Closing Remarks
Dr. Behrous Davani
NCI CRCHD

Speaker Bios

Speaker Bios

Sanya A. Springfield, Ph.D.—Director, CRCHD, NCI

Dr. Springfield is Director of the National Cancer Institute's (NCI's) Center to Reduce Cancer Health Disparities (CRCHD), where she supports programs, initiatives, and activities to spawn cancer health disparities research, increase workforce diversity, and create networks for community outreach, education, and engagement. Within NCI, Dr. Springfield is a member of the Scientific Program Leadership, where she champions the need for continued investment for diversity training and education programs and to reduce cancer health disparities.

Previously, Dr. Springfield was Chief of the NCI Diversity Training Branch, where she conceived, implemented, and oversaw the Continuing Umbrella of Research Experiences (CURE) program. Utilizing a unique, holistic, training pipeline approach, CURE seeks to increase the number of competitive cancer researchers from racial and ethnically diverse, and other underserved populations. Dr. Springfield expanded the CURE program, launching a middle school program as part of a CURE early intervention strategy and an Intramural CURE (iCURE) program aimed at enhancing the diversity of the NCI intramural research workforce. Prior to this, Dr. Springfield had expanded the diversity training landscape through the creation and implementation of the Partnerships to Advance Cancer Health Equity (PACHE). PACHE aims to improve the cancer research infrastructure at institutions serving underserved health disparity populations and underrepresented students and enhance the ability of NCI-Designated Cancer Centers to address cancer health disparities in their communities.

Dr. Springfield serves on a variety of trans-NCI and trans-NIH scientific and programmatic committees focused on disparities research, workforce diversity, and inclusive excellence, including NCI's Equity Council and the NIH Common Fund's Faculty Institutional Recruitment for Sustainable Transformation Program Working Group Coordinators. For her vision and leadership in promoting diversity in biomedical research, she was honored with the NIH Director's Award and the NCI Director's Award. Dr. Springfield also serves as a member of the American Association for Cancer Research Minorities in Cancer Research Council and the Science Education and Career Development Committee, and played a vital role in establishing the annual The Science of Cancer Health Disparities in Racial and Ethnic Minorities and the Medically Underserved conference.

Dr. Springfield received her Ph.D. in physiology and biophysics from Howard University and was the third African American neuroscientist in the world. After completing her postdoctoral studies at the Robert Wood Johnson School of Medicine, she joined the faculty at City College of New York. Dr. Springfield left the academic ranks to serve as a Program Director at the National Science Foundation and then entered NIH as a Grants Associate, after which she joined NCI.

H. Nelson Aguila, D.V.M.—Deputy Director, CRCHD, NCI

Dr. Aguila is Deputy Director of NCI's CRCHD. In this capacity, he plays a central role in coordinating the day-to-day functions of the Center and development of strategic planning, priority-setting, and management of CRCHD's disparities research, diversity training, and community education/outreach efforts.

Previously, Dr. Aguila served as Chief of CRCHD's Diversity Training Branch. Prior to coming to NIH, Dr. Aguila worked at the U.S. Food and Drug Administration as a Reviewer Toxicologist at the Center for Veterinary Medicine. Earlier in his career, Dr. Aguila held senior research scientist positions in neuropathology at the University of Miami and, later, in cancer gene therapy at Aventis-Gencell. Dr. Aguila earned his Doctor of Veterinary Medicine degree at Austral University in Chile and trained as a neurobiologist at The University of Texas Southwestern Medical Center, Dallas.

Speaker Bios

Gregory Adams, Jr., Ph.D., M.S.—Program Director, CRCHD, NCI

Dr. Adams has been a Program Director in the Office of the Director of NCI's CRCHD since 2021. In this role, his primary responsibility is program management for the Intramural Continuing Umbrella of Research Experiences program.

Prior to joining CRCHD, Dr. Adams was an Intramural Research Training Award postdoctoral fellow/research fellow at the National Heart, Lung, and Blood Institute under Dr. Clare Waterman, where he studied and surveyed the organization of organelles and actin regulatory proteins mediating leader bleb-based migration of cancer cells in nonadhesive confined environments mediating the microenvironment. As a postdoctoral fellow/research fellow, Dr. Adams joined the Office of Intramural Training and Education, where he helped expand the number of disadvantaged students interested in health disparities-focused biomedical and healthcare careers through training opportunities to explore the scientific enterprise.

Dr. Adams earned his B.S. in biology from Morehouse College. He received both his master's degree in biomedical research—where he studied colorectal cancer and adenomas under Dr. Felix Aikhionbare—and his Ph.D. in biomedical science in physiology—where he studied cytoskeleton interactions to facilitate breast cancer cell migration under Dr. Xuebiao Yao—from Morehouse School of Medicine. He is a member of the American Society for Cell Biology and the American Association for Cancer Research (AACR), as well as AACR's Minorities in Cancer Research.

Elva Arredondo, Ph.D.—Professor and Core Investigator, San Diego State University

Dr. Arredondo is a Professor in the Department of Psychology and Core Investigator at the Institute of Behavior and Community Health at San Diego State University. She graduated from the University of Washington with a degree in psychology and attained her Ph.D. in clinical psychology from Duke University. She completed her clinical internship in behavioral medicine at the University of California, San Diego.

Dr. Arredondo's research has focused on testing the effectiveness of new and adapted behavioral interventions to reduce Latino health disparities in cancer and other chronic diseases. Her training in implementation science has given her the skills to develop methods that promote the systematic uptake of evidence-based interventions into practice. She has led Center grants (U54) and been a collaborator on multisite community trials involving large cohorts. As Co-Lead of the Intervention Methods Group in HealthLINK, Dr. Arredondo supports researchers seeking to develop new or adapt existing evidence-based interventions for different populations and settings. Currently, Dr. Arredondo and her team are pilot testing the effectiveness of a 12-week mother-daughter intervention (Conmigo) promoting physical activity in pre-adolescent Latinas compared with a control condition. She is also leading randomized controlled trial aims to enhance the capacity in faith-based organizations to implement and sustain multilevel innovations to improve physical activity. Her overarching goal is to translate and scale up evidence-based interventions that promote physical activity and cancer screening in diverse communities.

LeeAnn Bailey, M.B.B.S., Ph.D., M.S.—Chief, Integrated Networks Branch, CRCHD, NCI

Dr. Bailey has been Chief of the Integrated Networks Branch of NCI's CRCHD since 2016. In this role, she manages, develops, and assesses strategies for enhancing the integration and dissemination of diversity training, women's health, and sexual and gender minority efforts within and across NCI, as well as within the scientific community and underserved communities through NCI-supported networks. She also identifies and leverages opportunities to address unmet needs in cancer health disparities research.

Prior to joining NCI, Dr. Bailey was a healthcare consultant at Deloitte Consulting LLP. She also has been a principal investigator researching tissue engineered products and cellular inflammatory responses at the National Institute of Standards and Technology as well as an adjunct professor at Morgan State University.

Speaker Bios

Dr. Bailey received her M.B.B.S. (M.D. equivalent) from the University of Adelaide Medical School with an emphasis on aboriginal health and pediatric oncology. She also has a Ph.D. in biochemistry and molecular genetics and an M.S. in biological and physical sciences from the University of Virginia School of Medicine.

Raymond D. Blind, Ph.D.—Assistant Professor, Vanderbilt University School of Medicine

Dr. Blind is an Assistant Professor of Medicine in the Division of Diabetes, Endocrinology and Metabolism with secondary appointments in the Biochemistry and Pharmacology Departments in the School of Medicine at Vanderbilt University. He completed his Ph.D. at New York University Medical Center, then did two postdocs at the University of California, San Francisco, one in traditional biomedical research and one in medical education in Dar es Salaam, Tanzania. Dr. Blind's scholarship, service, and advocacy for inclusion, outreach, and justice have elevated Vanderbilt's national and international reputation. While at Vanderbilt, he has won 5 young investigator awards and has been invited by external colleagues to organize 6 international scientific conferences and speak at 12 international conferences, as well as 15 national and 6 local institutions, including the Historically Black Colleges and Universities Meharry, Fisk, and Tennessee State University. His research articles have solved deep, longstanding barriers to progress in understanding nuclear receptor regulation using cutting-edge integrative structural biology, chemical genetics, and functional genomics, and now are being applied to discover new ways bile acid metabolism and gene expression are regulated.

As a Diversity Liaison and through a long list of other efforts, Dr. Blind has led a sustained effort to implement the values of inclusion and justice at Vanderbilt. He is a chartered member of an NIH study section and is sole principal investigator on pioneering projects to determine how signaling molecules directly regulate chromatin biology (R01 GM132592), establish small-molecule structure/activity relationships for full-length Liver Receptor Homolog-1 (R01 GM138873), and develop a chemical-genetic system to halt growth of human glioblastoma cells (R21 CA243036). He also serves as co-investigator on three other projects. Dr. Blind has worked with national policy makers within the Endocrine Society and American Association for Biochemistry and Molecular Biology to improve inclusion efforts, develop conferences, enhance career development, and expand society journals.

Behrous Davani, Ph.D.—Chief, Diversity Training Branch, CRCHD, NCI

Dr. Davani has been Chief of the Diversity Training Branch of NCI's CRCHD since 2022. In this capacity, he plays a central role in the strategic planning of the Branch and program implementation to enhance workforce diversity in cancer research. He oversees management of NCI's diversity-focused training programs, including the extramural Continuing Umbrella of Research Experiences (CURE) program.

Prior to his appointment as Chief, Dr. Davani served as a Program Director in the Division for Research Capacity Building, National Institute of General Medical Sciences (NIGMS), where he oversaw the IDeA Regional Entrepreneurship Development Program and managed grants for the Centers of Biomedical Research Excellence, IDeA Network of Biomedical Research Excellence, Science Education Partnership Awards, and Native American Research Centers for Health Programs. Before his time at NIGMS, Dr. Davani was a Program Director in NCI's CRCHD, during which time he led the development, implementation, and management of various programs that address cancer health disparities. In this capacity, Dr. Davani managed, comanaged, or coordinated multiple CURE programs, Comprehensive Partnerships to Advance Cancer Health Equity (CPACHE U54) partnerships, and the PACHE Feasibility Studies to Build Collaborative Partnerships in Cancer Research (P20) program, among others.

Prior to first joining NCI, Dr. Davani served as a Scientific Review Manager in the Peer Review Science and Management Division of SRA International. While at SRA, he oversaw and managed the scientific review process for multiple research programs, including breast, ovarian, and lung cancer, for the Congressionally

Speaker Bios

Directed Medical Research Programs. He received his Ph.D. in molecular endocrinology from Karolinska Institute in Stockholm, Sweden.

Dr. Davani is committed to developing innovative research and educational programs to advance health equity and promote diversity in biomedical research.

Brian Davis—Communications Manager, CRCHD, NCI

Mr. Davis joined NCI's CRCHD in 2019, first as Senior Health Communications Specialist and subsequently as Communications Manager. The Center works to reduce cancer disparities and increase workforce diversity in cancer research, and Mr. Davis leads the Center's Communications Core and supports communications for the NIH-wide Faculty Institutional Recruitment for Sustainable Transformation program. Before his time with NCI, he worked for nearly 3 years at the American College of Gastroenterology, where he served as Director of Media Relations and Managing Editor and Senior Writer for the College's magazine. From 2008–2016, Mr. Davis served in several communications and publications positions at the American Health Law Association. He holds a B.A. in journalism from the University of South Carolina.

Chyke Doubeni, M.D., M.P.H.—Chief Health Equity Officer, Wexner Medical Center, and Associate Director for Diversity, Equity and Inclusion, The Ohio State University Comprehensive Cancer Center–Arthur G. James Cancer Hospital and Richard J. Solove Research Institute, The Ohio State University

Dr. Doubeni joined The Ohio State Wexner Medical Center as Chief Health Equity Officer in July 2022. He also serves in The Ohio State University Comprehensive Cancer Center–Arthur G. James Cancer Hospital and Richard J. Solove Research Institute as Associate Director for Diversity, Equity and Inclusion and holds a faculty appointment as a Professor in the Department of Family and Community Medicine.

In addition to his roles within the Medical Center, Dr. Doubeni works closely with the Office of Academic Affairs (OAA). He is a leader in the RAISE initiative focused on recruiting new faculty who have a research focus on health equity topics. Working with OAA and the health science colleges, he leads development and implementation of a new Center for Health Equity within the University that will serve as an interdisciplinary incubator for health equity research collaboration across campus and provide opportunities for professional development and training in the field.

Dr. Doubeni is tasked with leading Ohio State's continued efforts to address the underlying drivers of disparities in healthcare that adversely impact marginalized groups and foster more equitable care and health outcomes. With a land-grant focus on research, education, and patient care that improves the lives of those in the community and state, Dr. Doubeni leads the vision and strategic direction of the Medical Center's health equity and healthy community initiatives in collaboration with leaders, faculty, staff, and learners. He brings together diversity, equity, and inclusion; anti-racism; and civic and community engagement efforts. Under his leadership, the Center will develop new and enhance existing clinical programs and care delivery mechanisms, as well as community engagement and outreach programs, to equitably improve health outcomes in the communities it serves.

Prior to joining Ohio State, Dr. Doubeni served at Mayo Clinic as the inaugural Director of the Mayo Clinic Center for Health Equity and Community Engagement Research. In addition, he served as Director of the Community Engagement Program in the Mayo Clinic Center for Clinical and Translational Science; Deputy Director for community outreach and engagement, including minority accrual, on the Mayo Clinic Cancer Center Executive Committee; and Professor of Family Medicine at Mayo Clinic College of Medicine and Science. Nationally recognized for his work in cancer prevention and public health, he is a current member of the NCI Board of Scientific Advisors, as well as a Section Editor for diversity, equity, and inclusion of the

Speaker Bios

American Gastroenterological Association's *Gastroenterology* journal. He served from 2017 to 2021 as one of 16 members of the prestigious U.S. Preventive Services Task Force (USPSTF), which makes evidence-based recommendations about clinical preventive services to promote the health of all Americans and beyond. In 2021, he served as lead author for the USPSTF's report "Addressing Systemic Racism Through Clinical Preventive Service Recommendations from the US Preventive Services Task Force" published in the *Journal of the American Medical Association*.

Prior to joining Mayo Clinic, Dr. Doubeni was the Harrison McCrea Dickson, M.D., and Clifford C. Baker, M.D., Presidential Professor at the University of Pennsylvania and served as Chair of the Department of Family Medicine and Community Health at the University of Pennsylvania School of Medicine.

Dr. Doubeni received his Bachelor of Medicine, Bachelor of Surgery, from the University of Lagos College of Medicine in Nigeria. After completing additional training in the United Kingdom, he completed a family medicine residency at Duke University and a preventive medicine residency at the University of Massachusetts, where he also earned a Master of Public Health degree and served as the medical school's interim Associate Vice Provost for Diversity. He also completed a fellowship with the NCI Scholars Program. His numerous accolades include a 2010 Presidential Early Career Award for Scientists and Engineers from the U.S. President for accomplishments in research, mentoring, and community service and the 2019 Sadie Gerson Distinguished Scholar Award from the University of Pittsburgh.

Efrén J. Flores, M.D.—Associate Professor, Harvard Medical School, and Associate Chair, Mass General Brigham Radiology Inclusion, Equity and Community Health

Dr. Flores currently serves as a board-certified staff radiologist in thoracic radiology at Massachusetts General Hospital (MGH), is an Associate Professor of Radiology at Harvard Medical School, and is also an Associate Chair of Mass General Brigham Radiology Inclusion, Equity and Community Health. Dr. Flores received a B.S. in biology from the University of Puerto Rico, Rio Piedras, and an M.D. from the University of Puerto Rico School of Medicine, and subsequently completed both a residency and a fellowship as a board-certified radiologist at MGH. Dr. Flores' health services research focuses on understanding health disparities to inform the development of novel transdisciplinary programs at the intersection of equity, innovation, and healthcare to enhance health access among high-priority patient populations. In partnership with a growing network of collaborators and community organizations, he has developed a nationally recognized health services research program focused on elucidating disparities in radiology care due to the social determinants of health and developing community-based, culturally sensitive interventions focused on increasing access to cancer screening and radiology care. Dr. Flores' clinical experience and personal background serve as strong foundations for his nationally recognized research work, resulting in multiple peer-reviewed publications, scientific presentations, extramural grant funding, and service on local and national committees focused on health equity.

Chantel F. Fuqua, Ph.D.—Program Director, CRCHD, NCI

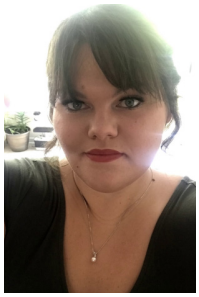
Dr. Fuqua has been a Program Director in the Diversity Training Branch of NCI's CRCHD since March 2023. She supports Diversity Supplements and the NIH Common Fund's Faculty Institutional Recruitment for Sustainable Transformation program.

Before joining NCI, Dr. Fuqua worked as a Program Director at the American Association for the Advancement of Science (AAAS), where she managed a National Science Foundation-funded STEM education portfolio focused on broadening participation in science, workforce diversity, and inclusive pedagogy at the undergraduate, graduate, and postdoctoral levels. Prior to that, Dr. Fuqua worked as a Director of Faculty and Educational Initiatives at the Association of American Medical Colleges, where she managed the Minority Faculty Leadership Development Seminar, Mid-Career Faculty Leadership Development Seminar, and Grant Writers Coaching Workshop for National Institutes of Health Awards, and previously led a pilot program

Speaker Bios

at seven academic medical centers focused on holistic review for faculty recruitment and retention to increase faculty diversity. In addition, Dr. Fuqua served as a 2016–2018 AAAS Science and Technology Policy Fellow in the National Science Foundation's Directorate for STEM Education, supporting the Louis Stokes Alliance for Minority Participation program. Dr. Fuqua also has experience teaching biology, biochemistry, and chemistry at various higher education institutions, most recently at the University of Maryland Global Campus. Before arriving at AAAS, Dr. Fuqua oversaw and managed several programs as a Health Science Analyst at Leidos, providing scientific, evaluation, and technical support to NIH and the U.S. Army Medical Research and Materiel Command in the management of the Congressionally Directed Medical Research Programs and other federal scientific programs.

Dr. Fuqua received her B.S. in chemistry from Saint Louis University and her Ph.D. in biomedical sciences from Meharry Medical College.



Katelyn Garfinkel, M.B.A—Program Specialist, CRCHD, NCI

Ms. Garfinkel has been a Program Specialist in CRCHD's Diversity Training Branch since 2021. She contributes to many CRCHD-supported programs and activities, such as the Intramural Continuing Umbrella of Research Experiences, Early Investigator Advancement Program, Career Track, and Diversity Supplements. Before joining NCI, she worked with Aramark for 6 years at a Wildlife Training Center in Shepherdstown, WV and 1 year at American Public University as an academic advisor. She holds a bachelor's degree in sociology (2014, Shepherd University) and a master's degree in business administration (2021, Southern New Hampshire University), and her passion is working with people and finding ways to get them engaged and feel included.

Sangeeta Ghosh, Ph.D.—Program Director, CRCHD, NCI

Dr. Ghosh has been a Program Director in the Diversity Training Branch of NCI's CRCHD since December 2022. In this role, she manages NCI Exploratory/ Developmental Grant (R21), Youth Enjoy Science Research Education Program (R25), and Kirschstein National Research Service Award Predoctoral Fellowship to Promote Diversity in Health-Related Research (F31-Diversity) programs for NCI.

Prior to joining CRCHD, Dr. Ghosh served as Scientific Review Manager at GDIT, supporting peer review of the Congressionally Directed Medical Research Programs (CDMRP). While at GDIT, Dr. Ghosh oversaw the peer review process of biomedical applications submitted in basic and clinical research areas, including the Peer Reviewed Medical Research Program and Ovarian Cancer Research Program of CDMRP. She was also responsible for planning, coordinating, directing, and implementing all tasks to determine the scientific and technical merit of applications submitted for review.

Before joining GDIT, Dr. Ghosh was a Scientific Manager at Leidos supporting programmatic review of CDMRP, where she was involved in managing preapplication reviews, programmatic reviews, and business meetings of CDMRP's biomedical research programs.

Dr. Ghosh received her Ph.D. in biotechnology from Jamia Hamdard University, New Delhi, India. After completing her postdoctoral studies at the University of Virginia, she joined as faculty at The University of Texas Health Science Center, San Antonio, where she developed translational projects focused on understanding the effect of exercise on diabetes, aging, and cardiovascular and metabolic diseases.

Speaker Bios

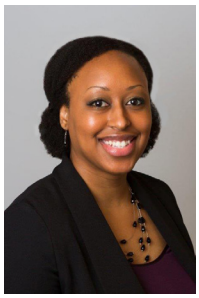
Derek M. Griffith, Ph.D.—Co-Director, Racial Justice Institute; Director, Center for Men's Health Equity; Chair, Global Action on Men's Health; and Professor, Georgetown University

Dr. Griffith is a Founding Co-Director of the Racial Justice Institute, Founder and Director of the Center for Men's Health Equity, Member of the Lombardi Comprehensive Cancer Center, and Professor of Health Management & Policy and Oncology at Georgetown University. He also serves as Chair of Global Action on Men's Health, a global men's health advocacy organization. Trained in psychology and public health, Dr. Griffith's program of research focuses on developing anti-racism approaches to achieve racial, ethnic, and gender equity in health. His research has explored how notions of manhood, trustworthiness, intersectionality, and individual tailoring can be incorporated into community-based and policy strategies to promote health and well-being. Dr. Griffith is a contributor to and editor of three books and the author of over 150 peer-reviewed manuscripts. He has been the principal investigator of research grants from the American Cancer Society, the Robert Wood Johnson Foundation, and several NIH Institutes. Dr. Griffith serves on the editorial boards of several public health and men's health journals. Recently, he received a citation from the President of the American Psychological Association "for his extraordinary leadership in addressing the impacts of racism on the health and well-being of the nation and specifically for African American and Latino men."

Brandy Heckman-Stoddard, Ph.D., M.P.H.—Chief, Breast and Gynecologic Cancer, NCI

Dr. Heckman-Stoddard received a Doctor of Philosophy degree in molecular and cellular biology at Baylor College of Medicine focusing on the intersection of Rho and IGF signaling in mammary gland development and breast cancer before joining NCI as a Cancer Prevention Fellow. During the fellowship, she completed a master's degree in public health at the Johns Hopkins Bloomberg School of Public Health working with the Institute for Global Tobacco Control and the Evidence-Based Practice Center. During her time at NCI as a Fellow, she focused on breast cancer prevention research, including preclinical development and early clinical trials.

Dr. Heckman-Stoddard's research focuses on drug development for breast cancer prevention and biomarker development. She is particularly interested in local delivery of agents, alternate dosing strategies, biomarkers of efficacy to reduce the number needed to treat, and targeting of stem cells. She serves as Program Director for the Early Phase Breast Cancer Prevention Clinical Trials grants portfolio and Scientific Monitor of early phase breast cancer clinical trials within the NCI Division of Cancer Prevention Early Phase Prevention Consortia. Dr. Heckman-Stoddard is also the NCI lead for an NCIN/National Institute of Diabetes and Digestive and Kidney Diseases collaboration examining cancer incidence within the Diabetes Prevention Program Outcomes Study, a randomized study of metformin and lifestyle intervention versus placebo.



Leah Hubbard, Ph.D.—Program Director, Translational Research Program, and Chief Diversity Officer, Division of Cancer Treatment and Diagnosis, NCI

Dr. Hubbard is a Program Director in the Translational Research Program and Chief Diversity Officer in the Division of Cancer Treatment and Diagnosis at NCI. She has been a member of the Translational Research Program since 2013 and manages Specialized Programs of Research Excellence (SPOREs) in brain and head & neck cancer. Dr. Hubbard also participates in a number of NIH and NCI initiatives, including the U19 Glioblastoma Therapeutics Network Request for Applications (RFA), the P20 Cancer Health Disparities SPORE RFA, the NIH UNITE Initiative, and the NCI Equity and Inclusion Program. Dr. Hubbard received her Ph.D. in immunology at the University of Michigan and continued her postdoctoral training at the National Institute of Allergy and Infectious Diseases at NIH. Her research training is in the area of innate immunity, with specific expertise in lung macrophage function and host defense, eicosanoid signaling, and syngeneic mouse modeling.

Speaker Bios

Shadab Hussain, Ph.D.—Program Director, CRCHD, NCI

Dr. Hussain joined the CRCHD Office of the Director as a Program Director in December 2022. She is part of the team contributing to the Center's overall evaluation efforts and supporting the administration and programmatic management of the NIH Common Fund's Faculty Institutional Recruitment for Sustainable Transformation program.

Prior to NCI, Dr. Hussain joined the National Center for Advancing Translational Sciences (NCATS) in 2020 as a Presidential Management Fellow in the Education Branch. In this capacity, she assisted with different projects of the Education Branch, such as programming course evaluation surveys, conducting statistical analyses for research reports, and contributing to translational science education research. During her fellowship, she completed a 6-month rotation at the NIH Chief Officer for Scientific Workforce Diversity office supporting NIH-wide Diversity, Equity, Inclusion, and Accessibility (DEIA) initiatives and programs. She also contributed to NCATS-wide DEIA initiatives through participation in the Health Disparities Working Group and supporting Center-wide initiatives as a DEIA strategist. Dr. Hussain received her doctorate in developmental and psychological sciences from the Stanford Graduate School of Education. For her dissertation, she examined the role of bicultural identity development in the psychological and academic outcomes of South Asian college students. She also assisted on research projects focusing on the socioemotional outcomes of American Indian/Alaska Native secondary school students.

Lillian Kuo, Ph.D.—Program Director, NCI

Dr. Kuo is a Program Director at NCI in the Cancer Immunology, Hematology, and Etiology Branch. Prior to joining NCI, she served as a Program Officer at the National Institute of Allergy and Infectious Diseases and a Program Analyst at the National Center for Advancing Translational Sciences. She was a postdoctoral fellow at NCI and received her Ph.D. from The University of Texas Southwestern and her B.S. from the University of Wisconsin.

Adana A. M. Llanos, Ph.D., M.P.H.—Associate Professor, Mailman School of Public Health, Columbia University

Dr. Llanos is an Associate Professor in the Department of Epidemiology at Columbia Mailman School of Public Health. As a cancer and molecular epidemiologist, her independent research program—which is supported largely by funding from NCI and the National Institute on Minority Health and Health Disparities—seeks to understand the molecular and sociobiologic causes of inequities in cancer outcomes. A major focus of Dr. Llanos' work for the past 10+ years has been the examination of factors that contribute to increased breast cancer incidence at younger ages, increased incidence of more aggressive tumors, and increased mortality among Black and African American women. In addition to her academic research, scholarship, and consulting work, Dr. Llanos is actively involved in service through engagement with community-based organizations, particularly those whose mission includes providing timely public health advocacy, education, and outreach, and cancer survivorship support among members of the community. Dr. Llanos is also the founder and principal of Wynn Biomedical Consultants.

Dr. Llanos received a doctorate in genetics and human genetics from Howard University and a Master of Public Health degree in epidemiology from The Ohio State University (OSU) College of Public Health. She received postdoctoral training in the Division of Oncology at the Lombardi Comprehensive Cancer Center at Georgetown University and in the Center for Population Health and Health Disparities at the OSU Comprehensive Cancer Center.

Speaker Bios

Sylvia E. Long, Ph.D., C.P.H.—Program Director, CRCHD, NCI

Dr. Long has been a Program Director in the Integrated Networks Branch of NCI's CRCHD since December 2022. Before joining NCI, Dr. Long was Communications Director in the Office of Clinical Research Training and Medical Education, NIH Clinical Center, and, earlier, a Management Analyst at the Centers for Medicare & Medicaid Services. Additionally, she is a faculty member at the Foundation for Advanced Education in the Sciences at NIH, where she teaches Communication in Biomedical Science, Special Topics in Communication for Healthcare Leadership, and Communication and Health Disparities.

Dr. Long received her Ph.D. in communication studies from Indiana University of Pennsylvania, where her research explored associations between perceived credibility, health belief, and willingness to comply with nonpharmaceutical intervention recommendations. Her findings yielded the development of the Perceived Credibility and Severity Model of Compliance, which she continues to employ in health communication and social determinants of health research.

Lauren E. McCullough, Ph.D., M.S.P.H.—Associate Professor, Rollins School of Public Health, Emory University, and Visiting Scientific Director, American Cancer Society

Dr. McCullough is an Associate Professor in the Department of Epidemiology at the Rollins School of Public Health and Visiting Scientific Director at the American Cancer Society. Her overarching research interests are in cancer epidemiology; specifically, the role of social and structural determinants of health in the breast tumor microenvironment, as well as disparities in cancer outcomes. Her research program integrates molecular epidemiology, epigenetics, and other biomarkers for disease risk and progression; environmental and social epidemiology; health services research; and causal inference methods. The goal of her research program, BRIDGE, is to improve cancer outcomes in underserved populations by *bridging* clinical and population research, molecular and social epidemiology, and scientists with the communities they serve. In doing so, she hopes to identify culturally relevant and sustainable targets for pharmacologic, behavioral, and policy intervention.

Jasmine McDonald, Ph.D.—Assistant Director, Cancer Research, Training, and Education Center, and Assistant Professor, Mailman School of Public Health, Columbia University

Dr. McDonald is an Assistant Professor in the Department of Epidemiology at the Mailman School of Public Health. She received her doctorate from the Biological Sciences in Public Health Program at Harvard University with a concentration in immunology and infectious disease. She has postdoctoral training in breast cancer epidemiology from the University of Pennsylvania and Columbia University. Her research portfolio integrates individual-level factors (e.g., health behaviors), the macroenvironment (e.g., physical, social, microbial environment), and biology (e.g., endocrine disruption, tumor microenvironment) to inform breast cancer etiology across and within critical windows of breast development—including the prenatal, puberty, pregnancy, and postpartum windows. While relevant to all women, Dr. McDonald's portfolio is nested within populations that have a higher burden of cancer, including those with a genetic predisposition, racial and ethnic minorities, and young women. An avid teacher and mentor, Dr. McDonald was awarded the 2021 Columbia University Teaching Award for her dedication and excellence in teaching, mentoring, and community engagement. She teaches Cancer Epidemiology within Mailman; is Assistant Director of the Cancer Research, Training, and Education Center at the Herbert Irving Comprehensive Cancer Center (HICCC); and is Co-Director of the YES! in THE HEIGHTS Program at HICCC (formerly known as CURE). The YES! program aims to engage students from overlooked and underserved backgrounds, New York-based high school teachers, and the community in discussions around cancer health equity.

Speaker Bios

Juan L. Mendoza, Ph.D.—Assistant Professor, Pritzker School of Molecular Engineering, University of Chicago

Dr. Mendoza is an Assistant Professor at the Pritzker School of Molecular Engineering at the University of Chicago. He has expertise in cancer research, bioinformatics, protein engineering, structural biology, and immunology. He received a bachelor's degree in biochemistry from San Francisco State University and a doctorate in molecular biophysics with an emphasis in computational and systems biology from The University of Texas Southwestern Medical Center. As a postdoctoral scholar at Stanford University, his honors include an NCI Career Development Award (K01) and prestigious fellowships from the Helen Hay Whitney Foundation and the Damon Runyon Cancer Research Foundation. He also received the Young Investigator Award from the International Cytokine and Interferon Society in 2019 and recently was named a 2023 Freeman Hrabowski Scholar by the Howard Hughes Medical Institute, a new program supporting outstanding early-career faculty who are committed to advancing diversity, equity, and inclusion in science.

Dr. Mendoza's research focuses on understanding basic principles of protein function relevant to human health and disease. Protein families of interest include the interferon (IFN) superfamily of cytokines, which are an essential part of the innate immune system, protecting against the spread of viral infections and cancerous growths. There are three families of IFNs, type I-III, each with distinct ligand-receptor systems. By applying a protein engineering approach, Dr. Mendoza's lab can rapidly evolve IFNs and visualize the three-dimensional shapes of interferon cytokines bound to their cellular receptors. When the sequence and structural information of the newly evolved interferon cytokines is combined with comprehensive biophysical and functional studies, the new insights into cytokine signaling create new opportunities for developing promising molecules for basic research and clinical use.

Patricio I. Meneses, Ph.D.—Associate Dean and Director, Honors Program, Fordham University, Rose Hill

Dr. Meneses received his graduate degree in 1999 from Cornell University Joan and Sanford Weill Graduate School in New York, NY. He continued his career as a postdoctoral fellow at Harvard Medical School (1999–2003) and the University of Pennsylvania School of Medicine (2003–2006).

In 2006, he opened his laboratory as an Assistant Professor studying the basic biology of human papillomavirus at Chicago Medical School/Rosalind Franklin University of Medicine and Science in North Chicago, IL. In 2011, he moved to Fordham University as an Associate Professor and has since risen to the rank of Professor (2023).

After being awarded tenure in 2016 in the department of Biological Sciences, he began saying "yes" to leadership opportunities and served as Department Associate Co-Chair (fall semester 2017), Associate Chair (spring and summer 2018), and Department Chair (2018–2021). Dr. Meneses then was asked to serve as Interim Associate Dean for Student Support and Academic Initiatives at Fordham College of Rose Hill (2022–present) and currently serves as Associate Dean and Director of the Honors Program at Fordham University, Rose Hill. He also serves Fordham as the National Collegiate Athletic Association Faculty Athletic Representative since 2017 and serves his faculty colleagues as American Association of University Professors advocacy chapter representative since 2016.

Speaker Bios

Yamilé Molina, Ph.D.—Associate Professor, University of Illinois Chicago, and Associate Director, University of Illinois Cancer Center

Dr. Molina is an Associate Professor in the Division of Community Health Sciences in the School of Public Health; Associate Director for Community Engaged Research for the Mile Square Health Center, one of the oldest federally qualified health centers and one of the few affiliated with an academic medical center; and Associate Director for Community Outreach & Engagement at the University of Illinois Cancer Center. Inequities in breast cancer have been the primary focus of Dr. Molina's efforts as a researcher, administrative leader, and community advocate, with a particular focus on racial/ethnic minorities, rural residents, and LGBTQ communities. Their NIH-funded work leverages an asset-based perspective to address adverse social determinants of health and barriers to cancer care, including costs and transportation, through enriched community-clinical partnerships and mobilization of participants' networks.

April Oh, Ph.D., M.P.H.—Senior Advisor, NCI

Dr. Oh is a Senior Advisor for Implementation Science and Health Equity on the Implementation Science Team in the Office of the Director in the Division of Cancer Control and Population Sciences at NCI. She leads efforts to advance the intersection of implementation science and health equity research. Dr. Oh provides scientific leadership for NCI's Implementation Science in Cancer Control Program, which supports the rapid development, testing, and refinement of innovative approaches to implement a range of evidence-based cancer control interventions. She also co-directs the Speeding Research Tested Interventions into Practice training program.

Dr. Oh previously served as a Program Director in the NCI Behavioral Research Program with research interests in multilevel health communication, implementation science, social determinants of health, neighborhood and policy effects on community health, obesity-related behaviors, and digital health technologies to promote behavior change and cancer prevention and control. She previously served as Senior Policy Advisor to the 19th U.S. Surgeon General on nutrition and obesity-related programs.

Dr. Oh holds a doctoral degree in public health (community health sciences) from the University of Illinois at Chicago, a master's degree in public health from the University of Michigan, and a bachelor's degree in public health from the University of North Carolina at Chapel Hill.

Elyse R. Park, Ph.D.—Professor, Massachusetts General Hospital (MGH)/Harvard Medical School, and Associate Director, MGH Cancer Center

Dr. Park is a Professor of Psychiatry and Medicine at MGH/Harvard Medical School. A clinical health psychologist and behavioral scientist, she founded the Health Promotion and Resiliency Intervention Research Program, an initiative through the Departments of Psychiatry and Medicine. She is Associate Director of Survivorship and Psychosocial Services for the MGH Cancer Center. The primary focus of her research is on the integration and implementation of behavioral interventions into clinical care, healthcare systems, and communities. Her research is dedicated to furthering the development and implementation of accessible digital interventions for all cancer patients and survivors. She co-chairs the ECOG-ACRIN Health Promotion Subcommittee within the Cancer Control and Survivorship Committee.

Dr. Park is a recipient of an NCI K24 to promote mentoring of clinician scientists to develop skills to conduct behavioral trials in cancer prevention, treatment, and survivorship. She is a primary mentor on NIH K development awards for physicians and psychologists and on three NCI Diversity Supplements. She is conducting two NCI-funded effectiveness-implementation trials integrating telehealth-delivered tobacco treatment into patients' care during cancer screening and at the time of a cancer diagnosis. Dr. Park also is conducting an NCI-funded R01 trial to assess the effectiveness of a synchronous vs asynchronous healthcare navigation intervention for cancer survivors as well as an NCI-funded U01 trial to enhance the racial and

ethnic diversity representation in cancer clinical trials. Additionally, she created an evidence-based Stress Management and Resiliency Training: Relaxation Response Resiliency Program (SMART-3RP) and developed a cadre of multidisciplinary investigators dedicated to resiliency clinical delivery and clinical research for vulnerable populations. She has adapted this resiliency treatment to diverse populations and settings and has conducted many trials assessing the SMART-3RP with clinicians (cancer interpreters, palliative care clinicians) and cancer survivors. She is conducting an R21 to assess the feasibility and acceptability of a survivor/caregiver resiliency intervention and exploring emotional well-being and healthcare utilization.

Manuel L. Penichet, M.D., Ph.D.—Professor, University of California, Los Angeles

Dr. Penichet holds an M.D. and a doctorate in biochemistry from the Havana Advanced Institute of Medical Sciences in Cuba. Early in his scientific career in Cuba, he worked on the development of a recombinant vaccine against parasites, which has been marketed and used internationally. Since 1996, he has worked at the University of California, Los Angeles (UCLA) in the fields of immunology, immunotherapy, antibody engineering, and nanotechnology, focusing on cancer and infectious diseases, and his research has both a basic science and a translational component. He is a tenured Professor in the Division of Surgical Oncology in the UCLA Department of Surgery with a joint appointment in the Department of Microbiology, Immunology, and Molecular Genetics. He is also a member of the AIDS Institute, the Molecular Biology Institute, the California NanoSystems Institute, and the Jonsson Comprehensive Cancer Center.

Dr. Penichet is author or co-author of more than 100 publications, including original papers, reviews, book chapters, and a book. Many of his senior-authored articles have been published in prestigious journals such as *Proceedings of the National Academy of Sciences*; *Nature Communications*; and *Journal of Immunology, Leukemia, and Blood*. He is also the lead inventor of four granted patents based on work conducted at UCLA. Additionally, Dr. Penichet has been invited to present his results at more than 80 seminars and conferences in 12 countries.

Dr. Penichet has received more than 25 grants, including 5 NIH/NCI R01 grants as principal investigator. He is also the recipient of awards such as the 2001 Amgen Award and the 2017 NIH/NCI Lifetime Achievement Award. He has been deeply involved in teaching and training undergraduate, graduate, and medical students, for which he has received numerous teaching awards. Importantly, he also has worked extensively as a consultant or reviewer of research projects for the biotech sector and for state and federal agencies in the United States and abroad.

Laritza M. Rodriguez, M.D., Ph.D.—Program Director, CRCHD, NCI

Dr. Rodriguez has been a Program Director in the NCI CRCHD since 2021. In this capacity, she manages NCI Exploratory/Developmental Grants (R21), Comprehensive Partnerships to Advance Cancer Health Equity (CPACHE U54) programs, and Minority Health and Health Disparities Coding and Data Processing addressing cancer health disparities and minority health.

Prior to joining CRCHD, Dr. Rodriguez served as a Staff Scientist at the Lister Hill Center at the National Library of Medicine, where she contributed her clinical expertise to projects including drug analytics and annotation of multiple collections of radiology reports, clinical narratives, and consumer questions for biomedical concepts and relations. She also has leveraged her clinical knowledge from 15 years as a practicing obstetrician/gynecologist to apply machine learning techniques and data analysis of the biomedical literature, with a focus on maternal morbidity and mortality.

Dr. Rodriguez earned her medical degree from the Universidad del Rosario, Hospital de San Jose School of Medicine in Bogota, Colombia and received her degree as an obstetrician/gynecologist from the same institution. She is a certified Maternal Fetal Medicine Specialist through postdoctoral training from John's Mercy Medical Center in St. Louis, MO. She received her Ph.D. from the University of Utah School of Medicine, where her dissertation focused on machine learning applied to labor and delivery data.

Speaker Bios

Charles R. Rogers, Ph.D., M.P.H., M.S., M.C.H.E.S.®—Associate Professor, Medical College of Wisconsin

Dr. Rogers currently serves as an Associate Professor of Epidemiology & Social Sciences in the Institute for Health & Equity at the Medical College of Wisconsin (MCW). He is also an MCW Cancer Center Research Scholar Endowed Chair and the inaugural Associate Director of Community Outreach & Engagement for MCW's Cancer Center. In addition to being the Founding Director of his Men's Health Inequities Research Lab, Dr. Rogers is also an Associate Member of the University of Michigan Mixed Methods Program. His career has permitted him to study, partner with, and/or be a vociferous advocate for various underserved and socially vulnerable groups, including community-dwelling older adult, African American, homeless, Somali, adolescent and young adult, Hispanic, rural, Indigenous, and sexual minority populations.

Dr. Rogers is committed to dismantling systems of oppression to ensure equitable health for all. His capabilities and potential have been recognized locally and nationally by the receipt of several competitive scholarships, grant awards, and fellowships. For instance, since 2018, he has been awarded over $3.3M from NCI, the Research Foundation of the American Society of Colon and Rectal Surgeons, Exact Sciences, the Medical College of Wisconsin, 5 For The Fight, and the V Foundation for Cancer Research for his community-engaged, mixed-methods research aiming to eradicate inequalities in both colorectal cancer (CRC) screening completion among African American men and early-onset CRC among individuals younger than the previously recommended CRC screening age of 50.

Fulera Salami, M.P.H.—Health Specialist, CRCHD, NCI

Ms. Salami has been a Health Specialist in the Diversity Training Branch of NCI's CRCHD since January 2023. In this role, she contributes to CRCHD's programs promoting workforce diversity in cancer research and supports administrative and programmatic management of Diversity Supplements.

Prior to joining CRCHD, Ms. Salami served as a Public Health Analyst and a Project Officer with the Health Resources and Services Administration (HRSA), Maternal and Child Health Bureau, Office of Epidemiology and Research, Division of Research. At HRSA, she managed maternal and child health research grants and cooperative agreements aimed at improving the health and well-being of American mothers and their children and led numerous other programs. Her areas of interest include global health, quality of and access to healthcare, disease prevention, health disparities, and environmental health.

Ms. Salami is a Dr.P.H. student in the Global Health, Policy and Evaluation concentration at the Johns Hopkins Bloomberg School of Public Health. She received her M.P.H. from the University of Illinois, Springfield. She is also an Army veteran.

Whitney Barfield Steward, Ph.D.—Program Director, CRCHD, NCI

Dr. Steward has been a Program Director in the Integrated Networks Branch of NCI's CRCHD since August 2021. Before joining NCI, she completed a second postdoctoral fellowship at Emory University in the Behavioral Genetics of Addiction Laboratory coordinating a multisite, University-based study that examined both behavioral and genetic risks for substance use development in young adults. Prior to that, Dr. Steward worked as a Program Officer at the National Heart, Lung, and Blood Institute's Center for Translational Research and Implementation Science, supporting strategies to accelerate the integration of evidence-based genomic discoveries into clinical and public health settings. She entered the extramural workforce as an S&T Policy Fellow with the American Association for the Advancement of Science's Office of Research on Women's Health, where she helped manage the Office's minority health and health disparities research portfolio.

Speaker Bios

Dr. Steward completed her first postdoctoral fellowship at Children's National Health System, where she performed genetic profiling and developed fitness tracking applications for home monitoring in children with metabolic conditions. She received her Ph.D. in microbiology from Howard University.

Shahrooz Vahedi, Ph.D.—Program Director, CRCHD, NCI

Dr. Vahedi has been a Program Director in the Diversity Training Branch of NCI's CRCHD since September 2022. He primarily oversees and manages K01 and K22 career development awards. Prior to joining CRCHD, he was a Scientific Review Officer at the Immunology and Infectious Diseases A review branch at the Center for Scientific Review, organizing the Innate Immunity and Inflammation study section.

Dr. Vahedi received his Ph.D. in microbiology and immunology from Rosalind Franklin University of Medicine and Sciences and completed his post-Ph.D. training as an NCI intramural fellow. His primary research interest focused on defining the role of lymphocyte-specific tyrosine kinase in cancer development and progression.

Anil Wali, Ph.D.—Program Director, CRCHD, NCI

Dr. Wali has been a Program Director in the Integrated Networks Branch of NCI's CRCHD since 2009. In this role, he contributes to the grants management of CRCHD's Geographic Management of Cancer Health Disparities Program. He also provides technical and scientific expertise to the Comprehensive Partnerships to Advance Cancer Health Equity (CPACHE U54) program.

Prior to joining NCI, Dr. Wali served as Associate Professor in the Departments of Surgery and Pathology at the NCI-Designated Comprehensive Cancer Center Barbara Ann Karmanos Cancer Institute, Wayne State University in Detroit. While at Wayne State University, Dr. Wali served as principal investigator on a Veterans Administration Merit Review Award-funded project on the role of the ubiquitin-proteasome pathway in mesothelioma carcinogenesis. Dr. Wali conducted an NCI clinical trial on asbestos-exposed patient populations to determine their risk for developing lung cancer and mesothelioma using high-throughput genomics and proteomics technologies.

Dr. Wali received his B.S. and M.S. degrees from the University of Kashmir in Srinagar, India and earned his Ph.D. at the Postgraduate Institute in Chandigarh, India. He completed postdoctoral fellowships at the Institute for Environmental Medicine at the University of Pennsylvania, at the FELS Institute for Cancer Research at Temple University, and in the Department of Pathology at Thomas Jefferson University in Philadelphia, PA.

Tiffany Wallace, Ph.D.—Program Director, CRCHD, NCI

Dr. Wallace is a Program Director at NCI within CRCHD. In this role, she serves as the Lead for the Disparities and Equity Program and works to coordinate and strengthen NCI's overall cancer disparity research portfolio, encompassing basic, clinical, translational, and population-based research. Additionally, she oversees a portfolio of grant mechanisms promoting basic/translational cancer research and develops initiatives to stimulate research in underfunded areas.

Prior to joining CRCHD, Dr. Wallace was an Oncology Scientist at Human Genome Sciences, where she managed clinically relevant research programs and conducted preclinical development of promising cancer therapeutics.

Speaker Bios

Dr. Wallace received her Ph.D. in biomedical sciences from the University of Florida in Gainesville. She completed her postdoctoral training in the Laboratory of Human Carcinogenesis at NCI, where she conducted basic and translational research to identify biomarkers of aggressive prostate and breast cancer.

Clayton C. Yates, Ph.D.—John R. Lewis Professor of Pathology; Professor of Pathology, Oncology, and Urologic Oncology; Director, Translational Health Disparities and Global Health Equity Research, Sidney Kimmel Comprehensive Cancer Center, Johns Hopkins University School of Medicine

Dr. Yates is a recognized expert in health disparity research. He earned his Ph.D. from the University of Pittsburgh School of Medicine in molecular pathology, as well as a certificate of training in tissue engineering and regenerative medicine from the McGowan Institute of Regenerative Medicine. He went on to complete a postdoctoral fellowship at Emory University School of Medicine Department of Urology. After completing his postdoctoral training, Dr. Yates started as a tenure track Assistant Professor at Tuskegee University in the Department of Biology and Center for Cancer Research and was promoted through the ranks to Full Professor. He holds adjunct faculty positions at Clark Atlanta University Department of Biology and the Department of Pathology at the University of Alabama at Birmingham (UAB). Cell-MENTOR (an online resource from Cell Press and Cell Signaling Technology) has recognized him among the 100 most inspiring Black Scientists in America.

Dr. Yates's lab focuses on prostate and breast cancers, particularly in African Americans. His lab has established several cell-line-based models derived from African American patients used by many labs today to study molecular events that lead to prostate cancer development and metastasis. Additionally, Dr. Yates identified a subtype of breast cancer called quadruple negative breast cancer, which is more frequent in women of African Ancestry. This novel approach used genetic admixture analysis and identified a specific ancestry-associated immune-related signature in both breast and prostate cancers. Using this signature, Dr. Yates' lab was able to identify and develop a novel therapeutic that targets specific immune cells termed tumor-associated macrophages (TAMs) that contribute to increased tumor aggressiveness and therapeutic resistance. This novel therapy was licensed to Aurinia Pharma in October 2021 for clinical development and is poised to enter clinical trials.

Dr. Yates' lab has been continuously funded by NIH and the Department of Defense Congressionally Directed Medical Research Programs/Prostate Cancer Research Program (CDMRP/PCRP) over the last 15 years. He has trained 17 Ph.D. students, 28 master's level students, and 5 postdoctoral trainees during this time. He also has mentored 10+ junior faculty through the Morehouse School of Medicine/Tuskegee University/University of Alabama at Birmingham O'Neal Comprehensive Cancer Center (MSM/TU/UAB) Health Disparities Training Program for UAB and TU faculty. Currently, five of these mentees have received tenure at their respective universities.

Dr. Yates has spoken at many universities and conferences, including the inaugural NCI Health Disparities Conference and the American Association for Cancer Research (AACR) Plenary Session and numerous grand rounds and distinguished lecture series. He is featured on NCI's website for the NCI Annual Plan & Budget Proposal for Fiscal Year 2023, which was provided to Congress, President Joe Biden, and the public.

Dr. Yates currently serves on the UAB External Advisory Board for Training of Oncology Surgeons and the University of North Carolina NCI T32 External Advisory Board and is Chair of the University of Florida/University of Southern California/Florida A&M University U54 NCI Comprehensive Partnership to Advance Cancer Health Equity. He also has received numerous research honors and awards, authored over 100+ peer-reviewed publications, and is a member of the editorial board of *Scientific Reports*. He serves as Co-Director for the Trans-Atlantic Prostate Cancer Consortium, which focuses on understanding tumor biology in native African men in Nigeria. He Co-Chaired the AACR Conference in April 2022 and was the 2022–2023 Chair of the Minorities in Cancer Research Council within AACR, which serves 7,000+ minority cancer-focused scientists.

Speaker Bios

Dr. Yates is also principal investigator (PI) of the National Institute on Minority Health and Health Disparities Research Centers at Minority Institutions, site PI of a Clinical and Translational Science Award (jointly with the UAB hub institution), and PI of the NCI U54 Cancer Health Disparities grant with Morehouse School of Medicine and the University of Alabama at Birmingham.

Jelani C. Zarif, Ph.D., M.S.—Robert E. Meyerhoff Endowed Professor, Sidney Kimmel Comprehensive Cancer Center, Johns Hopkins University School of Medicine

Dr. Zarif is an Assistant Professor of Oncology and the Robert E. Meyerhoff Endowed Professor at the Johns Hopkins University School of Medicine Sidney Kimmel Comprehensive Cancer Center. Dr. Zarif is also a member of the Bloomberg-Kimmel Institute for Cancer Immunotherapy. The Zarif laboratory studies molecular mechanisms by immune cells within the tumor microenvironment that ultimately promote tumor growth, therapeutic resistance, and metastasis. The laboratory also focuses on discovering and investigating new biomarkers that may be expressed on myeloid cells known as macrophages that could predict clinical response to standard-of-care treatments for prostate cancer. Dr. Zarif has mentored countless underrepresented trainees at the high school, undergraduate, and graduate levels, and has mentored six graduate students, one M.D./Ph.D. student, and several postdoctoral fellows.

A Chicago native, Dr. Zarif obtained both his B.S. and M.S. degrees from Jackson State University. Upon completion, he earned his Ph.D. in cell and molecular biology from Michigan State University. He then completed two postdoctoral fellowships at the Johns Hopkins University School of Medicine. He has been the recipient of several research awards, including the Prostate Cancer Foundation's Young Investigator Award, the Patrick C. Walsh Prostate Cancer Research Fund, Allegheny Health Network Fund, Department of Defense Translational Research Award, a National Institute of General Medical Sciences T32, and the NCI K22 Career Transition Award (K22

Flash Talk Poster Presentation List

Flash Talk Poster Presentation List

1 Correlates of HPV Test History and Acceptability of HPV Self-Sampling Among Black Women
Adebola Adegboyega *(Early-Stage Investigator)*

2 Rapid Detection of Mutations in CSF-Cftna With the Genexus Integrated Sequencer (K08)
Leomar Ballester *(Early-Stage Investigator)*

29 Preclinical Efficacy Evaluation of Hybrid Oncolytic VSV-MORG in Acute Myeloid Leukemia
Oumar Barro *(Postdoctoral Fellow)*

30 Vagal-Cd8+ T Cell Neuroimmune Axis Modulates Liver Cancer
Kylynda Bauer *(Postdoctoral Fellow)*

3 Ablation of Tumor Specific IGFBP-3 Attenuates PDAC-Related Skeletal Muscle Wasting via Downregulation of the Ubiquitin Proteasome Pathway
Calvin Cole *(Early-Stage Investigator)*

33 Pan-Viral Serology Uncovers Distinct Virome Patterns Among Hepatocellular Carcinoma and Control Populations
Whitney Do *(Postdoctoral Fellow)*

4 Facilitators and Barriers to Communication About Cancer Genetic Testing in African American Families (K01)
Katrina Ellis *(Early-Stage Investigator)*

25 Characteristics of Extra-Endocrine Features in a Cohort of Patients With MEN2B Seen at the NIH
Mahider Enyew *(Postbaccalaureate/Graduate)*

5 Targeting CTPS1 and Nucleotide Metabolism as a Novel Therapeutic Approach in MYC-Amplified Medulloblastoma
Myron Evans *(Early-Stage Investigator)*

34 HIV-1 Antisense RNA Is Detected in Infected Cells In Vivo
Toluleke Famuyiwa *(Postdoctoral Fellow)*

Flash Talk Poster Presentation List

35 Understanding the Role of Immune Infiltrate Following Intense Neoadjuvant Androgen Deprivation Therapy in Locally Advanced Prostate Cancer
John Fenimore *(Postdoctoral Fellow)*

6 The Role of Cholecystokinin-B Receptor Expression in Stem Cell Activation During Liver Injury (K01)
Martha Gay *(Early-Stage Investigator)*

7 Influence of Social Determinants of Health on Pancreatic Neuroendocrine Tumor Stage at Presentation
Andrea Gillis *(Early-Stage Investigator)*

8 Computational Modeling as a Clinical Decision Support Tool in Fanconi Anemia
Leonard Harris *(Early-Stage Investigator)*

9 Early Results From a Pilot Rideshare Intervention to Improve Colonoscopy Completion (K08)
Rachel Issaka *(Early-Stage Investigator)*

10 Describing Uptake and Results of Multigene Panel Testing, Family Risk Communication, and Risk-Reduction Behaviors of Racially/Ethnically Diverse Young Breast Cancer Survivors: Preliminary Survey Results
Tarsha Jones *(Early-Stage Investigator)*

26 Longitudinal Follow-Up and Outcomes of Pediatric and Adult Patients With SDH-Deficient GIST (iCURE)
Erika Kaschak *(Postbaccalaureate/Graduate)*

12 Epigenetic Control of Apoptosis Is Suppressed by Oncogenic MYC-Dependent Reprogramming
Daniel Liefwalker *(Early-Stage Investigator)*

28 Hinging on Success: Leveraging the Power of CAR T-Cell Therapy Through In-Silico Modeling of Hinge Length and Epitope Location
Justin Mirazee *(Postbaccalaureate/Graduate)*

15 Examining the Association of Cardiovascular Health Attainment With Cancer Screening Adherence in Community-Dwelling Black Women
Timiya Nolan *(Early-Stage Investigator)*

Flash Talk Poster Presentation List

16 Enhancing the Antitumor Activity of Anti-CD47 Antibodies Through FC Optimization
Juan Osorio *(Early-Stage Investigator)*

17 Red Blood Cell Polyunsaturated Fatty Acid Composition and Mortality Following Breast Cancer: Results From the Women's Healthy Eating and Living (WHEL) Study (K01)
Humberto Parada *(Early-Stage Investigator)*

19 ERK Activation: An Actionable Driver of Intrinsic and Acquired CDK4i/6i Resistance in Acral Melanoma
Vito Rebecca *(Early-Stage Investigator)*

22 HMGB1 Localization and Its Effect on the Immune Response in the Lung Tumor Microenvironment
Glenn Simmons, Jr. *(Early-Stage Investigator)*

39 The Relationship Between Social Determinants of Health and Neurocognitive and Mood-Related Symptoms Among the Primary Brain Tumor Population
Macy Stockdill *(Postdoctoral Fellow)*

Poster
Abstract List

Poster Abstract List

1 Correlates of HPV Test History and Acceptability of HPV Self-Sampling Among Black Women
Adebola Adegboyega *(Early-Stage Investigator)*

2 Rapid Detection of Mutations in CSF-Cftna With the Genexus Integrated Sequencer (K08)
Leomar Ballester *(Early-Stage Investigator)*

3 Ablation of Tumor Specific IGFBP-3 Attenuates PDAC-Related Skeletal Muscle Wasting via Downregulation of the Ubiquitin Proteasome Pathway
Calvin Cole *(Early-Stage Investigator)*

4 Facilitators and Barriers to Communication About Cancer Genetic Testing in African American Families (K01)
Katrina Ellis *(Early-Stage Investigator)*

5 Targeting CTPS1 and Nucleotide Metabolism as a Novel Therapeutic Approach in MYC-Amplified Medulloblastoma
Myron Evans *(Early-Stage Investigator)*

6 The Role of Cholecystokinin-B Receptor Expression in Stem Cell Activation During Liver Injury (K01)
Martha Gay *(Early-Stage Investigator)*

7 Influence of Social Determinants of Health on Pancreatic Neuroendocrine Tumor Stage at Presentation
Andrea Gillis *(Early-Stage Investigator)*

8 Computational Modeling as a Clinical Decision Support Tool in Fanconi Anemia
Leonard Harris *(Early-Stage Investigator)*

9 Early Results From a Pilot Rideshare Intervention to Improve Colonoscopy Completion (K08)
Rachel Issaka *(Early-Stage Investigator)*

10 Describing Uptake and Results of Multigene Panel Testing, Family Risk Communication, and Risk-Reduction Behaviors of Racially/Ethnically Diverse Young Breast Cancer Survivors: Preliminary Survey Results
Tarsha Jones *(Early-Stage Investigator)*

Poster Abstract List

11 Telomerase Reactivation Induces Progression of Mouse BrafV600E-Driven Thyroid Cancers and Triggers Non-Telomeric Effects
Inigo Landa *(Early-Stage Investigator)*

12 Epigenetic Control of Apoptosis Is Suppressed by Oncogenic MYC-Dependent Reprogramming
Daniel Liefwalker *(Early-Stage Investigator)*

13 Development and User Testing of a Website With Information for Cancer Patients Who Have Pre-Existing Autoimmune Disease and Are Considering Immune Checkpoint Inhibitors (ICIS)
Maria Lopez-Olivo *(Early-Stage Investigator)*

14 Physical Activity, Sedentary Behavior, and Social Cognitive Factors in Inactive Breast and Endometrial Cancer Survivors Using Ecological Momentary Assessment
Whitney Morelli *(Early-Stage Investigator)*

15 Examining the Association of Cardiovascular Health Attainment With Cancer Screening Adherence in Community-Dwelling Black Women
Timiya Nolan *(Early-Stage Investigator)*

16 Enhancing the Antitumor Activity of Anti-CD47 Antibodies Through FC Optimization
Juan Osorio *(Early-Stage Investigator)*

17 Red Blood Cell Polyunsaturated Fatty Acid Composition and Mortality Following Breast Cancer: Results From the Women's Healthy Eating and Living (WHEL) Study (K01)
Humberto Parada *(Early-Stage Investigator)*

18 Provider Perspectives, Cancer Patient, and Carer Supportive Needs in Mild Cognitive Impairment and Cancer
Katherine Ramos *(Early-Stage Investigator)*

19 ERK Activation: An Actionable Driver of Intrinsic and Acquired CDK4i/6i Resistance in Acral Melanoma
Vito Rebecca *(Early-Stage Investigator)*

Poster Abstract List

20 The Effect of Epigenetic Alterations on the 3D Epigenome and Replication Timing of Prostate Cancer
Suhn Rhie *(Early-Stage Investigator)*

21 Multiple Myeloma Progression Associated Long Non-Coding RNA 1 Binds to Chromobox 4 Protein in Melphalan Resistance Cells and Can Be Targeted With Locked Nucleic Acid Antisense Oligos
Jessica Silva-Fisher *(Early-Stage Investigator)*

22 HMGB1 Localization and Its Effect on the Immune Response in the Lung Tumor Microenvironment
Glenn Simmons, Jr. *(Early-Stage Investigator)*

23 The Human Connection: Oncologist Characteristics and Behaviors That Facilitate Therapeutic Bonding With Latino Patients With Advanced Cancer (K08)
Ana Tergas *(Early-Stage Investigator)*

24 Survival Outcomes in Older People Living With HIV and Cancer
Tishiya Carey *(Postbaccalaureate/Graduate)*

25 Characteristics of Extra-Endocrine Features in a Cohort of Patients With MEN2B Seen at the NIH
Mahider Enyew *(Postbaccalaureate/Graduate)*

26 Longitudinal Follow-Up and Outcomes of Pediatric and Adult Patients With SDH-Deficient GIST (iCURE)
Erika Kaschak *(Postbaccalaureate/Graduate)*

27 Neuroimmune Modulation of Hepatic Tumors by Cholinergic Agonists
Amran Nur *(Postbaccalaureate/Graduate)*

28 Hinging on Success: Leveraging the Power of CAR T-Cell Therapy Through In-Silico Modeling of Hinge Length and Epitope Location
Justin Mirazee *(Postbaccalaureate/Graduate)*

Poster Abstract List

29 Preclinical Efficacy Evaluation of Hybrid Oncolytic VSV-MORG in Acute Myeloid Leukemia
Oumar Barro *(Postdoctoral Fellow)*

30 Vagal-Cd8+ T Cell Neuroimmune Axis Modulates Liver Cancer
Kylynda Bauer *(Postdoctoral Fellow)*

31 Epigenetic Mechanisms in Oncogenic Genome Organization
Theodore Busby *(Postdoctoral Fellow)*

32 RNA-Seq Analysis of Endogenous Retroviral Elements in Tumor Cell Lines and Extracellular Vesicles
Stephanie Chidester *(Postdoctoral Fellow)*

33 Pan-Viral Serology Uncovers Distinct Virome Patterns Among Hepatocellular Carcinoma and Control Populations
Whitney Do *(Postdoctoral Fellow)*

34 HIV-1 Antisense RNA Is Detected in Infected Cells In Vivo
Toluleke Famuyiwa *(Postdoctoral Fellow)*

35 Understanding the Role of Immune Infiltrate Following Intense Neoadjuvant Androgen Deprivation Therapy in Locally Advanced Prostate Cancer
John Fenimore *(Postdoctoral Fellow)*

36 Efficacy of Tempol and Deferiprone in Combination With Renal Cell Carcinoma Current Treatments
Julia Medina Velazuez *(Postdoctoral Fellow)*

37 Discovery of Chemical Probes that Modulate the Association of E3 Ligase E6AP With the 26S Proteasome
Christine Muli *(Postdoctoral Fellow)*

Poster Abstract List

38 Single Cell Characterization of the Adjacent Tissues and Metastatic Microenvironment of Adrenocortical Carcinoma Reveals Profound Molecular and Cellular Reprogramming Consistent With Metastatic Progression and Disease Outcome
Mohd Omar Sikder *(Postdoctoral Fellow)*

39 The Relationship Between Social Determinants of Health and Neurocognitive and Mood-Related Symptoms Among the Primary Brain Tumor Population
Macy Stockdill *(Postdoctoral Fellow)*

40 Exploiting Metabolic Vulnerabilities in Neuroblastoma Through Pharmacological Inhibition of NAD Production to Impair Cell Survival, Energy Metabolism, and Tumor Growth
Sophia Varriano *(Postdoctoral Fellow)*

41 Persistent Disparities of Cervical Cancer Among American Indians/Alaska Natives: Are We Maximizing Prevention Tools? (K08)
Amanda Bruegl *(Postdoctoral Fellow)*

42 Loss of Glutamine Synthetase Exacerbates Myeloid Cell Induced Immunosuppression in β-Catenin-Mutated Hepatocellular Carcinomas
Evan Delgado *(Postdoctoral Fellow)*

43 Exploring HPV Vaccine Concerns and Communication Needs Among Parents Who Have not Initiated nor Completed the HPV Vaccine Series for Their Child (K01)
Jennifer Erves *(Postdoctoral Fellow)*

44 Insurance Type Drives Disparities in Stage at Diagnosis, Treatment, and Survival in Patients With Hepatocellular Carcinoma (1K08CA255413-01)
Patricia Jones *(Postdoctoral Fellow)*

45 Telehealth Utilization by Rural Older Cancer Survivors: A Qualitative Assessment by Rural Older Cancer Survivors, Caregivers, and Healthcare Professionals
Marquita Lewis–Thames *(Postdoctoral Fellow)*

46 High Frequency of NAPRT Silencing in Neuroblastoma and Rhabdomyosarcoma Confers Sensitivity to NAMPT Inhibitors
Juan Vasquez *(Postdoctoral Fellow)*

Early-Stage Abstracts

Early-Stage Abstracts

1: Correlates of HPV Test History and Acceptability of HPV Self-Sampling Among Black Women

Adebola, A.

College of Nursing, University of Kentucky, Lexington, KY

Introduction/Background: Cervical cancer screening rates among Black women (74.8%) are lower than the Healthy People 2030 target (84.3%). Unscreened and underscreened women have the greatest risk of developing invasive cervical cancer. HPV self-sampling is an alternative method that can potentially increase screening participation. To determine the acceptability of HPV self-sampling among Black [African American (AA) and sub-Saharan African immigrant (SAI)] women, information is needed regarding their HPV test history and willingness to use self-sampling. The purpose of this study was to examine factors associated with having had at least one HPV test and willingness to use HPV self-sampling among AA and SAI women.

Methods: AA and SAI women (n = 91) recruited from community settings completed a survey in a cross-sectional study. Data on demographics, HPV test history, HPV testing knowledge, and willingness to use a HPV self-sampling test were collected. Logistic regressions were performed to evaluate associations with having had a HPV test and willingness to use self-sampling.

Results: Participants mean age was 38.2 years (SD = 12.6) and 65% were SAIs. The majority (84%) reported having had at least one Pap test and (36%) had at least one HPV test. Sixty-seven percent were willing to have self-sampling. Younger age, higher education, and higher HPV testing knowledge were associated with having had a HPV test. Being uninsured and the likelihood of accepting a Pap test if recommended were associated with willingness to use self-sampling at home for an HPV test.

Conclusion: Future research should target women at higher risk of not being current with cancer screening due to older age, lower educational attainment, and significant gaps in HPV knowledge. Providers' recommendations are important tools to enlighten women about guidelines and options for cervical cancer screening. HPV self-sampling may be a promising screening strategy to decrease the cervical cancer burden experienced by disparate groups.

Funding: National Cancer Institute grant K01CA251487

2: Rapid Detection of Mutations in CSF-cfTNA With the Genexus Integrated Sequencer (K08)

Ballester, L.[1,2], Arjuna, S.[1], Shah, M.[1], Dono, A.[3], Pichardo, P.[3], Nunez-Rubiano, L.[4], Zhu, J-J.[3,5], Riascos-Castaneda, R.[4], Luthra, R.[6], Roy-Chowdhuri, S.[2], Duose, D.[1], Lang, F.[7], Esquenazi, Y.[3,5]

[1]Department of Translational Molecular Pathology, University of Texas MD Anderson Cancer Center, Houston, TX; [2]Department of Pathology, University of Texas MD Anderson Cancer Center, Houston, TX; [3]Vivian L. Smith Department of Neurosurgery, McGovern Medical School at UTHealth, Houston, TX; [4]Department of Diagnostic and Interventional Imaging, McGovern Medical School at UTHealth, Houston, TX; [5]Memorial Hermann Texas Medical Center, Houston, TX; [6]Department of Hematopathology, University of Texas MD Anderson Cancer Center, Houston, TX; [7]Department of Neurosurgery, University of Texas MD Anderson Cancer Center, Houston, TX

Introduction/Background: Brain metastases commonly arise in patients with lung and breast carcinomas and pose distinctive clinical challenges. Genomic alterations are fundamental for diagnosis and therapy selection in cancer patients. However, the turn-around-time (TAT) of standard next generation sequencing (NGS) assays is a limiting factor in the timely delivery of genomic information for clinical decision making. The GenexusTM Sequencer automates the NGS workflow delivering results in less than 24hrs. The Oncomine Precision Assay (OPA) evaluates somatic mutations, copy number variations and fusions in hot-spot regions of 50 cancer-related genes.

Methods: In this study, we evaluated genomic alterations in 46 cerebrospinal fluid (CSF) samples from patients with metastatic lung cancer (mLC) (N=32) and metastatic breast cancer (mBC) (N=14) to the brain. Cell-free total nucleic acids, (cfTNA) were extracted using QIAamp Circulating Nucleic Acid kit with cfDNA concentrations ranging from 0.1-11.2(ng/ul) and 0.5-6.6 (ng/ul) for mLC and mBC samples, respectively. Median base coverage was 37,913X and 33,576.5X for mLC and mBC samples, respectively, with cfDNA input ranging from 2-20ng.

Results: Mutations were detected in 15/32 (46.8%) mLC and 8/14 (57.1%) mBC CSF samples. Among the samples with no mutations detected, 18/23 (78.2%) had suboptimal DNA input (<20ng). The EGFR exon 19 deletion was detected in 4 samples from one patient, with increasing mutant allele fraction in CSF over time, highlighting the potential of CSF-cfDNA analysis for monitoring patients. Moreover, the EGFR T790M mutation was detected in a patient with prior EGFR inhibitor treatment. In addition, ESR1 D538G and ESR1::CCDC170 alterations, associated with endocrine therapy resistance, were detected in 2 mBC patients. The average TAT from cfTNA-to-results was <16hrs.

Conclusion: In summary, our results indicate that CSF-cfTNA analysis with the OPA in the Genexus instrument can provide clinically relevant information in patients with brain metastases with a short TAT.

Funding: This work was partly supported by the National Cancer Institute of the National Institutes of Health under Award Number K08CA24165.

3: Ablation of Tumor Specific IGFBP-3 Attenuates PDAC-Related Skeletal Muscle Wasting via Downregulation of the Ubiquitin Proteasome Pathway

Cole, C.[1,2,3], Sechrist, Z.[1,2,4]

[1]Division of Surgical Oncology, University of Rochester, Rochester, NY; [2]Center for Musculoskeletal Research, University of Rochester, Rochester, NY; [3]Wilmot Cancer Institute, University of Rochester, Rochester, NY; [4]Department of Immunology, University of Rochester, Rochester, NY

Introduction/Background: Pancreatic ductal adenocarcinoma (PDAC) is the fourth leading cause of cancer-related death. Skeletal muscle wasting (SMW), is a prognostic factor in PDAC, and is an independent predictor of infectious disease and postoperative mortality in resected patients. Research substantiates the role of increased levels of Insulin-like Growth Factor Binding Protein-3 (IGFBP-3) in PDAC-related SMW. Additionally, igfbp3 is significantly upregulated in PDAC tumor cells and its increase is associated with the activation of TGF-βRI signaling. Activation of TGF-βRI induces a cascade of events that lead to an increase in genes associated with the ubiquitin proteasome pathway (UPP), in skeletal muscle. In this study, we investigate the relationship between IGFBP-3, TGF-βRI, and PDAC-related SMW. We hypothesize that ablation of IGFBP-3 in PDAC tumor cells will attenuate SMW via downregulation of the UPP.

Methods: C57BL/6J female mice (6-8 weeks) (n=4 per group) were randomized to one of three groups 1) no tumor control (NTC), 2) KCKO (PDAC), or 3) KCKO (KO) (IGFBP-3 knockout). Tumor cells were injected orthotopically. Lean mass was monitored longitudinally using dual energy x-ray absorptiometry (DEXA). At sacrifice, quadriceps muscles were collected for transcriptional analysis and serum for ELISA.

Results: KO mice displayed significantly higher levels of lean mass when compared to PDAC mice ($p<0.01$). In addition, PDAC mice experienced a significantly lower survival ($p<0.01$). ELISA analysis showed significantly elevated IGFBP-3 in PDAC compared to NTC ($p<0.01$) and KO mice ($p<0.001$), while no difference was observed between NTC and KO animals. Transcriptionally, muscle from PDAC mice displayed significantly higher expression of igfbp3, and tgfbr1, and UPP associated genes fbxo32, and trim63 when compared to NTC and KO mice.

Conclusion: Depletion of tumor specific IGFBP-3 results in the attenuation of SMW via downregulation of UPP activation through TGF-βRI signaling.

Funding: NIH/NCI 1K01CA240533

4: Facilitators and Barriers to Communication About Cancer Genetic Testing in African American Families

Ellis, K.[1,2,3], Smith, M.[1,4], Wayas, F.[1], Contreras, A.[1], Vincent, C.[1], Perez, S.[1], Resnicow, K.[2], Stoffel, E.[5]

[1]School of Social Work, University of Michigan, Ann Arbor, MI; [2]School of Public Health, University of Michigan, Ann Arbor, MI; [3]Research Center for Group Dynamics, Institute for Social Research, Ann Arbor, MI; [4]Western Michigan University, Kalamazoo, MI; [5]School of Medicine, University of Michigan, Ann Arbor, MI

Introduction/Background: African Americans are less likely than other racial groups to engage in genetic testing for hereditary cancer syndromes, which can uncover genetic mutations that increase cancer risk. African Americans also have lower rates of cancer cascade testing, a process for providing genetic counseling and testing for relatives of people who are known carriers of specific cancer-related genetic mutations. Furthermore, African Americans report difficulties communicating about genetic testing results and family health history, and face barriers to healthy behavior engagement that can reduce cancer risk. This study aims to identify intrapersonal and interpersonal facilitators and barriers to African American family communication about cancer genetic testing and associated risk reduction behaviors.

Methods: Using a descriptive study design, this research conduct focus groups with African Americans who were the first in their biological families to have completed cancer genetic testing for Hereditary Breast and Ovarian Cancer (HBOC) syndrome and Lynch Syndrome (i.e., probands) and family members of these individuals. The intended sample size is 8 focus groups (N=8), with 4-6 people per group. Groups with probands (n=4) and family members (n-=4) will be separated by gender (male vs. female). In addition, the probands will be grouped by test results (positive vs. other results). Qualitative data will be coded and analyzed using a directed content analytic approach.

Results: Data collection and analysis of qualitative data is underway. Preliminary findings suggest that test result outcomes, family norms regarding cancer- and health-related communication, and gendered beliefs regarding usefulness of testing may be important drivers of communication about cancer genetic testing and risk reduction behaviors amongst families.

Conclusion: As cancer genetic testing become increasingly available, it is imperative to design family interventions to support communicaton about testing and cancer risk reduction that are accessible to, and supportive of underserved African American families.

Funding: This study was supported by the National Cancer Institute of the National Institutes of Health under Award Number K01CA255137. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

5: Targeting CTPS1 and Nucleotide Metabolism as a Novel Therapeutic Approach in MYC-Amplified Medulloblastoma (K22)

Evans, M.[1,2,3]; Hathaway, M.[1]; Jagana, H.[1]; Meechan, M.[1]; Slusher, B.[4]

[1]Ben Towne Center for Childhood Cancer Research, Seattle Children's Research Institute, Seattle, WA; [2]Human Biology Division, Fred Hutchinson Cancer Research Center, Seattle, WA; [3]Department of Pediatrics, School of Medicine, University of Washington, Seattle, WA; [4]Department of Neurology and Johns Hopkins Drug Discovery, Johns Hopkins School of Medicine, Baltimore, MD

Introduction/Background: Medulloblastoma (MB) is the most common pediatric CNS malignancy and comprises a biologically heterogeneous group of embryonal tumors of the cerebellum. MYC-amplified MB tumors are the deadliest of all subgroups with a <50% 5-year overall survival rate. High risk patients receive large doses of chemo- and radio- therapy (CT/RT) with most survivors experiencing debilitating neurological and cognitive sequelae making the identification of novel targets of the utmost importance. Recent whole-genome CRISPR screening identified the enzyme CTPS1 and others in the de novo pyrimidine biosynthesis pathway as a possible cancer-specific vulnerability in MYC-amplified MB. Our study tested targeted inhibitors of this pathway to identify rational combinations to test in pre-clinical models.

Methods: A stable of treatment-naïve, patient-derived models of MYC-amplified MB were used and treated with various de novo pyrimidine synthesis inhibitors alone and in combinations. Analysis of proliferation, cell survival, and downstream signaling were performed both in vitro and in vivo.

Results: Inhibition of CTPS1 (pharmacologically or with RNAi) led to a significant decrease in cell growth with only moderate increases in cell death. Screening of survival pathways revealed heightened DNA damage response (DDR) as a possible mechanism of cell survival. Combining DDR and CTPS1 inhibitors yielded significant tumor cytotoxicity both in vitro and in vivo. We further demonstrated robust synergy of DDRi with other clinically relevant pyrimidine metabolism inhibitors.

Conclusion: The results of this investigation confirm CTPS1 and the de novo pyrimidine metabolism pathway as targets in MYC-amplified MB and identifies rational combinations for treatment in patients.

Funding: Funded by K22CA262343, Unravel Pediatric Cancer Foundation, and generous donor funds to Evans Lab.

6: The Role of Cholecystokinin-B Receptor Expression in Stem Cell Activation During Liver Injury (K01)

Gay, M.[1], Drda, J.[2], Shivapurkar, N.[1], Chen, W.1, Smith, J.[1]

[1]Department of Medicine, Georgetown University, Washington, DC; [2]Department of Biology, Dickinson College, Carlisle, PA

Introduction/Background: Nonalcoholic steatohepatitis (NASH) is becoming a global epidemic in the United States. These conditions are an increasing cause of hepatocellular carcinoma (HCC). Cholecystokinin (CCK) receptors have also been identified in tissue fibroblasts. We believe that HCC may arise from liver progenitor cells or stem cells. Examining fibrosis and the role of CCK-B receptor expression after liver injury using 3,5-diethoxycarbonyl-1,4-dihydrocollidine (DDC) diet inflammatory pressure in CCK-B receptor+ cells will be evaluated. Our hypothesis states that if stem cells with CCK-B receptor expression are progenitors of tumor formation, the population of these cells is expected to increase with time in the livers during injury and may decrease following proglumide treatment.

Methods: Four-week diet to induce liver injury using four male cohorts of C57BL/6, with ten mice per group. Control group with regular chow and untreated water, DDC and untreated water, proglumide with regular chow, and a combination group of DDC and proglumide. Proglumide-supplemented drinking water for both cohorts were used to sustain the blockade of CCK-B receptors. Blood was analyzed for liver injury markers, liver tissues were collected for histological evaluation, and livers were enriched for CD133+ hepatic stem cell markers for identification of cancerous tissue.

Results: The DDC diet elevated liver injury markers compared to the control mice. Proglumide-treated DDC mice decreased liver injury and proglumide-alone group significantly reduced the expression of those markers. Flow data confirmed that the CD 133+ liver stem cells express the CCK-B receptors, which increased during liver injury, and the DDC cohort significantly increased fibrotic tissue. This confirmed that the DDC diet was successful and treatment with proglumide was more efficacious alone and in the combination group.

Conclusion: The data highlights translational potential of proglumide in this nonclinical study by improving hepatic histology and decreasing fibrosis in mice with liver injury.

Funding: NCI Award K01CA255572

7: Influence of Social Determinants of Health on Pancreatic Neuroendocrine Tumor Stage at Presentation (PACHE U54 Early Surgeon Scientist Program Administrative Supplement)

Gillis, A., Herring B., Guenter R., Chen, W., Chen D., Chen, H., Rose, B., Bhatia, S., Manne, U.

University of Alabama at Birmingham, Birmingham, AL

Introduction/Background: Tumor size and lymph node involvement (stage) have been utilized to prognosticate pancreatic neuroendocrine tumors (PNETs) but the influence of race and social vulnerability on these factors remains unexplored.

Methods: We reviewed records of all patients who underwent surgical resection for PNET from 2006-2022 at a single NCI cancer center in the Deep South. Patient demographics including self-reported race, tumor characteristics, and contextual level social determinants of health (SDOH) were analyzed. SDOHs were determined using publically available census data from geocoding patient billing addresses at the time of surgery. Large tumor size (≥2cm) and lymph node involvement (LNI) at presentation were the primary outcomes.

Results: A total of 179 patients were included. The median age at diagnosis was 60y, 52% were female and 30% were African American (AA). Compared to their White counterparts, AA patients were younger (median age 57y vs 60y, $p<0.01$) and more female (69% vs 45%, $p<0.01$). Overall, 121 patients presented with larger tumors. AA patients were more likely to have larger tumors (4.1 vs 3.1cm, $p<0.01$). There were no differences in SDOH between those with and without large tumors at presentation. Overall, 35 patients presented with LNI. AA patients were not more likely to present with LNI ($p=0.83$). Those with LNI lived in neighborhoods characterized by less poverty (12% vs 16%, $p=0.05$), similar median income ($53K vs $48K, $p=0.32$), higher educational attainment (92% vs 87% $p=0.04$), and lower uninsurance rates (14% vs 17% $p=0.03$). There was no difference in median Social Vulnerability Index or Area Deprivation Index ($p>0.05$)

Conclusion: In our cohort, there were differences in SDOH between those with more aggressive tumor characteristics of LNI but not larger size being associated with less vulnerable neighborhoods. This may indicate a protective factor due to surgeon access or referral bias indicating a need for targeted outreach.

Funding: Early Surgeon Scientist Program Administrative Supplement: 3U54CA118948-17S1

8: Computational Modeling as a Clinical Decision Support Tool in Fanconi Anemia (K22)

Harris, L.[1], Domínguez-Hüttinger, E.[2], Rodríguez, A.[3,4], Velleuer, E.[5,6], Carlberg, C.[7,8]

[1]Department of Biomedical Engineering, University of Arkansas, Fayetteville, AR; [2]Departamento de Biología Molecular y Biotecnología, Instituto de Investigaciones Biomédicas, Universidad Nacional Autónoma de México, Ciudad de México 04510, México; [3]Departamento de Medicina Genómica y Toxicología Ambiental, Instituto de Investigaciones Biomédicas, Universidad Nacional Autónoma de México, Ciudad de México 04510, México; [4]Instituto Nacional de Pediatría, Ciudad de México 04510, México; [5]Department of Cytopathology, Heinrich Heine University, 40225 Düsseldorf, Germany; [6]Center for Child and Adolescent Health, HELIOS Klinikum, 47805 Krefeld, Germany; [7]Institute of Animal Reproduction and Food Research, Polish Academy of Sciences, 10-748 Olsztyn, Poland; [8]Institute of Biomedicine, School of Medicine, University of Eastern Finland, 70211 Kuopio, Finland

Introduction/Background: Fanconi anemia (FA) is the most common inherited bone marrow failure syndrome, caused by inherited variants of the genome that dysregulate the cell's DNA repair pathways. Individuals with FA often develop aplastic anemia and head and neck squamous cell carcinoma (SCC) in the oral cavity. However, very little is known about how SCC develops in patients with FA due to a lack of good animal and cell line models.

Methods: We report on our early efforts to build a detailed computational model (aka, a "Digital Twin") integrating three important biological processes in FA: cell-cell interactions in the oral microbiome, DNA damage sensing and repair, and multi-step tumorigenesis. Each submodel is being built based on known FA biology, public computational model databases, and expert clinical knowledge from decades of experience treating FA patients.

Results: We have constructed initial versions of all three submodels. Each submodel will be calibrated and tested against multi-omic experimental data, including tissue transcriptomics, genomics, proteomics, and metagenomics for detection of pathogens inhabiting the tumor, and merged into a "master model." In silico interventions (e.g., vitamin D supplementation, change in diet) will then be performed on the master model by adjusting input conditions and/or parameter values to predict patient responses.

Conclusion: Computational models of FA have the potential to revolutionize FA patient care by providing a life-long tool for clinical decision-making regarding treatments and disease prevention. Although still in the early stages, this project is an important step toward the development of FA Digital Twins.

Funding: This work was supported by an NIH/NCI Transition Career Development Award to Promote Diversity (K22-CA237857).

9: Early Results From a Pilot Rideshare Intervention to Improve Colonoscopy Completion (K08)

Issaka, R.[1,2,3], Bell-Brown, A.[1], Hopkins, T.[1], Balmadrid, B.[4], Chew, L.5, Hirayama, L.[4], Kimura, A.[1], Riveira, M.[4], Strate, L.[3,4], Weiner, B.[6,7]

[1]Hutchinson Institute for Cancer Outcomes Research, Fred Hutchinson Cancer Research Center, Seattle, WA; [2]Public Health Sciences and Clinical Research Divisions, Fred Hutchinson Cancer Research Center, Seattle, WA; [3]Division of Gastroenterology, University of Washington School of Medicine, Seattle, WA; [4]Division of Gastroenterology, Harborview Medical Center, Seattle, WA; [5]Division of Internal Medicine, Harborview Medical Center, Seattle, WA; [6]Department of Global Health, University of Washington, Seattle, WA; [7]Department of Health Services, University of Washington, Seattle, WA

Introduction/Background: Lack of transportation is a frequently cited barrier to colonoscopy completion in colorectal cancer (CRC) screening. Rideshare non-emergency medical transportation (NEMT), has not been explored in settings that administer sedation. Rideshare NEMT services are scheduled by the healthcare team and billed directly to the organization. Our pilot study aimed to evaluate the safety and acceptability of rideshare NEMT for patients who required sedation for CRC screening at Harborview Medical Center (HMC), a large, urban, safety-net healthcare system

Methods: Eligible patients were identified by endoscopy staff during pre-procedure phone calls and referred to the study team, who contacted patients to obtain informed consent. On the day of the colonoscopy, the discharging nurse requested a ride home for the patient via the rideshare NEMT platform (Lyft Concierge). One to two weeks post-colonoscopy, patients completed a semi-structured interview that detailed their experience.

Results: Between February 2022 and May 2023, 28 patients consented to participate; 24 rides have been completed, 2 are forthcoming, 1 patient was excluded after consenting due to a motorized wheelchair that did not fit in the rideshare vehicle, and 1 patient opted for alternative NEMT on the day of the procedure. Patient demographics are summarized in Table 1. All participants to date (n=24), shared that they would use the rideshare program again for care that required sedation. Patients highlighted benefits including short wait-times, convenience, no costs, and not having to delay their colonoscopy. Recruitment is ongoing.

Conclusion: Our pilot study suggests that rideshare NEMT is a safe, reliable, and acceptable transportation option after receiving procedural sedation. To improve CRC screening, including follow-up of non-invasive tests, interventions that address identified barriers are needed. Rideshare NEMT program could be a cost-effective and potentially scalable solution to address a persistent challenge in CRC screening

Funding: Dr. Issaka reported receiving grants from National Institutes of Health / National Cancer Institute award number K08 CA241296 and P50CA244432 for the conduct of this study.

10: Describing Uptake and Results of Multigene Panel Testing, Family Risk Communication, and Risk Reduction Behaviors of Racially/Ethnically Diverse Young Breast Cancer Survivors: Preliminary Survey Results (NCI K01)

Jones, T

Christine E. Lynn College of Nursing, Florida Atlantic University, Boca Raton, FL

Introduction/Background: Breast Cancer (BC) in young women diagnosed prior to age 50 years is an urgent public health issue, especially for young Black and Hispanic women who suffer disproportionately from breast cancer health disparities. We sought to gain contextual understanding of the experiences and needs of diverse young breast cancer survivors (YBCS).

Methods: Eligible participants were ages 18 to 49 years at the time of their BC diagnosis and within 10 years of diagnosis. Participants were recruited from cancer centers, completed a survey online or via telephone, including open-ended items. Diagnosis, treatment data, and genetic test results were abstracted from medical records.

Results: A total of 30 young breast cancer survivors (YBCS) were included in this preliminary analysis (43.3% White, 33.3% Hispanic, 13.4% Bi-racial, 6.7% Black, 3.3% Asian). Bilateral (double) mastectomy was the most common surgery reported by young survivors (70%) and (13%) of women reported having a hysterectomy. The majority of YBCS reported completing multigene panel testing at the time of their diagnosis (97%); of whom, 5 women (17%) had pathogenic variants in BRCA1, BRCA2, CHEK2, CDH1, and PTEN genes. Provider recommendation was the most common reason for completing genetic testing (87%). The majority of the women (90%) reported communicating their genetic test results to family members, such as their moms, sisters, and daughters. Through open-ended survey items, participants expressed their needs.

Conclusion: Our preliminary data shows the need for targeted efforts to increase the diversity of our sample, particularly in recruiting more young black survivors. Data suggests that young survivors will benefit from an intervention that is tailored based on young age at diagnosis and provides decision support for genetic testing and family risk communication. Additionally, young survivors are in need of peer-support and resources to manage mental health needs such as fear of recurrence and depression.

Funding: NCI 1K01CA241393-01A1

11: Telomerase Reactivation Induces Progression of Mouse BrafV600E-Driven Thyroid Cancers and Triggers Non-Telomeric Effects (K22)

Landa, I.[1,2,3], Thornton, C.[1,2], Xu, B.[3], Haase, J.[1,2], Krishnamoorthy, G.[4], Hao, J.[1,2], Knauf, J.[5], Herbert, Z.[6], Blasco, M.[7], Ghossein, R.[3], Fagin, J.[4,8]

[1]Division of Endocrinology, Diabetes and Hypertension, Brigham and Women's Hospital, Boston, MA; [2]Harvard Medical School, Boston, MA; [3]Department of Pathology, Memorial Sloan Kettering Cancer Center, New York, NY; [4]Human Oncology and Pathogenesis Program, Memorial Sloan Kettering Cancer Center, New York, NY; [5]Lerner Research Institute, Cleveland Clinic, Cleveland, OH; [6]Molecular Biology Core Facilities, Dana-Farber Cancer Institute, Boston, MA; [7]Telomeres and Telomerase Group, Molecular Oncology Program, Spanish National Cancer Centre, Madrid, Spain; [8]Department of Medicine, Memorial Sloan Kettering Cancer Center, New York, NY

Introduction/Background: Mutations in the promoter of the telomerase reverse transcriptase (TERT) gene are the paradigm of a cross-cancer alteration in a non-coding region. TERT promoter mutations (TPMs) are biomarkers of poor prognosis in several tumors, including thyroid cancers. TPMs enhance TERT transcription, which is otherwise silenced in adult tissues, thus reactivating a bona fide oncoprotein.

Methods: To study TERT deregulation and its downstream consequences, we generated a Tert mutant promoter mouse model via CRISPR/Cas9 engineering of the murine equivalent locus (Tert-123C>T) and crossed it with thyroid-specific BrafV600E-mutant mice. We also employed an alternative model of Tert overexpression (K5-Tert).

Results: Whereas all BrafV600E animals developed well-differentiated papillary thyroid tumors, 29% and 36% of BrafV600E+Tert-123C>T and BrafV600E+K5-Tert mice progressed to poorly differentiated thyroid cancers at week 20, respectively. Tert-reactivated tumors showed increased mitosis and necrosis in areas of solid growth, and older animals from these cohorts displayed anaplastic-like features, i.e., spindle cells and macrophage infiltration. Murine Tert promoter mutation increased Tert transcription in vitro and in vivo, but temporal and intra-tumoral heterogeneity was observed. RNA-sequencing of thyroid tumor cells showed that processes other than the canonical Tert-mediated telomere maintenance role operate in these specimens. Pathway analysis showed that MAPK and PI3K/AKT signaling, as well as processes not previously associated with this tumor etiology, involving cytokine and chemokine signaling, were overactivated.

Conclusion: These models constitute useful pre-clinical tools to understand the cell-autonomous and microenvironment-related consequences of Tert-mediated progression in advanced thyroid cancers and other aggressive tumors carrying TPMs.

Funding: This research was funded by the National Institutes of Health grants 1R01CA249663-02 and 5R01CA050706-31 (to J.A.F.), the National Cancer Institute Career Transition Award, grant number 1K22CA230381–01A1 (to I.L.) and the Brigham and Women's Hospital, Department of Medicine, Innovation Evergreen Fund (to I.L.).

12: Epigenetic Control of Apoptosis Is Suppressed by Oncogenic MYC-Dependent Reprogramming (K01)

Liefwalker, D.[1], Anchang, B.[2], Sears, R.[1,3]

[1]Department of Molecular and Medical Genetics and Knight Cancer Institute, Oregon Health and Science University, Portland, OR; [2]Biostatistics and Computational Biology Branch, National Institute of Environmental Health Sciences, National Institutes of Health, Bethesda, MD; [3]Brenden-Colson Center for Pancreatic Care, Oregon Health and Science University, Portland, OR

Introduction/Background: The proto-oncogene c-MYC (MYC) is a transcription factor that regulates much of the genome and is deregulated in most cancers. Using transgenic models of MYC-driven lymphoid malignancies we discovered the JmjC containing class of transcriptional repressive histone demethylases are suppressed by MYC-dependent cells. This suppression confers evasion of cell death, enhanced tumor progression, and shutdown of a feedback loop that regulates MYC expression. We are currently testing therapeutic strategies to restore the epigenetic functions that oppose MYC-dependent directives in malignant cells.

Methods: We employed a nested-effects model (NEM) that infers hierarchical relationships anchored to master transcription factors that govern underlying features of lymphoid malignancies. CRISPR based KO of critical nodes reveal an epigenetic switch that controls apoptosis. ChIP-seq experiments uncovered multiple select targets of the epigenetic switch

Results: We detail an epigenetic apoptosis feedback loop that is disrupted in MYC-dependent oncogenic settings and demonstrate the clinical relevancy in patient samples.

Conclusion: To date there are no compelling MYC-dependent target genes that have been identified that mediate these pro-apoptotic responses. Our discovery and characterization of a mechanism for how MYC-dependent cells avoid cell death significantly advances the field and may ultimately lead to new therapeutic alternatives.

Funding: Burroughs Wellcome Fund Postdoctoral Enrichment Award, Tumor Biology Training grant (Stanford University (NCI) 5T32CA009151-38), Research Supplement Award (NCI), 3U01CA188383-03S1 (NIH), K01 CA234453-04S1, Collins Medical Trust, K01-CA234453-05 (NCI)

13: Development and User Testing of a Website With Information for Cancer Patients Who Have Pre-Existing Autoimmune Disease and Are Considering Immune Checkpoint Inhibitors (ICIs) (K08)

Lopez-Olivo, M.[1], Duhon, G.[1], Erck, M.[2], Calabrese, C.[3], Altan, M.[4], Tawbi, H.[5], Meara, A.[6], Bingham, C.[7], Diab, A.[5], Leal, V.[1], Suarez-Almazor, M.[1,8], Volk, R.[1]

[1]Department of Health Services Research, University of Texas MD Anderson Cancer Center, Houston, TX; [2]Department of Palliative Care & Rehabilitation Medicine, University of Texas MD Anderson Cancer Center, Houston, TX; [3]Division of Rheumatology, Department of Medicine, Johns Hopkins University, Baltimore, MD; [4]Department of Thoracic-Head & Neck Medical Oncology, University of Texas MD Anderson Cancer Center, Houston, TX; [5]Department of Melanoma Medical Oncology, University of Texas MD Anderson Cancer Center, Houston, TX; [6]Division of Internal Medicine, University of Texas MD Anderson Cancer Center, Houston, TX; [7]Department of Rheumatologic and Immunologic Disease, Cleveland Clinic Foundation, Cleveland Heights, OH; [8]Section of Rheumatology and Clinical Immunology, Department of General Internal Medicine, University of Texas MD Anderson Cancer Center, Houston, TX

Introduction/Background: Patients considering ICIs for their cancer who also have an underlying autoimmune disease need to be informed about the benefits and potential for severe immune-related adverse events and flares of the autoimmune condition. We developed and tested the usability and acceptability of an educational website designed to facilitate patient-doctor discussions.

Methods: This study consisted of 3 phases: content creation, development of website, and user testing. The list of learning topics, images that were relevant to the educational content, and website architecture, flow, and requirements were developed and iteratively reviewed by members of a community scientist program, a patient advisory group, and content experts. After developing the prototype website, a usability test using the Suitability Assessment Measure was conducted in cancer patients with diagnosis of an underlying autoimmune disease who had already received ICIs.

Results: The website components included a home page with general information about ICIs; learning modules with information about benefits, possible side effects, and what patients should expect before, during, and after treatment with ICIs. Participants had a median age of 60 years old. The median Suitability Assessment score was 75 (scale of 0-100). Participants agreed that the website was acceptable, balanced (benefits/harms ratio), and helpful. However, they were neutral about the length of information with users preferring more on ICIs infusions and probabilities of adverse events. Patients appreciated the medication overview and the glossary of terms used within the website. Recommendations for improvement mostly revolved around the navigation, accessibility, and adding functionalities.

Conclusion: The newly developed website was acceptable for patients, and it has the potential to become a supporting tool to facilitate patient-doctor discussion regarding ICIs. A pilot study testing the feasibility of website use in clinical settings and its effectiveness in decreasing decisional conflict and improving knowledge regarding ICI is underway.

Funding: This study was supported by the National Cancer Institute (Project number: K08CA237619), The University of Texas MD Anderson's Cancer Center Support Grant funded from NIH/NCI under award number P30CA016672 (using the Shared Decision Making Core), and the Rheumatology Research Foundation.

14: Physical Activity, Sedentary Behavior, and Social Cognitive Factors in Inactive Breast and Endometrial Cancer Survivors Using Ecological Momentary Assessment

Morelli, W.[1], Cadmus-Bertram, L.[2], Siddique, J.[3], Solk, P.[3], Hodgson, V.[2], Phillips, S.[3]

[1]Medical College of Wisconsin, Milwaukee, WI; [2]University of Wisconsin, Madison, WI; [3]Northwestern University, Chicago, IL

Introduction/Background: The Purpose of this study was to examine same-day relationships between social cognitive theory (SCT) factors and activity behaviors among insufficiently active breast and endometrial cancer survivors.

Methods: Survivors (N=127, Mage=56.3±9.6 years, MBMI=3.8±7.9 kg/m2) wore an accelerometer to assess daily activity (SB, light intensity physical activity (LPA), and moderate-to-vigorous physical activity (MVPA)) and completed ecological momentary assessment prompts in the morning to assess current day's SCT factors (exercise self-efficacy, function and psychological outcome expectations (OE) for exercise, exercise plans, and social support for exercise) for 7 consecutive days as part of baseline assessments for a physical activity intervention. Self-efficacy was on a 100-pt scale in increments of 10. All other SCT factors were on a 5-pt scale. Separate mixed-effects models assessed the between-person (BP) and within-person (WP) associations between each daily SCT factor and minutes of SB, LPA, and MVPA that same day, controlling for relevant covariates (age, BMI, cancer type, wear time).

Results: Survivors were highly sedentary (516±96 minutes) and averaged 271±90 minutes of LPA and 17±10 minutes of MVPA per day. WP associations were significant for functional (β=-32±14 minutes, p=0.028) and psychological OE (β=-55±22 minutes, p=0.013) and SB such that when participants reported higher OE than their average on a given day, they spent less time sedentary that day. There was a significant BP association for social support (β=5±1 minutes, p=0.004) on MVPA such that those with greater social support spent more time in MVPA. There were significant BP associations for self-efficacy (β=-1 ±0.3 minutes, p=0.02), functional OE (β=-27±12 minutes, p=0.02), and psychological OE (β=-23±11 minutes, p=0.04) on SB such that those with higher SCT ratings spent less time sedentary.

Conclusion: Results indicate that daily SCT factors are important to consider when developing interventions to reduce SB for inactive breast and endometrial cancer survivors.

Funding: K01CA125414; R37CA225877S1

15: Examining the Association of Cardiovascular Health Attainment With Cancer Screening Adherence in Community-Dwelling Black Women (K08)

Nolan, T.[1,2], Zhao, S.[1], Williams, A.[1], Brock, G.1, King, M.[1], Ogonuwue, S.[1], Mekonen, H.[1], Park, S.[1], Metlock, F.3[,4], McKoy, A.[2], Davis, L.[2], Ewing, A.[1], Williams, K.[1]; Paskett, E.[1,2], Joseph, J.[1,3,4]

[1]The Ohio State University, Columbus, OH; [2]The Ohio State University Comprehensive Cancer Center, Columbus, OH; [3]Johns Hopkins University, Baltimore, MD; [4]Wexner Medical Center, The Ohio State University, Columbus, OH

Introduction/Background: Cancer and cardiovascular disease are leading causes of death in the United States and share modifiable risk factors. Increasing ideal cardiovascular health as American Heart Association's Life's Simple 7 metrics (LS7: blood pressure, glucose, cholesterol, body mass index, physical activity, and smoking) and cancer screening adherence are associated with reducing cancer mortality. While cancer screening disparities between Black and White women have narrowed, there are disparities in LS7 attainment by race. Here, we examined the association of LS7 attainment with screen detectable cancer (breast, cervical, and colorectal) adherence in Black women.

Methods: The Partnering in Negating Statistics (P.I.N.S.) for Black Women Initiative promotes health and wellness among Black women. Its community wellness events provide health education, screenings (e.g., breast, cervical, and cardiovascular health), and research participation opportunities. At P.I.N.S. events, LS7 metrics (score range 0-14) and self-reported cancer screening history were collected. The associations of LS7 with cancer screening adherence were examined using multinomial logistic regression adjusting for age, income, and education.

Results: In 2021 and 2022, 296 Black women participated in cardiovascular health screenings. They had a mean age of 50.1 years (SD 14.1); 66% employed; 88% insured; 69% with educational attainment ≥ college. The 166 participants with complete LS7 data had intermediate cardiovascular health with a median score of 8 [7-10]. In age adjusted models, a 1-unit increase in LS7 attainment was associated with a 45% higher odds of breast cancer screening adherence (OR 1.45, 95%CI: 1.03, 2.05; n=93), which was similar with full adjustment (OR 1.50, 95% CI: 0.95, 2.39; n=91). There was no association of LS7 attainment with cervical (OR 1.09, p=0.40; n=124) or colorectal cancer (OR 1.05, p=0.70; n=84) screening adherence.

Conclusion: In community-dwelling Black women, increasing LS7 attainment may increase odds of breast cancer screening adherence. Research with a larger sample is needed.

Funding: This work was supported by generous partnership from The African American Male Wellness Agency, funding from the Oncology Nursing Foundation and The Ohio State University, in-kind donations and volunteers from P.I.N.S. Initiative Partners, and time support from the National Institutes of Health (K08CA245208, TSN & K23DK117041, JJJ).

16: Enhancing the Antitumor Activity of Anti-CD47 Antibodies Through Fc Optimization (K08)

Osorio, J.[1,2], Smith, P.[2], Knorr, D.[1,2], Ravetch, J.[2]

[1]Memorial Sloan Kettering Cancer Center, New York, NY; [2]Rockefeller University, New York, NY

Introduction/Background: Antibodies targeting CD47 represent a promising strategy for cancer immunotherapy. However, ealy clinical trials have shown limited clinical benefit, suggesting that CD47 alone may not be sufficient for effective tumor contol. We hypothesize that interactions between the antibody Fc and Fc gamma receptors (FcgRs) contribute to the optimal in vivo antitumor activity of these novel therapies.

Methods: Mice humanized for both CD47 and SIRPalpha (SIRPa) were generated on the C57BL/6J background by CRISPR/Cas9-mediated gene-target strategy, these mice were subsequently backcrossed with mice humanized for FcgRs, ultimately generating a hCD47/hSIRPa/hFcgR mouse. Murine cancer cell lines (MC38 and B16) were humanized for CD47 using similar CRISPR/Cas9 targeting strategy. Blocking and non-blocking antibodies targeting mouse or human CD47 antibodies were generated with distinct Fc domains that have different binding to either murine or human activating FcgRs. Species match models were used to test antitumor activity of Fc variants for murine, chimeric, and humanized CD47 antibodies

Results: Through multiple species matched systems, we found that anti-CD47 antibodies displayed enhanced in vivo antitumor activity only when the antibody Fc was optimized to engage activating FcgRs. This effect depends on both macrophages and antigen specific T cells. In the humanized mouse model for CD47, SIRPa and FcgRs we recapitulate the on-target toxicity seen in clinical trials by humanized anti-CD47 antibodies. Furthermore, we observe enhanced in vivo antitumor activity, abscopal antitumor effect, and minimal on-target toxicity by local administration of our Fc-optimized anti-CD47 antibody as monotherapy or in combination with PD-1 blockade.

Conclusion: Fc optimization is critical for the development of effective CD47 therapies, providing a novel approach to enhance the antitumor activity of this promising immunotherapy.

Funding: K08CA266740 and MSK Gerstner Physician Scholars program to JCO, R01CA244327 and R35CA196620 to JVR, #UL1TR001866 from National Center for Advancing Translational Sciences

17: Red Blood Cell Polyunsaturated Fatty Acid Composition and Mortality Following Breast Cancer: Results From the Women's Healthy Eating and Living (WHEL) Study (K01)

Parada, H., Jr.[1], Wu, T.[1], Hoh, E.[1], Rock, C.[2], Martinez, M.[2]

[1]San Diego State University, San Diego, CA; [2]University of California, San Diego, San Diego, CA

Introduction/Background: Polyunsaturated fatty acids (PUFA) may play an important role in the prevention of breast cancer; however, few studies have examined whether PUFAs impact survival following breast cancer. Herein, we examined the associations between red blood cell (RBC) PUFA composition and breast cancer mortality.

Methods: This nested case-control study included 1,104 women from the Women's Healthy Eating and Living Study, a randomized controlled trial of a plant-based diet on breast cancer survival. Cases were women who died from any cause (n=290) from 1995-2006. Controls were women who were alive at the end of follow-up matched to cases on age at diagnosis, years since randomization, intervention group, and stage (n=814). RBC fatty acid composition (four n-3 and seven n-6 PUFAS) was measured in baseline blood samples using gas chromatography. We examined each PUFA individually as well as factor analysis-derived scores in association with all-cause and breast cancer-specific mortality using conditional logistic regression to estimate odds ratios (ORs) and 95% confidence intervals (CIs).

Results: In fully-adjusted models, all-cause mortality ORs were elevated among women with PUFAs >median (versus ≤median) for alpha-linolenic acid (ALA; OR=1.56; 95%CI=1.14-2.14) and for linolenic acid (LnA, OR=1.54; 95%CI=1.15-2.07), and breast cancer-specific mortality ORs were elevated for LnA (OR=1.72, 95%CI=1.24-2.38) and gamma-linolenic acid (GLA; OR=1.40; 95%CI=0.97-2.00). Factor 1 [arachidonic acid; adrenic acid; and docosapentaenoic acid] scores >median (versus ≤median) were associated with lower odds of all-cause (OR=0.72; 95%CI=0.53-0.98) and breast cancer-specific (OR=0.67; 95%CI=0.47-0.94) mortality, and Factor 4 [ALA and GLA] scores >median (versus ≤median) were associated with increased odds of breast cancer-specific mortality (OR=1.42; 95%CI=1.01-2.01). Additionally, 1-SD increase in Factor 4 scores were associated with ORs of 1.22 (95%CI=1.04-1.42) for all-cause mortality and 1.24 (95%CI=1.05-1.47) for breast cancer-specific mortality.

Conclusion: RBC PUFA composition profiles are associated with all-cause and breast cancer-specific mortality risk among women with breast cancer.

Funding: NCI K01 CA234317; NCI U54 CA132384 & U54 CA132379

18: Provider Perspectives, Cancer Patient, and Carer Supportive Needs in Mild Cognitive Impairment and Cancer (K08)

Ramos, K.

School of Medicine, Duke University, Durham, NC

Introduction/Background: Among adults over the age of 65 in the U.S., 60% will be diagnosed with cancer. 1 in 5 older adults will experience problems with memory, thinking, and impaired judgment. These symptoms, known as Mild Cognitive Impairment (MCI), affect an estimated of 20-40% of older adults at the time of their cancer diagnosis and prior to receipt of chemotherapy which may exacerbate cognitive decline. Older adults living with cancer and MCI run a serious risk of being functionally and psychologically disadvantaged. For caregivers, caring for a loved one with cancer and MCI is often an overwhelming and highly stressful undertaking. Caregiving for an older adult with cancer is associated with high burden and declines in emotional and physical health. The focus of this qualitative study (year one of the NCI K08) is to elicit contributors to distress (i.e., anxiety and depression symptoms, and poor quality of life) in the context of cancer and MCI. Independent interviews with patients, caregivers, and providers were performed.

Methods: We will recruit 20 dyads (i.e., 40 patients and their caregivers), and 5 providers (geriatricians and oncologists). These numbers are our baseline for what we expect is needed to reach thematic saturation. Data will be examined using rapid qualitative analysis.

Results: We have collected data from 5 providers and 17 dyads (in the last 7 months of year one of the NCI K08). Results are in progress. We will examine transcripts for emergent themes across areas of session content, intervention structure (i.e., dyadic sessions), intervention length (i.e., 6 sessions), and delivery modality (e.g., videoconference).

Conclusion: Semi-structured interviews will not necessarily capture the needs of all older adults with cancer and MCI and their caregivers. However, in-depth interviews will guide the selection and tailoring of coping and communication skills to develop a supportive care intervention in this unique population.

Funding: The project is funded by the National Cancer Institute K08CA258947-01.

19: ERK Activation: An Actionable Driver of Intrinsic and Acquired CDK4i/6i Resistance in Acral Melanoma

Rebecca, V. Jagirdar, K., Portuallo, M., Wilhide, M., Ahmed, W., Wei, M., Robertson, B., Bravo, J., Alicea, G., Aguh, C., Xiao, M., Godok, T., Fingerman, D., Brown, G., Herlyn, M., Guo, B., Toska, E., Zabransky, D., Wubbenhorst, B., Nathanson, K., Ji, H., Liu, Q.

Patients with advanced acral lentiginous melanoma (ALM) suffer worse outcomes relative to patients with other forms of cutaneous melanoma, and do not equally benefit from targeted-/immuno-therapies. CDK4/6 pathway gene alterations in >60% of ALMs have propelled CDK4/6 inhibitor (CDK4i/6i) trials; however, a mPFS of 2.2 months (NCT03454919) suggests resistance rapidly arises in most cases. The ALM therapy resistance landscape remains little explored, and here we report MAPK activation drives both intrinsic-/acquired-CDK4i/6i resistance. CDK4i/6i exerts a robust, yet transient cell cycle arrest followed by reignition of proliferation. Reverse-phase protein arrays identify ERK activation occurring a) within hours of CDK4i/6i in therapy naïve cells and b) in models with acquired CDK4i/6i resistance. Importantly, concurrent MEKi + CDK4i/6i or ERKi + CDK4i/6i ablates intrinsic/acquired-CDK4i/6i resistance via triggering elevated cell cycle arrest and cell death in 3D spheroids. Mechanistically, ERK activation induces cyclin D levels to reactivate cell cycle, which when silenced blunts both intrinsic-/acquired-CDK4i/6i resistance. Concurrent MEKi + CDK4i/6i of ALM PDX-bearing mice confers the greatest antitumor activity via the induction of a defective DNA repair and apoptotic program. In parallel, we studied the concordance between DNA copy number variations and protein levels of CDK4-pathways nodes, as only patients with CDK4 gain, CCND1 gain and/or CDKN2A loss were eligible for CDK4i/6i. Notable discordance between the genomic status and protein levels of CDK4-pathway nodes exists, implying enrollment onto CDK4i/6i trials may benefit from IHC validation of protein levels. Our data identifies MAPK activity critical in both intrinsic-/acquired-CDK4i/6i resistance via induced cyclin D1 levels and we hypothesize concurrent MEKi + CDK4i/6i may have therapeutic efficacy.

This work was funded by a generous startup fund given by the Johns Hopkins University Department of Biochemistry and Molecular Biology and K01CA245124-01.

20: The Effect of Epigenetic Alterations on the 3D Epigenome and Replication Timing of Prostate Cancer (Other)

Rhie, S., Cao, H., Gonzalez-Smith, L., Wu, Z., Lee, B., Yang, S.

Department of Biochemistry and Molecular Medicine and Norris Comprehensive Cancer, University of Southern California, Los Angeles, CA

Introduction/Background: Recent studies showed that epigenetic alterations in regulatory elements can drive carcinogenesis. Regulatory elements such as promoters, enhancers, and insulators interact each other to mediate molecular processes including transcription process. Among regulatory elements, the activity of enhancers is most closely linked to cell identity.

Methods: We profiled the three-dimensional epigenomes in normal prostate and prostate cancer cells using DNA methylation, chromatin immunoprecipitation with sequencing (ChIP-seq), and chromatin conformation capture techniques (Hi-C, Micro- C) to identify epigenetic alterations. We used the CRISPR/Cas9 system to target epigenetic alterations. We used Repli-seq to determine the effect of epigenetic alterations on replication timing. RNA-seq was performed to measure gene expression changes upon altering the activities of epigenetic alterations.

Results: We identified prostate cancer-specific epigenetic alterations located in regulatory elements. Thousands of enhancers are specifically activated in prostate cancer in comparison to normal prostate. We found topologically associating domains where the identified prostate cancer-specific enhancers reside. By performing Micro-C and RNA-seq, we investigated the effect of enhancer activity changes in chromosome organization and gene regulation of prostate cancer. We also found that replication timing is potentially affected by the change of prostate cancer-specific enhancers.

Conclusion: This study provides valuable insights on understanding the chromatin interactions of regulatory elements. This study will help elucidate the molecular mechanisms of noncoding cancer drivers and identify potential therapeutic targets of prostate cancer.

Funding: NIH R21CA264637, R21CA260082, and R21HG011506

21: Multiple Myeloma Progression Associated Long Non-Coding RNA 1 Binds to Chromobox 4 Protein in Melphalan Resistance Cells and Can Be Targeted With Locked Nucleic Acid Antisense Oligos (K22)

Silva-Fisher, J.[1,2], Bagwill, K.[1], Thunuguntla, P.[1], Benton, A.[1], Gonzales, S.[1], Yao, L.[1], Jayasinghe, R.[1], Vij, R.[1,2], Ding, L.[1,2], DiPersio, J.[1,2]

[1]Washington University in St. Louis, St. Louis, MO: [2]Siteman Cancer Center, Washington University in St. Louis, St. Louis, MO

Introduction/Background: Multiple myeloma is one the most common hematologic malignancies. It is preceded by a premalignant phase called monoclonal gammopathy of undetermined significance (MGUS) and can then progress to smoldering multiple myeloma (SMM) and/or malignant myeloma. Despite advances in treatments most patients suffer relapse and the lack of reliable biomarkers to predict development of myeloma is a critical barrier. Long non-coding RNAs (lncRNAs) have important regulatory functions by binding to proteins and promoting cancer. We hypothesize that lncRNAs may serve as biomarkers in myeloma progression. Our objective is to identify lncRNAs that can contribute to myeloma progression to serve as markers and inform novel therapies.

Methods: We analyzed plasma cells from single-cell RNA sequencing (scRNA-Seq) from a publicly available dataset of normal (n=11), MGUS (n=7), SMM (n=6), and myeloma (n=12) patient samples and 18 myeloma samples from the Multiple Myeloma Research Foundation's CoMMpass Study. RT-qPCR and multiplexed Fluorescent RNA In situ Hybridization (mFISH) was used to detect expression. RNA immunoprecipitation (RIP) and individual-nucleotide resolution cross-linking immunoprecipitation (iCLIP) RT-qPCR was used to assess lncRNA-protein binding. Locked nucleic acid antisense oligonucleotides (LNA ASOs) were used to decrease expression.

Results: We identified six differentially expressed lncRNAs comparing MGUS to SMM, 14 lncRNAs comparing SMM to myeloma, and 19 lncRNAs comparing normal to myeloma, termed Multiple Myeloma Progression associated lncRNAs (MMPals). The top differentially expressed lncRNA, MMPal1, increased in MGUS to myeloma samples and when treated with melphalan. CBX4 RIP and iCLIP RT-qPCR show high enrichment of binding to MMPal1 in melphalan resistant cells. mFISH showed high nuclear MMPal1 expression in cells and patient samples. Lastly, MMPal1 LNA ASOs show a decrease in viability and increase in apoptosis.

Conclusion: We identified lncRNAs associated with myeloma progression and determined that MMPal1 is bound to CBX4. We believe that lncRNA-protein binding play important roles in myeloma progression and treatment resistance.

Funding: NIH NCI K22, Longer Life Foundation

22: HMGB1 Localization and Its Effect on the Immune Response in the Lung Tumor Microenvironment (K01)

Simmons, Jr., G.[1], Jafri, K.[2], Andre, N.[1]

[1]Department of Biomedical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, NY; [2]College of Human Ecology, Cornell University, Ithaca, NY

Introduction/Background: Lung adenocarcinoma is the leading cause of cancer mortality. High Mobility Group Box 1 (HMGB1) is a master regulator of innate immunity that is elevated in lung cancer tissues. HMGB1 contributes to PD-L1 expression in melanoma and promotes the secretion of inhibitory cytokines TGF-α and IL-10, which have roles in the differentiation of regulatory T cells. This indicates that HMGB1 influences an immunosuppressive signaling cascade in the tumor microenvironment. However, beyond showing that HMGB1 is elevated and modulates inflammation, studies to date have not comprehensively investigated the influence of HMGB1 on the immune response to lung cancer. Interestingly, unsaturated fatty acids are known for their anti-inflammatory capacity and effects on HMGB1 function. While treatment of lung cancers with immune checkpoint inhibitors (ICI) (anti-PD-1/PD-L1) has succeeded, there are limitations. One significant limitation is the failure of patients with advanced or refractory disease to generate a solid anti-tumor immune response. Therefore, we hypothesized that high monounsaturated fatty acids (MUFAs) promote an anti-tumor immune response in the tumor microenvironment by halting HMGB1 release from cancer cells.

Methods: To test this hypothesis we used a combination of proteomics, immunological assays, and 3-dimensional cell culture.

Results: Monounsaturated fatty acid (MUFA) availability negatively affected extracellular HMGB1 release from cancer cells and immune signaling myeoid derived cells

Conclusion: Exploiting the MUFA/HMGB1 signaling axis could be a novel approach to improve the efficacy of therapies in populations that are not currently benefiting from current strategies for eliminating tumors.

Funding: This work was funded by National Cancer Institute K01 Career Development award.

23: The Human Connection: Oncologist Characteristics and Behaviors That Facilitate Therapeutic Bonding With Latino Patients With Advanced Cancer (K08)

Tergas, A.[1,2,3], Prigerson, H[3,4], Penedo, F.[5], Maciejewski, P.[3,6]

[1]Department of Surgery, Division of Gynecologic Oncology, City of Hope Comprehensive Cancer Center, Duarte, CA; [2]Department of Population Science, Division of Health Equity, City of Hope Comprehensive Cancer Center, Duarte, CA; [3]Cornell Center for Research on End-of-Life Care, Weill Cornell Medicine, New York, NY; [4]Department of Medicine, Weill Cornell Medicine, New York, NY; [5]Departments of Psychology and Medicine, Miller School of Medicine, University of Miama, Miami, FL; [6]Department of Radiology, Weill Cornell Medicine, New York, NY

Introduction/Background: Therapeutic alliances (TA) between oncologists and patients are bonds characterized by mutual caring, trust, and respect. They lay the foundation for the provision of high-quality cancer care. We here relate oncologist characteristics and behaviors to TA in Latino vs. non-Latino patients with advanced cancer.

Methods: Participants included non-Latino oncologists (n=41) and their Latino (n=67) and non-Latino white (n=90) patients with advanced cancer who participated in Coping with Cancer III, a multi-site U.S.-based prospective cohort study of Latino/non-Latino disparities in end-of-life cancer care, conducted 2015-2019. Oncologist characteristics included age, sex, race, institution type, Spanish language proficiency, "familismo" practice style (embracing dedication, commitment, and loyalty to family) and clinical etiquette behaviors. Patient-reported TA was assessed using the average score of 6 items from The Human Connection Scale. Hierarchical linear modeling (HLM) estimated effects of oncologist characteristics on TA.

Results: Of 157 patients, most were female (n=92, 58.6%) and < 65 years old (n=95, 60.5%). Most oncologists were male (n=24, 58.5%), non-Latino white (n=25, 61%), and ≥ 40 years (n=25, 61%). An adjusted HLM in the full sample showed that Latino patient ethnicity was associated with significantly lower TA ($b = -0.25$, $p<0.001$). In an adjusted stratified HLM for TA, among Latino patients, oncologist "familismo" practice style ($b = 0.19$, $p= 0.012$), preference using first names ($b = 0.25$, $p= 0.023$), and greater Spanish fluency ($b = 0.11$, $p<0.001$) were positively associated with TA. In contrast, "familismo" practice style had no impact on TA for non-Latino white patients.

Conclusion: Latino patients with advanced cancer had worse therapeutic alliances with their oncologists vs. non-Latino patients. Modifiable oncologist behaviors may be targeted in an intervention designed to improve the patient-physician relationship between oncologists and their Latino patients with advanced cancer.

Funding: Dr. Tergas is a recipient of an NIH K08 Career Development Award (CA245193). Data collection was supported by R01 grant to Dr. Maciejewski and Dr. Prigerson on Latino/non-Latino cancer patient disparities in end-of-life care from the National Institute of Minority Health and Health Disparities (MD007652) and a UL1 grant to the Weill Cornell Clinical and Translational Science Center (CTSC) from the National Center for Advancing Translational Sciences (TR002384). Dr. Prigerson was supported by the National Cancer Institute R35 Outstanding Investigator Award (CA197730) and an National Institute of Minority Health and Health Disparities R21 (MD017704).

Postbaccalaureate/ Graduate Abstracts

24: Survival Outcomes in Older People Living With HIV and Cancer (iCURE)

Carey T., Lurain, K., Ramaswami, R.

National Cancer Institute, National Institutes of Health, Bethesda, MD

Introduction/Background: Antiretroviral therapy (ART) has led to improved survival outcomes for people with HIV (PWH), resulting in an aging population with HIV. In the United States, over 50% of the 1.3 million PWH are currently over the age of 50. There are limited data on cancer survivorship and cancer outcomes in older PWH.The objective of this retrospective study is to characterize cancer diagnoses in older PWH in the HIV and AIDS Malignancy Branch at the National Cancer Institute between 2001 and 2021.

Methods: We analyzed medical records of patients seen with HIV over 60 years old and evaluated cancer diagnoses before and after the age of 60 to determine cancers over the lifespan in older PWH. We calculated overall survival of patients diagnosed with cancer and HIV from the first clinic visit at or after 60 years of age to last follow-up/death by Kaplan-Meier method.

Results: We identified 32 patients 60 years or over seen in our clinic; 18 diagnosed with cancer < 60 years and 14 diagnosed > 60 years of age. Kaposi Sarcoma (62%) was the most common cancer diagnosed among all patients. Six patients (43%) > 60 years had more than one cancer diagnoses. Among all patients over the age of 60 years, the median overall survival OS was 6.8 years and the 5-year survival was 63%. Survival outcomes did not differ by age of initial cancer diagnosis before or after the age of 60 years (P=0.10).

Conclusion: There was no survival difference among those who were diagnosed with cancer after the age of 60 or prior to the age of 60 in the population studied. Cancer is an important comorbidity among PWH over the lifespan that can impact survival outcomes.

Funding: Funded by the Intramural Continuing Umbrella of Research Experiences (iCURE) Program and the Office of Intramural Training and Education (OITE).

25: Characteristics of Extra-Endocrine Features in a Cohort of Patients With MEN2B Seen at the NIH

Enyew, M.[1], Hogan, J.[1], Akshintala, S.[1], Acosta, M.[2], Glod, J.[1]

[1]Pediatric Oncology Branch, National Cancer Institute, National Institutes of Health, Bethesda, MD; [2]Undiagnosed Disease Program, National Human Genome Research Institute, National Institutes of Health, Bethesda, MD

Introduction/Background: Multiple endocrine neoplasia type 2B (MEN2B) is a rare autosomal dominant cancer predisposition syndrome caused by an activating germline mutation of the rearranged during transfection (RET) proto-oncogene (typically p.M918T). MEN2B is associated with 100% risk of medullary thyroid carcinoma (MTC), and approximately 50% lifetime risk of pheochromocytoma (PHEO). Mucosal ganglioneuromas, marfanoid habitus, gastrointestinal and musculoskeletal manifestations are common non-tumor manifestations of MEN2B syndrome.

Objective: To characterize the tumor and non-tumor features of MEN2B patients followed at the National Institutes of Health (NIH).

Methods: We analyzed the clinical data including physical exams, laboratory examinations, and imaging findings collected up to January 2023 for 55 patients with MEN2B followed at the NIH. Patients were enrolled in either the natural history study of children and adults with MEN2 (NCT01660984) or the natural history and biospecimen acquisition study for children and adults with rare solid tumors (NCT03739827). Data were obtained from medical records and evaluations performed during study visits at the NIH Clinical Center in Bethesda, Maryland, USA. Occurrence of non-tumor manifestations, PHEO, and MTC were assessed.

Results: All 55 patients had a germline RET p.M918T mutation. Age at diagnosis of MEN2B ranged from <1 year to 30 years old (median of 10 years), and 29 (52.7%) were female while 26 (47.3%) were male patients. Neurological, skeletal, gastrointestinal, and genitourinary manifestations were common in our population with 53 patients (96.4%) having neurological manifestations including hypotonia and breath holding spells in infancy. Forty-seven patients (85.5%) had skeletal features of MEN2B including scoliosis, pectus excavatum, and pes cavus. All 55 patients (100%) had gastrointestinal symptoms such as constipation and diarrhea, and 45 patients (81.8%) had genitourinary findings including kidney stones, history of undescended testicle, lesions, and urinary frequency/incontinence. All 55 patients (100%) had a history of MTC, and 14/55 patients (25.5%) had a confirmed diagnosis of PHEO by the data cut-off of January 2023. The median age of diagnosis of MTC and PHEO were 10 and 17 years respectively. The most common presenting symptoms leading to diagnosis of MEN2B/MTC included neck swelling (due to thyroid tumor formation or adenopathy), ganglioneuromas identified during routine medical care, and gastrointestinal issues that manifested early in life.

Conclusion: In addition to well-characterized extra-endocrine manifestations of MEN2B, patients with this syndrome may also have neurological, gastrointestinal, and genitourinary issues. Recognition of these features of MEN2B may facilitate earlier diagnosis of patients with this rare syndrome.

Funding: The National Institutes of Health Intramural Research Program

26: Longitudinal Follow-Up and Outcomes of Pediatric and Adult Patients With SDH-Deficient GIST (iCURE)

Kaschak, E.[1], Dagalakis, U.[1], Ghabra, S.[2], Ilanchezhian, M.[1], Del Rivero, J.[4], Miettinen, M.[5], Thomas, B.[1], Bernstein, D.[1], Raygada, M.[1], Flowers, C.[1], Killian, K.[6], Meltzer, P.[7], Reilly, K.[1], Wedekind-Malone, M.[1], Widemann, B.[1], Sankaran, H.[3], Blakely, A.[2], Glod, J.[1]

[1]Pediatric Oncology Branch, Center for Cancer Research, National Cancer Institute, National Institutes of Health, Bethesda, MD; [2]Surgical Oncology Program, Center for Cancer Research, National Cancer Institute, National Institutes of Health, Bethesda, MD; [3]Biometric Research Program, Division of Cancer Treatment and Diagnosis, National Cancer Institute, National Institutes of Health, Bethesda, MD; [4]Rare Rumor Initiative, Developmental Therapeutics Branch, Center for Cancer Research, National Cancer Institute, Bethesda, MD; [5]Laboratory Pathology, Center for Cancer Research, National Cancer Institute, Bethesda, MD; [6]Foundation Medicine, Inc., Cambridge, MA; [7]Genetics Branch, Center for Cancer Research, National Cancer Institute, Bethesda, MD

Introduction/Background: Gastrointestinal stromal tumor (GIST) are gastrointestinal non-epithelial neoplasms most commonly driven by somatic mutations in KIT or PDGFRA. Approximately ten percent of GIST are due to germline mutations in SDHx or epigenetic loss of expression of SDHC. While KIT and PDGFRa driven GIST respond to treatment with tyrosine kinase inhibitors, effective systemic therapies for SDH-deficient GIST have not yet been identified. A better understanding of the natural history of patients with these neoplasms is critical to identify effective treatments. A cohort of patients with SDH-deficient GIST is followed at the NIH Clinical Center.

Methods: Data from patients with SDH-deficient GIST enrolled in a study Natural History and Biospecimen Acquisition for Children and Adults with Rare Solid Tumors (NCT03739827) from January 2019 through January 2023 at the NIH were evaluated. In addition to review of medical records and imaging, when available, tumors were characterized by sequencing of SDH genes.

Results: Clinical information and specimens were collected from 60 GIST patients (median age at diagnosis 25, [range 7-73] years; 75% (45) female, 25% (15) male) were classified by molecular subtypes: 29.5% SDHC epimutation, 36.1% SDHA, 19.7% SDHB, 11.5% SDHC and 1.6% SDHD. Most commonly, patients had at least one surgery (58.3%) while a lower proportion had four or more (8.3%). Primary tumors occurred in the stomach with 15% having metastases at presentation the most common location being the liver (11.7%). Overall, 47.5% of patients were treated with imatinib, 21.3% were treated with sunitinib and 21.3% received other additional systemic therapies.

Conclusion: Longitudinal follow-up of an SDH-deficient GIST patient cohort will allow for better understanding of the natural history including treatment history and survival of patients with this rare disease and will help to optimize strategies for treatment and follow-up of these patients.

Funding: Funding provided by Cancer Moonshot to My Pediatric and Adult Rare Tumor Network.

27: Neuroimmune Modulation of Hepatic Tumors by Cholinergic Agonists

Nur, A.[1], Bauer, K.[1], Greten, T.[1,2]

[1]Thoracic and Gastrointestinal Malignancies Branch, Center for Cancer Research, National Cancer Institute, National Institutes of Health, Bethesda, MD; [2]Liver Cancer Program, Center for Cancer Research, National Cancer Institute, National Institutes of Health, Bethesda, MD

Hepatocellular carcinoma (HCC) is the fourth most common cause of cancer-related deaths, accounting for nearly 90% of all primary liver cancers (Llovet et al, 2021). Immunotherapy remains a viable standard of care treatment for HCC, but has limited efficacy. Emerging studies suggest that peripheral nerves shape cancer progression. Whether, and to what extent, these processes involve neuroimmune interaction remains largely unstudied.

Our lab recently showed that acetylcholine signaling influences cancer (unpublished). Bethanechol, an agonist for acetylcholine muscarinic receptors, alters HCC tumor burden hepatic immune features. Carbachol, an agonist for both muscarinic and nicotinic receptors, was also used to assess if it acts directly on tumor cells or the hepatic immune landscape. Here, we examine the in vivo impact of bethanechol on in vitro tumor cells. In addition, we explored how bethanechol and carbachol treatment shapes the immune populations in the absence of a tumor. We show that bethanechol and carbachol do not have a direct effect on the in vitro tumor growth or hepatic immune features.

28: Hinging on Success: Leveraging the Power of CAR T-Cell Therapy Through In-Silico Modeling of Hinge Length and Epitope Location (iCURE)

Mirazee, J.[1,2,3], Aranganathan, A.[4], Achar, S.[2], Jia, D.[2], Chen, X.[5], Vani, B.[4], Chien, C.[1], Pouzolles, M.[1], DeDe, K.1, Youkharibache, P.[6], Walters, K.[5], Tiwary, P.[4], Altan-Bonnet, G.[2], Taylor, N.[1]

[1]Pediatric Oncology Branch, Center for Cancer Research, National Cancer Institute, National Institutes of Health, Bethesda, MD; [2]Laboratory of Integrative Cancer Immunology, Center for Cancer Research, National Cancer Institute, National Institutes of Health, Bethesda, MD; [3]Department of Biology, Johns Hopkins University, Baltimore, MD; [4]Department of Chemistry and Biochemistry, University of Maryland, College Park, MD; [5]Center for Structural Biology, Center for Cancer Research, National Cancer Institute, National Institutes of Health, Frederick, MD; [6]Cancer Data Science Laboratory, Center for Cancer Research, National Cancer Institute, National Institutes of Health, Bethesda, MD

Introduction/Background: Genetic engineering of T-cells using synthetic proteins called chimeric antigen receptors (CARs) has shown promise in the treatment of relapsed/refractory B-cell leukemia. However, a significant percentage of patients will eventually relapse, often due to the emergence of tumor cells expressing low levels of the target antigen. Previous research has demonstrated that CAR the hinge domain plays a crucial role in regulating the cytotoxic capabilities of these cells.

Methods: In this study, we assessed the potential to model optimal hinge length for a given CAR in relation to the location of the targeted epitope. We designed an array of hinges originating from CD8-alpha and incorporated these hinges into CARs targeting CD19, CD22, CD33, and CD20 antigens. We then assessed the cytotoxic potential of each CAR design by using lentiviral transduction to introduce the synthetic gene to human T cells.

Results: We found that the location of the epitope is critical in determining the optimal hinge; a narrow range of short hinges is more effective against membrane-distal epitopes while long hinges are more effective against membrane-proximal epitopes. To build an in-silico model, we used Alphafold2 with a reduced the size of input MSA to increase the conformational diversity of the CAR hinges and target proteins. We discovered that CAR constructs with a predicted intermembrane distance of 15nm between the CAR and ligand were particularly effective against antigen-low leukemic cells.

Conclusion: Our findings suggest that CAR responsiveness against specific epitopes can be predicted based on intermembrane distance, enabling the rapid optimization of CAR constructs by an adjustment of hinge length. The development of this modeling strategy will facilitate the design of CARs with optimized cytotoxic potential against a range of novel targets.

Funding: The following work was made possible through the generous support of the NIH-Johns Hopkins University Graduate Partnerships program, Intramural Continuing Umbrella of Research Experiences program, and NIH Intramural program.

Postdoctoral Abstracts

29: Preclinical Efficacy Evaluation of Hybrid Oncolytic VSV-MORG in Acute Myeloid Leukemia (MD Diversity Supplement)

Barro, O.[1], Dumbauld, C.[1], Gabere, M.[1], Elliott, N.[1], Raupach, E.[1], Zhou, Y.[1], Moreno Cortes, E.[1], Borad, M.[1,2], Nagalo, B.[3,4]

[1]Division of Hematology and Medical Oncology, Mayo Clinic, Scottsdale, AZ; [2]Department of Molecular Medicine, Mayo Clinic, Scottsdale, AZ; [3]Department of Pathology, University of Arkansas for Medical Sciences, Little Rock, AR; [4]Winthrop P. Rockefeller Cancer Institute, University of Arkansas for Medical Sciences, Little Rock, AR

Introduction/Background: Acute myeloid leukemia (AML) is one of the most aggressive and deadliest blood malignancies. Many efforts have been made to improve the survival rate of patients but not all of them can benefit from the new therapeutic approaches, therefore, making chemotherapy the mainstay of therapeutic options despite its toxicity issues and being limited to a group of patients. Among these new approaches, oncolytic virotherapy, especially Vesicular stomatitis virus (VSV) vector-based is a promising tool with a great number of engineered recombinants having reached clinical trials, but its sensitivity to interferon is yet to be overcome and remains the most common main factor of resistance limiting its application for some cancer therapies.

Methods: In this study, we evaluated the efficacy of our hybrid oncolytic VSV-bearing Moreton (a close family to VSV) G envelope (VSV-MORG) in vitro and in vivo in human AML cells. Furthermore, a proteome analysis alongside the well-known and indispensable interferon analysis to assess this family of viruses to understand a potential interferon-independent resistance mechanism to this oncolytic virus.

Results: Our study demonstrated that VSV-MORG induces cell death in vitro and intravenously in the Cell-Derived Xenograph (CDX) model of MV4-11 human AML cells in the NOD/SCID murine model. However, resistance was noted when the remaining resistant populations of cells were cultured and re-infected. In addition, no significant enhancement of cell lysis was noted after inhibition of the interferon pathway before re-challenging the cells with the virus. Proteomics analysis of mock-infected, infected, and re-infected cells revealed 4426 proteins were differentially expressed ($p<0.05$), denoting the involvement of a potential mechanism of resistance independently of the interferon pathway to which VSV is highly sensitive.

Conclusion: VSV-MORG, a hybrid oncolytic virus, is a potent and safe biological agent candidate for combination therapy of AML; however, larger studies are required.

Funding: NCI/Diversity Supplement FP00086945-A1-PR02

30: Vagal-CD8+ T Cell Neuroimmune Axis Modulates Liver Cancer

Bauer, K.C.[1], Ruf, B.[1], Myojin, Y.[1], Benmebarek, M.R.[1], Nur, A.[1], Qi, J.[1], Green, B.L.[1,2], Wabitsch, S.[1], Ma, C.[1], Golino, J.L.[1], Korangy, F.[1], Greten, T.F.[1,3]

[1]Thoracic and Gastrointestinal Malignancies Branch, Center for Cancer Research, National Cancer Institute, National Institutes of Health, Bethesda, MD; [2]Surgery Branch, Center for Cancer Research, National Cancer Institute, National Institutes of Health, Bethesda, MD; [3]Liver Cancer Program, Center for Cancer Research, National Cancer Institute, National Institutes of Health, Bethesda, MD

Introduction/Background: Hepatocellular carcinoma (HCC) remains a leading cause of cancer-related death. Novel approaches are needed to alter the highly immunosuppressive liver landscape. Beyond immune innervation, peripheral nerves penetrate the tumor microenvironment. Yet the role of nerves or putative neuroimmune interactions within liver cancers remains unreported. The vagus nerve innervates the liver, regulating parasympathetic control via acetylcholine (ACh) release.

Methods: To assess HCC neuroimmunology, we performed a surgical hepatic branch vagotomy (HV) or sham procedure (SV). Our group assessed subsequent tumor growth in primary (RIL-175) and metastatic (B16-F10, A20) liver cancers with luciferase-labeled cancer cells assessed by in vivo imaging.

Results: Here we report that hepatic vagotomy reduces tumor burden across cancer models. Importantly, HCC findings remained organ specific as HV mice exhibited reduced tumor burden of intrahepatic, but not subcutaneous, models. Moreover, flow cytometry analyses revealed increased CD8+ T cells in HV tumor-bearing livers and increased expression of intracellular cytokines (IFNg, TNFa) within hepatic lymphocytes. To study if these findings were linked to ACh signaling, we measured hepatic ACh. HV livers exhibited decreased ACh levels (ELISA) and expression of ACh receptors Chrm1/Chrm3 (RT-qPCR) compared to SV controls. Treatment with bethanechol (ACh receptor agonist, 400 ug/mL drinking water) increased tumor burden. ACh exposure significantly reduced intracellular cytokine levels in ex vivo hepatic CD8+ T cells following anti-CD3/CD8 activation. Next, we assessed whether CD8+ T cells were required for vagal ACh-dependent outcomes. Bethanechol failed to promote tumor growth in Rag1KO mice lacking mature B and T cells, while targeted depletion of CD8+ T cells abrogated the effects of vagotomy.

Conclusion: These findings highlight a vagal-CD8+ T cell axis mediating HCC tumor burden via ACh signaling. This study furthers the emerging field of cancer neuroscience and identifies nerve-dependent targets to modulate immunosuppressive HCC features and outcomes.

Funding: Intramural Continuing Umbrella of Research Experiences grant ZIA BC 011345

31: Epigenetic Mechanisms in Oncogenic Genome Organization (iCURE)

Busby, T., Ozbun, L., Keikhosravi, A., Pegoraro, G., Misteli T.

Center for Cancer Research, National Cancer Institute, National Institutes of Health, Bethesda, MD

Introduction/Background: Regulation of the chromatin landscape during oncogenesis is important for the activation and maintenance of key oncogenes and cancer pathways. In particular, non-genetic factors, such as epigenetic modulators and higher order genome organization, modulate gene expression, DNA replication, and chromatin structure during tumorigenesis. However, the cellular factors and the mechanisms involved in establishing and maintaining oncogenic genome organization are largely unknown. The cohesin complex and the chromatin architectural protein CTCF have been identified as the prominent factors in shaping higher order genome organization via their involvement in the formation of Topologically Associating Domains (TADs). I hypothesize that additional factors are involved in the organization of the genome into higher order structures and that these factors are important for establishing the chromatin structure at oncogenic loci.

Methods: I am imaging the TAD comprising the human MYC gene as a model to investigate cancer-specific genome organization in colorectal cancer. To identify novel structural proteins, I am carrying out a high-throughput CRISPR screen using an sgRNA library targeting >1000 epigenetic regulators. CRISPR-Cas9 mediated deletions of target genes are generated followed by DNA fluorescence in situ hybridization (FISH) to visualize MYC TAD organization at the single cell level. Using high-throughput microscopy, I have begun to characterize structural chromatin features of the MYC oncogene to identify new epigenetic factors involved in regulating genome organization.

Results: My has screen identified hits that point to replication, chromatin remodeling, and histone modifying related genes in establishing TAD boundaries. Moreover, the leading hits caused a doubling in the distance between MYC TAD boundaries.

Conclusion: These results indicate chromatin remodeling and replication as major contributors to regulation of TAD structure, which is supported by literature in the field. Furthermore, investigating these hits will reveal mechanisms that regulate genome architecture at cancer related genes.

Funding: Intramural Continuing Umbrella of Research Experiences (iCURE)

32: RNA-seq Analysis of Endogenous Retroviral Elements in Tumor Cell Lines and Extracellular Vesicles (iCURE)

Chidester, S.[1], Krishnamurthy, S.[2], George, J.[1], Gertz, E.[3], Hutchins, E.[4], Fan, T.[5], Chen, Q.[5], Aldape, K.[1], Ruppin, E.[3], Belkaid, Y.[2], Meerzaman, D.[5], Van Keuren-Jensen, K.[4], Jones, J.[1]

[1]Laboratory of Pathology, Center for Cancer Research, National Cancer Institute, National Institutes of Health, Bethesda, MD; [2]Metaorganism Immunity Section, Laboratory of Host Immunity and Microbiome, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD; [3]Cancer Data Science Laboratory, Center for Cancer Research, National Cancer Institute, National Institutes of Health, Bethesda, MD; [4]Translational Genomics Research Institute, Phoenix, AZ; [5]Center for Biomedical Informatics & Information Technology, National Cancer Institute, National Institutes of Health, Bethesda, MD

Introduction/Background: Extracellular vesicles (EVs) can be mechanisms for viral or virus-like particle release. Murine leukemia virus (MLV)-related proteins have been shown to be enriched in mouse tumor cell line EVs relative to cells. No current study has surveyed endogenous retroviral (ERV) RNA repertoire in EVs, even though ERV elements account for ~9% of the human genome. In the current study we use RNA-seq to evaluate ERV RNA representation in mouse and human cell lines and corresponding EVs.

Methods: Cell line EVs were produced by incubating cultured cells with serum-free media for 48-72h. Conditioned culture media (CCM) was collected and concentrated by centrifuge ultrafiltration. EVs were isolated by size-exclusion chromatography. RNA-seq libraries were prepared from cell lines and EVs using the Takara SMARTer Stranded Total RNA-Seq Pico Input v3 library preparation kit. Sequencing was performed on the Illumina NextSeq550 platform. Data analysis was conducted using a novel ERV analysis pipeline.

Results: We conducted differential expression analysis to determine relative abundance of ERV transcripts in EVs vs cells and to identify ERVs significantly enriched in EVs. We evaluated the ratio of ERV transcripts to coding/non-coding transcripts in cells and EVs, as well as representation of ERV transcripts in the data. Intrinsic positive controls in the data included identification of known MMTV and MMLV sequences in mouse cell samples, as well as known cancer-associated hERV sequences in human samples.

Conclusion: In the process of establishing an RNA-seq pipeline and workflow for generating comprehensive EV ERV RNA data, we have identified a previously unreported pattern of ERV expression and processing in tumor cells. With this ERV RNA-seq pipeline, we observe a notable enrichment of ERV RNA elements secreted in EVs relative to their originating cells. Further analysis is required to evaluate how that enrichment varies with tumor type, cellular stress conditions, and route of EV biogenesis.

Funding: This project was supported by NIH UH3 TR002881, NIH ZIA BC011502, NIH ZIA BC011503, and NIH UH3 TR002881. SC is supported by an NCI Intramural Continuing Umbrella of Research Excellence (iCURE) award.

33: Pan-Viral Serology Uncovers Distinct Virome Patterns Among Hepatocellular Carcinoma and Control Populations

Do, W.[1], Wang, L.[1], Forgues,M.[1], Liu, J.[1], Rabibhadana, S.[2], Pupacdi, B.[2], Chaisaingmongkol, J.[2], Bhudhisawasdi, V.[3], Pairojkul, C.[3], Sukeepaisalkul, W.[3], Pugkhem, A.[3], Luvira, V.[3], Lertprasertsuke, N.[4], Chotirosniramit, A.[4], Auewarakul, C.[5], Ungtrakul, T.[5], Sricharunrat, T.[5], Sangrajrang, S.[6], Phornphutkul, K.[7], Budhu, A.[1,8], Harris, C.[1], Mahidol, C.[2], Ruchirawat, M.[1,9], Wang, X.[1,8,10]

[1]Laboratory of Human Carcinogenesis, Center for Cancer Research, National Cancer Institute, National Institutes of Health, Bethesda, MD; [2]Chulabhorn Research Institute, Bangkok, Thailand; [3]Khon Kaen University, Khon Kaen, Thailand; [4]Chiang Mai University, Chiang Mai, Thailand; [5]Chulabhorn Hospital, Bangkok, Thailand; [6]National Cancer Institute, Bangkok, Thailand; [7]Rajavej Hospital, Chiang Mai, Thailand; [8]Liver Cancer Program, Center for Cancer Research, National Cancer Institute, National Institutes of Health Bethesda, MD; [9]Center of Excellence on Environmental Health and Toxicology, Office of Higher Education Commission, Ministry of Education, Bangkok, Thailand

Introduction/Background: This study evaluates the pan-serological profiles of hepatocellular carcinoma (HCC) compared to several diseased and non-diseased control populations to identify novel risk factors and biomarkers of liver cancer.

Methods: Data derived from a Thai case-control study including individuals with HCC (n=663), chronic liver disease (CLD, n=199), and healthy population controls (PC, n=686). We used VirScan to screen patient serum samples for exposure to over 1280 strains of pathogenic and non-pathogenic viruses and develop a model discriminating HCC from PC using the XGBoost algorithm. Viral peptides which were differential between HCC and PC were further integrated with tumor mRNA expression data using trans-kingdom network analysis.

Results: We found 46 viral features which differentially discriminated HCC from PC. Integrating these features into a viral score showed differential enrichment across all populations adjusting for biological and lifestyle confounders. When examining the utility of the viral score in the at-risk population (CLD), we found the viral score was positively associated with several liver function markers (all $p<0.05$) and was also predictive of all-cause mortality (HR: 2.01, 95% CI: 1.06, 3.82), though clinical HCV and HBV were not predictive. Integrating the viral peptides with mRNA, the largest cluster included 406 peptides and 62 genes. Enriched pathways included innate and humoral immune response, killing of cells of another organism, and antimicrobial humoral response.

Conclusion: Our studies indicate that both hepatitis and non-hepatitis viral exposures were able to distinguish HCC from control populations. Using an aggregate 46 viral feature score, we found improved utility of the viral score at determining clinical risk and mortality in CLD patients compared to clinical hepatitis status. Finally, differential viral peptides clustered with immune response genes which may play a role in tumorigenesis.

Funding: This work was supported in part by grants (Z01 BC 010877, Z01 BC 010876, Z01 BC 010313, and ZIA BC 011870) from the intramural research program of the Center for Cancer Research, National Cancer Institute of the United States to X.W.W.

34: HIV-1 Antisense RNA Is Detected in Infected Cells In Vivo (iCURE)

Famuyiwa, T.[1], Capoferri, A.[1], Sklutuis, R.[1], Deeks, S.[2], Rausch, J.[1], Groebner, J.[1], Kearney, M.[1]

[1]HIV Dynamics and Replication Program, National Cancer Institute, National Institutes of Health, Frederick, MD; [2]Department of Medicine, Division of HIV/AIDS, University of California, San Francisco, San Francisco, CA

Introduction/Background: Natural antisense transcripts (NATs), which are transcribed from the opposite strand of protein-coding genes, regulate gene expression through epigenetic, post-transcriptional, and/or post-translational modifications. NATs have been described in many prokaryotic and eukaryotic systems, as well as in the viruses that infect them. The human immunodeficiency virus type I (HIV-1) has been shown to express one or more NATs. Several studies have demonstrated that HIV-1 antisense transcripts (Ast) is capable of inducing proviral latency in vitro. Our studies will determine if HIV-1 Ast is detected in vivo.

Methods: To answer this question, we developed and optimized a digital PCR-based assay to measure levels of Ast in small pools of infected cells using the ACH2 cell line. ACH2 cells each carry a single copy of an integrated HIV-1 provirus that expresses sense and antisense RNA at low levels.

Results: In 6 experimental replicates of 50 ACH2 cells, our digital PCR approach detected HIV-1 Ast in an average of 36% of the cells (range 22%-70%), consistent with previous studies. These results demonstrate that our assay can accurately detect and quantify levels of Ast in HIV-1 infected cells. We applied our new assay to PBMC collected from a donor with HIV-1 and on antiretroviral therapy. Testing 2 aliquots of 22 infected PBMC from this donor, we found that up to 54% of the infected cells contain HIV-1 Ast. .

Conclusion: These data demonstrate that Ast is expressed in people living with HIV-1 and lead to the question of whether HIV-1 Ast expression can contribute to viral latency and persistence during treatment. In our future studies, we will apply our quantitative digital Ast PCR approach to a cohort of about 50 people living with HIV-1 with varying levels of plasma viremia.

Funding: NIH

35: Understanding the Role of Immune Infiltrate Following Intense Neoadjuvant Androgen Deprivation Therapy in Locally Advanced Prostate Cancer (iCure)

Fenimore, J., Ku, A., Kissick, H., Sowalsky, A.

Introduction/Background: For patients with locally advanced (high-risk) prostate cancer, neoadjuvant therapies offer earlier opportunities for systemic therapy combined with the curative potential of definitive surgery. Prostate cancer is typically regarded as immunologically "cold" and the failure of neoadjuvant immune checkpoint therapies to treat prostate cancer has led to a wider discussion of how the immune activity of prostate tumors can be modulated. By contrast, earlier use of androgen deprivation therapy (ADT) intensified with AR antagonists in the neoadjuvant setting shows promise in delaying recurrence. Because ADT can also promote an immune response, the goal of this project is to assess changes to the local tumor immune microenvironment and determine whether there may be opportunities for combining neoadjuvant ADT and immunotherapy.

Methods: Patients who received six months of neoadjuvant ADT plus enzalutamide prior to surgery were subdivided into responders and non-responders based on the volume of residual tumor in the final surgical specimen. Radical prostatectomy (RP) sections were stained and immune cell populations were quantified to assess the presence of tumor proximal populations of CD4 and CD8 T-cells. Using laser capture microdissection, we are acquiring ultrapure populations of tumor cells and adjacent T cells. Our characterization of these T-cells will include determinations of T-cell receptor (TCR) diversity as well as the evaluation of differential gene expression associated with activation states of the tumor and proximal immune cell populations across each section of residual tumor. Included in this analysis will be an evaluation of residual tumor cells that lack dense immune infiltration, and how they contrast to more inflamed foci of treatment-resistant tumor.

Results: We observed a dramatic focal infiltration of lymphocytes adjacent to residual tumor foci. We also see a distinct difference pre and post therapy in the diversity of TCR and BCR populations in patients.

Conclusion: We see evidence of a potentially self-sustaining population of T-cells located in immunological lymph like structures within the human prostate. This population however lacks a clear inflammatory profile and expresses genes associated with a reduced level of immune activity. If this is due to an immunosuppressive phenotype by the tumor or its microenvironment, we will aim to determine whether this is due to T cell exhaustion. We will also determine whether immune checkpoint inhibition can augment the antitumor effect of neoadjuvant intense androgen deprivation therapies.

Funding: iCURE

36: Efficacy of Tempol and Deferiprone in Combination With Renal Cell Carcinoma Current Treatments (iCURE)

Medina, J., Ricketts, C., Crooks, D., Marston, L.

Urologic Oncology Branch, National Cancer Institute, National Institutes of Health, Bethesda, MD

Introduction/Background: Clear cell renal cell carcinoma (ccRCC) is the most common type of kidney cancer, characterized by the deficiency of the VHL gene. Loss of targeted degradation by the VHL complex results in the stabilization of HIF2α, allowing to induce the expression of the hypoxia response genes that can promote cancer growth. This led to the development of Belzutifan, an inhibitor of HIF2α that impedes the binding with ARNT cofactor necessary for transcriptional activation. Some VHL-deficient ccRCC tumors have shown resistance to Belzutifan, and we proposed a different method of inhibiting HIF2α by manipulating the mRNA translation. The 5' untranslated region of the HIF2α mRNA contains an IRE where IRP1 can bind and repress protein translation. The stable nitroxide Tempol indirectly represses HIF2α translation by oxidizing and disassembling the IRP1 Fe-S cluster into its APO form, which is necessary to bind to HIF2α mRNA. Also, since iron depletion is essential for inducing IRP1 activation, we could utilize iron chelators, such as Deferiprone, to repress HIF2α translation. Therefore, this project aims to inhibit HIF2α translation and activation with the long-term goal of enhancing the sensitivity of Belzutifan in our pre-clinical models of ccRCC.

Methods: We evaluated a series of drug combinations using Belzutifan, Tempol, and Deferiprone in ccRCC cell lines by assessing protein expression and gene targets of HIF2α through western blot and RT-PCR.

Results: We observed that Tempol + Deferiprone and Tempol + Belzutifan lowered HIF2α protein expression in UOK 121 and UOK 154. However, both combinations unexpectedly failed to downregulate genes induced by HIF2α.

Conclusion: Therefore, in our pre-clinical model, Tempol + Deferiprone can lower protein expression, in a similar manner to Belzutifan, but we have yet to demonstrate the previously observed downstream effects of reduced HIF2α protein levels.

Funding: JM is supported as a postdoctoral fellow by iCURE.

37: Discovery of Chemical Probes That Modulate the Association of E3 Ligase E6AP With the 26S Proteasome (iCURE)

Muli, C., Walters, K.

Protein Processing Section, Structural Biophysics Laboratory, Center for Cancer Research, National Cancer Institute, National Institutes of Health, Frederick, MD

Introduction/Background: The 26S proteasome is a sophisticated multi-catalytic enzymatic machine that performs regulated protein degradation of ubiquitinated substrates. Recently, induced degradation of non-native substrates by hijacking the E1-E2-E3 ubiquitination cascade with bifunctional molecules has emerged as a viable therapeutic strategy to reduce cellular concentrations of overexpressed proteins implicated in disease. E6AP is a privileged E3 ligase for the proteasome by its interaction with proteasome subunit Rpn10, and to date, the only E3 ligase with a known binding site at proteasomes. The role of E3 ligases at the 26S proteasome is unknown, and we hypothesize that E6AP's function involves branching of ubiquitin chains and/or substrate recruitment. Therefore, to understand how proteasome-associated E6AP can be utilized therapeutically, we aim to identify chemical probes that modulate the Rpn10:E6AP interaction to determine how loss or gain of proteasomal E6AP affects substrate processing and degradation.

Methods: Our discovery efforts utilize two approaches: virtual structure-based screening and high-throughput (HTP) small molecule screening. Using Schrödinger's virtual screening workflow, we have docked over 500,000 compounds from a diversity library onto the solved NMR E6AP:Rpn10 structure. For HTP development, Rpn10's C-terminal domain (RAZUL) is disordered when unbound but folds into a helical conformation when interacting with E6AP's N-terminal domain (AZUL). We are harnessing this conformational change to screen for inhibitors and molecular glues.

Results: For virtual screening, the top hits have been prioritized through computational refinement. Candidate molecules will be assessed for protein binding by NMR. For HTP assay development, successful acrylodan- and Atto610-labeling of RAZUL yielded significant fluorescent response when bound to AZUL, and assay optimization is ongoing.

Conclusion: Biophysical characterization of hits is ongoing. We expect that chemical probes discovered to perturb the RAZUL:AZUL interaction will not only be useful tools to improve our understanding of E6AP's role at the proteasome but also provide preliminary scaffolds for future drug design of E6AP:Rpn10 modulators.

Funding: Christine S. Muli is funded by the Intramural Continuing Umbrella of Research Experiences (iCURE) program.

38: Single Cell Characterization of the Adjacent Tissues and Metastatic Microenvironment of Adrenocortical Carcinoma Reveals Profound Molecular and Cellular Reprogramming Consistent With Metastatic Progression and Disease Outcome

Sikder, M.1,2, Gopalan, V.1, Burrola, C.2, Hannenhalli, S.1, Kaplan, R.2

1Cancer Data Science Laboratory, National Cancer Institute, National Institutes of Health, Bethesda, MD; 2Pediatric Oncology Branch, National Cancer Institute, National Institutes of Health, Bethesda, MS

Adrenocortical carcinoma (ACC) is a rare adrenal cancer that has high mortality due to aggressive metastasis. The heterogeneous compositions of immune/stromal cells in the metastatic microenvironment (MME) and adjacent tissue microenvironment (AME), and their crosstalk with malignant cells are critical determinants of cancer metastatic progression and response to therapies, which are yet poorly understood for ACC. Therefore, to comprehensively characterize MME and AME, and to identify global and metastatic tissue-specific reprogramming, we performed single cell RNA sequencing of MME and AME from the liver and lung. Our studies revealed that MME undergoes aberrant vascularization with global depletion of capillary endothelial cells (ECs) and enrichment for Tip/ Stalk-like ECs, which are significantly associated with poor disease outcome. Moreover, ECs in MME are highly pro-tumorigenic, showing global upregulation of tumor-promoting gene signatures, and key signaling pathways responsible for angiogenesis, proliferation, migration, and immune suppression. Furthermore, there is a major shift in tissue-specific myeloid populations in MME and AME, exhibiting global enrichment of dysfunctional DCs and immunosuppressive macrophages, and pDCs in MME, whereas MDSCs are broadly enriched in both MME and AME suggesting that these adjacent tissues may be primed for metastasis and show key features of pre- metastatic niche such as core immune suppressive program. CD8 T-cells in MME undergoes dysfunction, showing downregulation of key effector function genes. We also found an enrichment of immune suppressive CD4-T-regs in MME that represent dysregulation of immune checkpoint molecules including CTLA4 and TIGIT. Finally, immune/stroma cell populations in MME and AME show a common reprogramming program, favorable for tumor growth and metastasis, across diverse cell and tissue types in comparison to healthy tissue samples. Taken together, these findings suggest that ACC-MME and its adjacent lung and liver tissue microenvironment (AME) are reprogrammed to immunosuppressive and tumor-promoting states that orchestrate metastasis of ACC.

Funding: Intramural Continuing Umbrella of Research Experiences grant ZIA BC 011345, Center for Cancer Research

39: The Relationship Between Social Determinants of Health and Neurocognitive and Mood-Related Symptoms Among the Primary Brain Tumor Population (iCURE)

Stockdill, M.[1], Johnson, M.[1], Karim, Z.[1], Cooper, D.[2], Armstrong, T.[1], King, A.[1]

[1]Neuro-Oncology Branch, National Cancer Institute, National Institutes of Health, Bethesda, MD; [2]National Institutes of Health Library, National Institutes of Health, Bethesda, MD

Introduction/Background: Social determinants of health (SDoH) may contribute to half of health outcomes, but their impact on symptoms is less understood especially among the primary brain tumor (PBT) population.The aim of this systematic and narrative review of the literature was to examine the relationships between SDoH and neurocognitive and mood-related symptoms among the PBT population.

Methods: PubMed, EMBASE, and CINAHL were searched and 3,006 publications identified. SDoH were defined using PROGRESS criteria (Place of residence, Race/ethnicity, Occupation, Gender, Religion, Education, Socioeconomic status [SES], Social capital). Two individuals screened articles for eligibility and assessed study quality using the NHLBI Quality Assessment Tool for Observational Cohort and Cross-sectional Studies.

Results: Of 3,006 abstracts identified, 150 studies were assessed for eligibility and 48 included. 27 studies were cross-sectional with the majority from single institutions in the US (12), Germany (10), or Netherlands (5). 22 studies examined the relationship with 1 of the PROGRESS categories. No study examined all 8. Four studies measured place of residence, 2 race/ethnicity, 13 occupation, 42 gender, 1 religion, 18 education, 4 SES, and 15 social capital. Fifteen studies assessed neurocognitive symptoms and 37 mood-related symptoms with 4 measuring both. Results were disparate between symptoms and SDOH with evidence for higher education relating to less neurocognitive symptoms, and rural residence among individuals with a PBT or sustained unemployment after surgery among those with meningioma relating to depressive symptoms. Sixty-one percent of studies were fair in quality.

Conclusion: This is the first systematic review examining the relationship of symptoms in the PBT population and SDoH to date. Most studies were descriptive or exploratory and lacked comprehensive inclusion or definitions of SDoH. Future interventions should standardize SDoH collection, reduce study bias, and recruit diverse samples.

Funding: This work was supported by the NCI Center for Cancer Research Health Disparities Award.

40: Exploiting Metabolic Vulnerabilities in Neuroblastoma Through Pharmacological Inhibition of NAD Production to Impair Cell Survival, Energy Metabolism, and Tumor Growth (iCURE and Sallie Rosen Kaplan Fellowship)

Varriano, S.[1], Lee, U.[1], Collins, V.[1], Nguyen, R.[1], Sun, M.[1], Nelson, A.[1], Issaq, S.[2], Yeung, C.[1], Mendoza, A.[1], Thomas, C.[3], Thiele, C.[1], Heske, C.[1]

[1]Pediatric Oncology Branch, National Cancer Institute, National Institutes of Health, Bethesda, MD; [2]Urologic Oncology Branch, National Cancer Institute, National Institutes of Health, Bethesda, MD; [3]Division of Preclinical Innovation, National Center for Advancing Translational Sciences, National Institutes of Health, Rockville, MD

Introduction/Background: Neuroblastoma is the most common pediatric extracranial solid tumor. High-risk patients have a poor prognosis with an overall survival rate of <50%, thus novel therapeutic strategies are critically needed. To address this need, we performed a high-throughput drug screen and identified nicotinamide phosphoribosyltransferase inhibitors (NAMPTis) as highly active against neuroblastoma cells compared to other cancer cell types. NAMPT is the rate-limiting enzyme in the nicotinamide adenine dinucleotide (NAD+) salvage pathway, which cancer cells preferentially utilize to generate NAD+, a key co-factor that plays essential roles in energy metabolism. In this study, we investigated the translational potential and mechanistic effects of NAMPT inhibition in preclinical neuroblastoma models.

Methods: combined with results (see below)

Results: Using 3 NAMPTis in a panel of 10 neuroblastoma cell lines, we confirmed by Incucyte Live-Cell proliferation analysis that a majority of these models were highly responsive to NAMPTis. Notably, we observed complete growth inhibition in most models at doses <3.2nM of OT-82, a clinically relevant NAMPTi with a more favorable toxicity profile. On-target activity was confirmed by dose-dependent depletion of intracellular NAD+ in cells treated with OT-82. As NAD+ is necessary for energy metabolism, particularly ATP production, we performed a Cell-Titer Glo assay to indirectly assess ATP levels and observed decreases in all models with OT-82 treatment. Glycolysis and oxidative phosphorylation (OXPHOS) are two processes involved in ATP production that utilize NAD+, thus we measured activity of both processes via biochemical and extracellular flux analyses. With OT-82 treatment, we observed decreases in glucose consumption and lactate production in most models, and decreases in OXPHOS in some cell lines. Finally, an in vivo pilot study testing OT-82 in an orthotopic neuroblastoma PDX model demonstrated antitumor activity.

Conclusion: Together, these data show that neuroblastoma is susceptible to disruption of the NAMPT pathway and suggest that NAMPTis have translational potential as a novel therapy for neuroblastoma patients.

41: Persistent Disparities of Cervical Cancer Among American Indians/Alaska Natives: Are We Maximizing Prevention Tools? (K08)

Bruegl, A.[1], Emerson, J.[1], Tirumala, K.[2]

[1]Oregon Health and Science University; Portland, OR; [2]Northwest Portland Area Indian Health Board Tribal Epidemiology Center, Portland, OR

Introduction/Background: Bruegl AS (Oregon Health and Science University); Emerson J (Oregon Health and Science University); Tirumala, K (Northwest Portland Area Indian Health Board Tribal Epidemiology Center)

Methods: Cervical cancer incidence and mortality disparities experienced by American Indian/Alaska Native (AI/AN) women have persisted for decades. Pap smear screening and HPV vaccination are powerful tools to prevent cervical cancer. We evaluated the utilization of these tools among AI/ANs living in the Pacific Northwest (PNW).

Results: The Indian Health Service (IHS) National Data Warehouse's Epi Data Mart was analyzed using all healthcare visits from 2010 to 2020 from IHS, Tribal, and Urban Indian clinics in the PNW. Women ages 21-64 were included and considered up-to-date on pap smears if they had either cytology within 3 years, or cytology with HPV testing within 5 years of the most clinical encounter. HPV vaccination rates for both sexes were calculated for individuals ages 9-26. HPV vaccination was considered complete if: two vaccines were received prior to age 15 or after three vaccinations if initiated after age 15.

Conclusion: Cervical cancer screening rates are below the national average of 73.5% ranging between 57.1%-65.0%. Sub-analysis of age groups shows substantially lower rates of up-to-date pap smear screening in the 50-64 age group. HPV vaccination rates have increased over time for both sexes across all age groups. However, the current vaccination rate of 58.6% is well below the Healthy People 2030 goal of 84.3%.

Funding: Funding for this project was provided by the National Cancer Institute K08 Mentored Clinical Scientist Development Award.

42: Loss of Glutamine Synthetase Exacerbates Myeloid Cell Induced Immunosuppression in β-Catenin-Mutated Hepatocellular Carcinomas (K22)

Delgado, E.[1,3], Patel, P.[2], Tao, J.[1,3], Liu, S.[1,3,] Lehrich, B.[1], Poddar, M.[1], Singh, S.[1], Bell, A.[1,3], Monga, S.[1,3]

[1]School of Medicine, University of Pittsburgh, Pittsburgh, PA; [2]University of Pittsburgh, Pittsburgh, PA; [3]Liver Research Center,University of Pittsburgh, Pittsburgh, PA

Introduction/Background: Around 30% of HCC cases harbor mutations in CTNNB1, the gene that encodes β-catenin. These tumors can be identified with high sensitivity and specificity on histology by tumor-wide positivity for Glutamine Synthetase (GS), encoded by Glul, a well-known β-catenin target gene. Previously, the β-catenin-GS-glutamine-mTORC1 axis has been examined in β-catenin-mutated HCCs which demonstrate high susceptibility to mTOR inhibitors in preclinical models. Here, we investigate this axis further by targeting GS in β-catenin-mutated HCCs with a major focus on tumor immune environment.

Methods: HCCs were induced in Glul-floxed mice by co-delivering mutant-β-catenin-(T41A) and mutant nuclear factor erythroid 2–related factor 2 (Nrf2-G31A) (B+N model). Glul elimination following tumorigenesis was achieved by delivering AAV8 carrying Cre-recombinase. HCC burden was assessed in Glul-KO and controls. We preformed 10x Genomics Spatial Gene Expression profiling to measure mRNA with fluorescent immunostaining in tandem, as well as single-cell RNA-sequencing (scRNA-Seq) to identify changes in the immune microenvironment in CTNNB1-mutated HCCs ± GS.

Results: Glul was sufficiently deleted by two weeks from HCCs in the B+N model using AAV8-Cre. This was associated by decreased intratumoral glutamine levels. Interestingly, 4.5-weeks post Glul elimination, a significant increase in LW/BW ratio indicating a greater tumor burden was observed which also persisted at 7-weeks. Spatial expression analysis showed decreases in Mrc1, Adgre1, Cd68, Vsig4, and Cxcr4, markers of myeloid cells, which was also validated by immunohistochemistry. sc-RNA-Seq data identified a specific increase in the numbers of myeloid cells expressing immunosuppressive patterns in GS-knockdown HCCs.

Conclusion: Our data indicate that GS-loss increases the immunosuppressive function of myeloid cells thus contributing to the increase in disease burden in Glul-KO animals. Since myeloid cells are still detected in these tissues, our data suggests intrinsic Glul expression and in turn tumoral glutamine levels in β-catenin-mutated HCCs alter resident macrophage function and physiology

Funding: This research was supported by NIH/NCI R01CA250227-02 and NIH/NIDDK P30DK120531-01.

43: Exploring HPV Vaccine Concerns and Communication Needs Among Parents Who Have not Initiated nor Completed the HPV Vaccine Series for Their Child (K01)

Cunningham-Erves, J., Stewart, E.

Department of Internal Medicine, School of Medicine, Meharry Medical College, Nashville, TN

Introduction/Background: HPV vaccine rates remain suboptimal with only 58.6% of adolescents completing the vaccine series. Addressing parental vaccine hesitancy and non-compliance is a priority and tailoring communication strategies has been recommended. We explored vaccine concerns and communication needs of parents who have not initiated nor completed the HPV vaccine series for their child.

Methods: We applied concurrent multi-methods study (brief surveys and semi-structured interviews) with 32 parents whose adolescent had not initiated the vaccine series and 35 parents whose adolescent had initiated but not completed the vaccine series. Descriptives were used to analyze the survey data, and thematic analyses were used to analyze interview data.

Results: For parents who had not initiated the vaccine series, the top three concerns were: (1) child too young; (2) vaccine safety, and (3) child thinks its' okay to have sex post-vaccination. The top three concerns of parents who initiated but completed the vaccine series were: (1) the child did not like needles (91.4%), (2) the parent did not think the child needed additional shot (65.7%), and (3) the parent felt the child could wait until he/she was older (74.2%). Additionally, four themes emerged: 1) perceived benefits to initiating and completing the HPV vaccine series; 2) perceived barriers of initiating and completing the HPV vaccine series; 3) actual versus perceived information sources on HPV vaccination by child's vaccine status; and 4) actual versus preferred information sources on HPV vaccination by child's vaccine status.

Conclusion: Tailoring communication to fit best parental preferred information sources is integral to increasing HPV vaccine uptake and completion. Our next steps include testing HPVVaxFacts, a mobile-based webpage, which provides tailored messages to parents top concerns on HPV vaccination, in a pilot study to increase vaccination rates among parents who have not initiated or completed the HPV vaccine series for their child.

Funding: This work was supported by the National Cancer Institute of the National Institutes of Health (Award Number: 1K01CA237748-01) and American Cancer Society (Award Number: (ACS DICRIDG-21-071-DICRIDGT).

44: Insurance Type Drives Disparities in Stage at Diagnosis, Treatment, and Survival in Patients With Hepatocellular Carcinoma (1K08CA255413-01)

Jones, P.,[1,2], Kleyman, L.,[3] Prieto Bello, D.,[2] Mehta, V.,[4] Castaneda, S.,[2] Figueroa, G.,[2] Gaddipati, S.[5]

[1]Sylvester Comprehensive Cancer Center, Miller School of Medicine, University of Miami, Miami, FL; [2]Division of Digestive Health and Liver Diseases, Department of Medicine, Miller School of Medicine, University of Miami, Miami, FL; [3]Miller School of Medicine, University of Miami, Miami, FL; [4]Department of Hospital Medicine, Miller School of Medicine, University of Miami, Miami, FL; [5]Jackson Health System/ University of Miami Internal Medicine Residence, Miller School of Medicine, University of Miami, Miami, FL

Introduction/Background: Most of our knowledge regarding determinants of racial and ethnic disparities in hepatocellular carcinoma (HCC) risk and outcomes is derived from retrospective analyses. We aim to prospectively characterize how social factors interact with genetic ancestry to drive disparities in HCC risk and survival. This interim analysis explores the association between race/ethnicity and detailed insurance type on diagnosis, treatment, and survival in diverse HCC patients.

Methods: Since 2018, we have enrolled 1,052 patients with chronic liver disease, cirrhosis and HCC into an observational cohort. We also retrospectively follow HCC patients who could not enroll. We extract clinical data from the medical record. We performed chart review to categorize insurance type at diagnosis. We examined associations between race, insurance type and treatment using Pearson's chi-squared.

Results: We are following 1,029 HCC patients. We included 872 HCC patients with complete clinical and insurance information; the sample is 11.3% Black, 32.5% White, 51.5% Latino, 3.1% Asian, and 1.8% other. Insurance type is associated with stage at diagnosis, $p<0.001$. Individuals with private insurance or Medicare were more likely to receive treatment, 81.2% and 76.6% respectively, compared to 67.5% of Medicaid and 48.3% of uninsured patients, $p<0.001$. When stratified by Medicare type, 80.8% of Traditional Medicare (TM) patients were treated compared to 71.1% of Medicare Advantage (MA) patients, p 0.05; TM patients survived longer (783 d) than MA patients (405 d), $p < 0.001$. Only 50% of Black Medicare patients received treatment compared to 83.5% of White, 78.9% of Latino, and 100% of Asian individuals with Medicare, $p<0.001$.

Conclusion: Despite insurance, Black patients have limited access to HCC treatment. Insurance type is critically important to outcomes. Efforts to educate communities about coverage limitations and to assess and improve the quality of managed care Medicare and Medicaid are critically needed to improve HCC disparities.

Funding: 1K08CA255413-01

45: Telehealth Utilization by Rural Older Cancer Survivors: A Qualitative Assessment by Rural Older Cancer Survivors, Caregivers, and Healthcare Professionals

Lewis-Thames, M.[1,2], Trimble, S.[1], Rosen, F.[1],

[1]Center for Community Health, Feinberg School of Medicine, Northwestern University, Chicago, IL; [2]Department of Medical Social Sciences, Feinberg School of Medicine, Northwestern University, Chicago, IL

Introduction/Background: Rural telehealth utilization has increased, with some rural health hospitals reporting a 1700% increase in telehealth volume compared to pre-pandemic utilization. Yet, rural older adults are slower to adopt telehealth. Considering the rise of telehealth-delivered cancer care, it is imperative to understand the challenges rural older cancer survivors face when incorporating telehealth into their complex cancer care. Guided by the Andersen Healthcare Utilization Model, we qualitatively examined barriers and facilitators of telehealth utilization for rural older cancer survivors.

Methods: Ongoing interviews began March 2022. We purposively sampled rural older cancer survivors, caregivers of rural older cancer survivors, and healthcare professionals treating this population. All participants had to have used telehealth as- or with- a rural older cancer survivor. Interviews were conducted either in-person or by telephone. The interview guides probed participants regarding telehealth utilization for survivorship care. Two analysts conducted deductively coded to the Andersen Model constructs.

Results: Preliminary findings are based on 19 rural older cancer survivors, caregivers, and healthcare professionals. Most participants were Non-Hispanic White, female, traveled greater than 31 minutes to an oncologist, used their cell phone or computer to access telehealth. We coded similar perceptions between survivors, caregivers, and healthcare providers. Themes related to telehealth utilization for rural older cancer survivors included (1) Telehealth provided critical and necessary services, (2) Existing technological literacy and infrastructure served as enabling factors for telehealth uptake, (3) Geographic isolation related to the rural environment impaired participant's ability to access telehealth-delivered care, and (4) Virtual face-to-face provider-patient communication at critical points in participants' care facilitated telehealth utilization.

Conclusion: Preliminary findings revealed that telehealth was useful for overcoming barriers common to rurality and often a suitable strategy to deliver cancer care. Although telehealth was well-received, community-based programming focused on improving telehealth access and usability for rural older cancer survivors is warranted.

Funding: Dr. Lewis-Thames was supported by a grant from the National Cancer Institute (Grant Number: K01CA262342), a Northwestern University Clinical and Translational Sciences Institute grant (Grant Numbers: NUCATS; UL1TR001422, PI: D'Aquilla), a National Institutes of Health's National Institute on Aging (Grant Number: P30AG059988), and funds from the Northwestern University Center for Community Health (Grant Number: N/A). Opinions and comments expressed in this paper belong to the authors and do not necessarily reflect those of the National Institutes of Health.

46: High frequency of NAPRT silencing in neuroblastoma and rhabdomysarcoma confers sensitivity to NAMPT inhibitors (K08)

Vasquez, J.[1], Zhao, S.[2], Heer, C.[2], Muraleedharan, R.[2], Sundaram, R[2], Kalen, A.[3], Spitz, D.[3], Bindra, R.[2]

[1]Department of Pediatrics, Yale School of Medicine, New Haven, CT; [2]Department of Therapeutic Radiology, School of Medicine, Yale University, New Haven, CT; [3]Free Radical and Radiation Biology Program, Department of Radiation Oncology, University of Iowa, Iowa City, IA

Introduction/Background: Cells primarily synthesize nicotinamide adenine dinucleotide (NAD+) through the Preiss-Handler and Salvage pathways, driven by nicotinic acid phosphoribosyltransferase (NAPRT) and nicotinamide phosphoribosyltransferase (NAMPT), respectively. NAMPT inhibitors (NAMPTi) have been tested in clinical trials, but their efficacy has been limited by (a) a lack of appropriate biomarkers and (b) significant dose-limiting toxicities (DLTs). We sought to identify tumor types with loss of NAPRT expression, and to determine if NAPRT loss can sensitize tumors to NAMPTi treatment.

Methods: We probed methylation and gene expression data from the Cancer Dependency Map (DepMap). NAPRT protein expression was detected by western blot, and in vitro growth delay assays were performed with and without nicotinic acid (NA) supplementation. NAD(H) levels were determined using the NAD/NADH-Glo assay kit (Promega). For in vivo experiments, RD cells were injected into the flank of athymic nude mice (n = 10 mice/group). Mice were treated with vehicle control or FK866 at 20 mg/kg/dose by intraperitoneal injection twice daily for 4 days per week for a total of 4 cycles.

Results: Rhabdomyosarcoma and neuroblastoma cells exhibit a high rate of NAPRT promoter methylation and concurrent loss of NAPRT protein expression. NAPRT-silenced cells showed exquisite sensitivity to NAMPTi, while NAPRT expressing cells were completely rescued when NA was added to growth media. NAMPTi-sensitivity correlated with decreased NAD levels in NAPRT-silenced cells. In vivo, NAMPTi treatment resulted in significant tumor regression in a NAPRT-silenced rhabdomyosarcoma xenograft model.

Conclusion: These findings suggest that NAPRT loss may serve as a biomarker of NAMPTi sensitivity in rhabdomyosarcoma and neuroblastoma. Future studies are needed to validate these findings in isogenic and patient-derived xenograft (PDX) models, and to evaluate the expression of NAPRT in primary patient tumors. Targeting NAD metabolism also warrents further study as a strategy to enhance the efficacy of existing cytotoxic chemotherapy in these difficult-to-treat tumors.

Funding: American Cancer Society-Yale Cancer Center Pilot Grant, Curing Kids Foundation Award, Institutional start-up funds

Participant List

Participant List

2023 Professional Development Workshop Participants

Lucile Adams-Campbell, Ph.D.
University Professor of Oncology
Georgetown University Medical Center
Washgington, DC 20012
Email: lla9@georgetown.edu

Gregory Adams, Jr., M.S., Ph.D.
Program Director
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: gregory.adams2@nih.gov

Adebola Adegboyega, Ph.D.
Assistant Professor
University of Kentucky College of Nursing
Lexington, KY 40536
Email: adebola.adegboyega@uky.edu

H. Nelson Aguila, D.V.M.
Deputy Director
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: aguilah@mail.nih.gov

Askal Ali, Ph.D.
Associate Professor
Florida A&M University
Tallahassee, FL 32307
Email: askal.ali@famu.edu

Shantel Angstadt, Ph.D.
Postdoctoral Fellow
National Cancer Institute
Bethesda, MD 20892
Email: shantel.angstadt@nih.gov

LeeAnn Bailey, M.B.B.S, Ph.D., M.S.
Chief, Integrated Networks Branch
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: leeann.bailey@nih.gov

Francine Baker, M.S.
Graduate Student
National Institute of Neurological Disorders and Stroke
Gaithersburg, MD 20877
Email: francine.baker@nih.gov

Leomar Y. Ballester, M.D., Ph.D.
Assistant Professor
University of Texas MD Anderson Cancer Center
Houston, TX 77030
Email: lyballester1@mdanderson.org

Oumar Barro, M.D.
Postdoctoral Fellow
Mayo Clinic
Scottsdale, AZ 85259
Email: barro.oumar@mayo.edu

Kylynda C. Bauer, Ph.D.
Postdoctoral Fellow
National Cancer Institute
Bethesda, MD 20892
Email: kylynda.bauer@nih.gov

Jazmyn Bess, M.S.
Postbaccalaureate Fellow
National Cancer Institute
Rockville, MD 20850
Email: jazmyn.bess@nih.gov

Traci N. Bethea, Ph.D.
Assistant Professor
Office of Minority Health and Health Disparities Research
Georgetown Lombardi Comprehensive Cancer Center
Washington, DC 20003
Email: tb988@georgetown.edu

Raymond D. Blind, Ph.D.
Assistant Professor
Vanderbilt University School of Medicine
Nashville, TN 37204
Email: ray.blind@vanderbilt.edu

Participant List

Corinne Boulanger-Espeut, Ph.D.
Program Director
Cancer Training Branch
National Cancer Institute
Gambrills, MD 21054
Email: boulangc@mail.nih.gov

Molly Boyajian, A.A.
Program Specialist
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: boyajianm@mail.nih.gov

Amanda S. Bruegl, M.D.
Associate Professor
Oregon Health and Science University
Portland, OR 97211
Email: bruegl@ohsu.edu

Dionne Burt
Program Analyst
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: burtd@mail.nih.gov

Theodore Busby, Ph.D.
Postdoctoral Fellow
National Cancer Institute
Bethesda, MD 20892
Email: theodore.busby@nih.gov

Jessica M. Calzola, Ph.D., P.M.P.
Program Director
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: jessica.calzola@nih.gov

Tishiya Carey, B.A.
Postbaccalaureate Fellow
National Cancer Institute
Bethesda, MD 20852
Email: tishiya.carey@nih.gov

Lesley M. Chapman Hannah, Ph.D.
Postdoctoral Fellow
National Cancer Institute
Gaithersburg, MD 20878
Email: lesley.chapman@nih.gov

Stephanie Chidester, Ph.D.
Postdoctoral Fellow
National Cancer Institute
Bethesda, MD 20892
Email: stephanie.chidester@nih.gov

Veronica Clavijo Jordan, Ph.D.
Assistant Professor of Radiology
Massachusetts General Hospital
Boston, MA 02129
Email: mclavijojordon@mgh.harvard.edu

Victoria Coan
Program Specialist
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: victoria.coan@nih.gov

Calvin L. Cole, Ph.D.
Assistant Professor
University of Rochester
Webster, NY 14642
Email: calvin_cole@urmc.rochester.edu

Frank Colon-Matos, B.S.N.
Postbaccalaureate Fellow
Division of Cancer Epidemology & Genetics
National Cancer Institute
Rockville, MD 20850
Email: frank.colon-matos@nih.gov

Behrous Davani, Ph.D.
Chief, Diversity Training Branch
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: behrous.davani@nih.gov

Participant List

Brian Davis
Communications Manager
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: brian.davis@nih.gov

Evan Delgado, Ph.D.
Assistant Professor
University of Pittsburgh School of Medicine
Pittsburgh, PA 15216
Email: evd7@pitt.edu

Anthony DiBello, Ph.D.
Program Director
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: anthony.dibello@nih.gov

Whitney Do, Ph.D.
Postdoctoral Fellow
National Cancer Institute
Bethesda, MD 20814
Email: whitney.leet@nih.gov

Nadine Dogbe, M.Sc.
Postbaccalaureate Fellow
Division of Cancer Epidemology & Genetics
National Cancer Institute
Bethesda, MD 20852
Email: nadine.dogbe@nih.gov

Katrina R. Ellis, Ph.D.
Assistant Professor
University of Michigan School of Social Work
Ann Arbor, MI 48176
Email: kahe@umich.edu

Mahider M. Enyew, B.S.
Postbaccalaureate Fellow
National Cancer Institute
Bethesda, MD 20814
Email: mahider.enyew@nih.gov

Jennifer C. Erves, Ph.D.
Associate Professor
Department of Internal Medicine
Meharry Medical College
Nashville, TN 37208
Email: jerves@mmc.edu

Arnold Etame, M.D., Ph.D.
Associate Professor
Moffitt Cancer Center
Tampa, FL 33612
Email: arnold.etame@moffitt.org

Myron K. Evans, Ph.D.
Principal Investigator
Seattle Children's Research Institute
Seattle, WA 98101
Email: myron.evansii@seattlechildrens.org

Adaora Ezeani, M.D.
Postdoctoral Fellow
National Cancer Institute
Rockville, MD 20850
Email: adaora.ezeani@nih.gov

Toluleke O. Famuyiwa, Ph.D.
Postdoctoral Fellow
National Cancer Institute at Frederick
Frederick, MD 21702
Email: famuyiwatoluleke@gmail.com

John M. Fenimore, Ph.D.
National Cancer Institute
Bethesda, MD 20814
Email: john.fenimore@nih.gov

Efrén J. Flores, M.D.
Associate Chair
Mass General Brigham Radiology Equity, Inclusion & Community Health
Massachusetts General Hospital
Boston, MA 02114
Email: ejflores@mgh.harvard.edu

Participant List

Chantel F. Fuqua, Ph.D.
Program Director
National Cancer Institute
Rockville, MD 20850
Email: chantel.fuqua@nih.gov

Musa Gabere, Ph.D.
Researcher
Mayo Clinic
Mesa, AZ 85205
Email: gabere.musa@mayo.edu

Katelyn Garfinkel, M.B.A.
Program Specialist
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: katelyn.garfinkel@nih.gov

Martha D. Gay, Ph.D.
Assistant Professor
Georgetown University
Washington, DC 20007
Email: MDG111@georgetown.edu

Sangeeta Ghosh, Ph.D.
Program Director
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: sangeeta.ghosh@nih.gov

Annie K. Gilbert, Ph.D.
Postdoctoral Fellow
National Cancer Institute
Bethesda, MD 20894
Email: annie.gilbert@nih.gov

Andrea Gillis, M.D.
Assistant Professor
University of Alabama at Birmingham
Birmingham, AL 35068
Email: agillis@uabmc.edu

Ellen Gomez, M.A.
Program Manager
Hollings Cancer Center
Medical University of South Carolina
Charleston, SC 29425
Email: gomezel@musc.edu

Derek M. Griffith, Ph.D.
Professor
Georgetown University
Washington, DC 20057
Email: derek.griffith@georgetown.edu

Leonard A. Harris, Ph.D.
Assistant Professor
University of Arkansas
Fayetteville, AR 72701
Email: harrisl@uark.edu

Brandy Heckman-Stoddard, Ph.D., M.P.H.
Chief, Breast and Gynecologic Cancer Research Group
Division of Cancer Prevention
National Cancer Institute
Rockville, MD 20850
Email: heckmanbm@mail.nih.gov

Karen T. Hicklin, Ph.D.
Assistant Professor
Icahn School of Medicine at Mount Sinai
Newberry, FL 32669
Email: karenhicklin@gmail.com

Max H. Hogshead, B.Sc.
Postbaccalaureate Fellow
Division of Cancer Epidemiology & Genetics
National Cancer Institute
Rockville, MD 20850
Email: max.hogshead@nih.gov

Hunter K. Holt, M.D.
Assistant Professor
University of Illinois Chicago
Chicago, IL 60612
Email: hholt2@uic.edu

Participant List

Leah Hubbard, Ph.D.
Program Director
National Cancer Institute
Rockville, MD 20850
Email: leah.hubbard@nih.gov

Shadab Hussain, Ph.D.
Program Director
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: shadab.hussain@nih.gov

Rachel Issaka, M.D.
Assistant Professor
Fred Hutchinson Cancer Research Center
Seattle, WA 98109
Email: rissaka@fredhutch.org

Sonia B. Jakowlew, Ph.D.
Program Director
National Cancer Institute
Rockville, MD 20850
Email: jakowles@mail.nih.gov

Janice Jeter, B.A.
Program Analyst
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: jeterj@mail.nih.gov

Patricia D. Jones, M.D.
Associate Professor
Sylvester Comprehensive Cancer Center
Miami, FL 33136
Email: pdjones@med.miami.edu

Tarsha Jones, Ph.D.
Assistant Professor
Florida Atlantic University College of Nursing
Boca Raton, FL 33431
Email: jonest@health.fau.edu

Erika S. Kaschak, B.A.
Postbaccalaureate Clinical Research Fellow
Pediatric Oncology Branch
National Cancer Institute
Bethesda, MD 20892
Email: erika.kaschak@nih.gov

Lillian Kuo, Ph.D.
Program Director
Division of Cancer Biology
National Cancer Institute
Rockville, MD 20850
Email: lillian.kuo@nih.gov

Iñigo Landa, Ph.D.
Principal Investigator
Brigham and Women's Hospital & Harvard Medical School
Boston, MA 02115
Email: ilanda@bwh.harvard.edu

Rita LaPointe, M.B.A.
Program Analyst
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: rita.lapointe@nih.gov

Marquita Lewis-Thames, Ph.D., M.P.H.
Assistant Professor
Feinberg School of Medicine
Northwestern University
Chicago, IL 60619
Email: marquita.lewis-thames@northwestern.edu

Daniel Liefwalker, Ph.D.
Research Assistant Professor
Oregon Health and Science University
Portland, OR 97201
Email: liefwalk@ohsu.edu

Jason Liu, Ph.D.
Program Director
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: liuj9@mail.nih.gov

Participant List

Saria Lofton, Ph.D.
Assistant Professor
University of Illinois Cancer Center
Chicago, IL 60612
Email: Slofto4@uic.edu

Sylvia E. Long, Ph.D., C.P.H.
Program Director
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: sylvia.long@nih.gov

Maria A. Lopez-Olivo, M.D., Ph.D.
Associate Professor
University of Texas MD Anderson Cancer Center
Houston, TX 77047
Email: amlopezo@mdanderson.org

Melissa Lucero, Ph.D.
Postdoctoral Fellow
Chemical Biology Laboratory
National Cancer Institute
Frederick, MD 21702
Email: lucerom2@nih.gov

JoBeth McCarthy, D.H.Sc., M.P.H.
Program Director
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: jobeth.mccarthy-jean@nih.gov

Corinne McDaniels-Davidson, Ph.D., M.P.H.
Assistant Professor of Public Health
San Diego State University
San Diego, CA 92182
Email: cmcdaniels@sdsu.edu

Jasmine McDonald, Ph.D.
Assistant Director
Cancer Research, Training, and Education Center
Herbert Irving Comprehensive Cancer Center
Columbia University
New York, NY 10032
Email: jam2319@cumc.columbia.edu

Julia I. Medina Velazquez, Ph.D.
Postdoctoral Fellow
Cancer Research Center
National Cancer Institute
Bethesda, MD 20892
Email: julia.medinavelazquez@nih.gov

Justin M. Mirazee, B.S.
Graduate Research Assistant
National Institutes of Health
Bethesda, MD 20814
Email: mirazeejm@nih.gov

Yamilé Molina, Ph.D.
Associate Director, Community Outreach & Engagement
University of Illinois Cancer Center
Chicago, IL 60612
Email: ymolin2@uic.edu

Whitney A. Morelli, Ph.D.
Assistant Professor
Medical College of Wisconsin
Milwaukee, WI 53226
Email: wmorelli@mcw.edu

Christine S. Muli, Ph.D.
Postdoctoral Fellow
National Cancer Institute
Frederick, MD 21702
Email: christine.muli@nih.gov

Mercedes Muwanguzi, Ph.D.
Postdoctoral Fellow
Center for Cancer Research
National Cancer Institute
Bethesda, MD 20892
Email: mercedes.cornelius@nih.gov

Bolni M. Nagalo, Ph.D.
Professor of Medicine
University of Arkansas for Medical Sciences
Little Rock, AR 72205
Email: nagalobolnim@uams.edu

Participant List

Timiya S. Nolan, Ph.D., APRN-CNP, ANP-BC
Associate Professor
Division of Preventive Medicine
University of Alabama at Birmingham
Birmingham, AL 35205
Email: sharel44@uab.edu

Felicite Noubissi, Ph.D.
Associate Professor
Jackson State University
Jackson, MS 39217
Email: felicite.noubissi_kamdem@jsums.edu

Amran A. Nur, B.A.
Postbaccalaureate Fellow
National Cancer Institute
Bethesda, MD 20892
Email: amran.nur@nih.gov

April Oh, Ph.D., M.P.H.
Senior Advisor
Division of Cancer Control and Population Sciences
National Cancer Institute
Rockville, MD 20850
Email: april.oh@nih.gov

Juan C. Osorio, M.D.
Assistant Attending
Memorial Sloan Kettering Cancer Center
New York, NY 10065
Email: osorioj@mskcc.org

Humberto Parada, Ph.D.
Associate Professor
Division of Epidemiology and Biostatistics
San Diego State University School of Public Health
San Diego, CA 92182
Email: hparada@sdsu.edu

Elyse R. Park, Ph.D., M.P.H.
Professor
Massachusetts General Hospital
Boston, MA 02114
Email: epark@mgh.harvard.edu

Stephanie Pitts, Ph.D.
Postdoctoral Fellow
National Cancer Institute
Bethesda, MD 20892
Email: stephanie.pitts@nih.gov

Katherine Ramos, Ph.D.
Assistant Professor
Duke University School of Medicine
Durham, NC 27705
Email: katherine.ramos@duke.edu

Vito W. Rebecca, Ph.D.
Assistant Professor
Johns Hopkins Bloomberg School of Public Health
Lutherville-Timonium, MD 21093
Email: vrebecc2@jh.edu

Dawn Reid
Program Specialist
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: dawn.reid@nih.gov

Maria Jamela R. Revilleza, Ph.D.
Program Director
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: mariajamela.revilleza@nih.gov

Jeyshka M. Reyes-Gonzalez, Ph.D., M.Sc.
Postdoctoral Fellow
Center for Cancer Research
National Cancer Institute
Bethesda, MD 20892
Email: jeyshka.reyes@nih.gov

Suhn K. Rhie, Ph.D.
Assistant Professor
University of Southern California
Los Angeles, CA 90033
Email: rhie@usc.edu

Participant List

Analiz Rodriguez, M.D., Ph.D.
Director of Neurosurgical Oncology
University of Arkansas for Medical Sciences
Little Rock, AR 72205
Email: arodriguez@uams.edu

Laritza M. Rodriguez, M.D., Ph.D.
Program Director
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: laritza.rodriguez@nih.gov

Charles R. Rogers, Ph.D., M.P.H.
Associate Professor and Endowed Chair
Institute for Health & Equity
Medical College of Wisconsin
Milwaukee, WI 53226
Email: crrogers@mcw.edu

Fulera Salami, M.P.H.
Health Specialist
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: fulera.salami@nih.gov

Sandra L. San Miguel-Majors, M.S., Dr.P.H.
Program Director
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: sandra.sanmiguel@nih.gov

Mohd Omar F. Sikder, Ph.D.
Postdoctoral Fellow
Cancer Data Science Laboratory
National Cancer Institute
Bethesda, MD 20814
Email: mohdomar.sikder@nih.gov

Jessica M. Silva-Fisher, Ph.D.
Assistant Professor
Washington University in St Louis
Saint Louis, MO 63110-1005
Email: silvajm@wustl.edu

Glenn E. Simmons, Jr., Ph.D.
Assistant Professor
Cornell University
Ithaca, NY 14853
Email: glenn.simmons@cornell.edu

Sanya A. Springfield, Ph.D.
Director (Office of the Director)
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: springfs@mail.nih.gov

Whitney Barfield Steward, Ph.D.
Program Director
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: whitney.barfield@nih.gov

Macy Stockdill, Ph.D.
Postdoctoral Fellow
National Cancer Institute
Bethesda, MD 20814
Email: macy.stockdill@nih.gov

Janae D. Sweeney, Ph.D.
Assistant Professor
Department of Biological & Physical Sciences
South Carolina State Univeristy
Orangeburg, SC 29117
Email: jsweene4@scsu.edu

Sarah M. Szurek, Ph.D.
Program Director
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: sarah.szurek@nih.gov

Fred K. Tabung, Ph.D., M.P.H.
Assistant Professor
The Ohio State University
Columbus, OH 43210
Email: fred.tabung@osumc.edu

Participant List

Irene Tami-Maury, Dr.P.H.
Assistant Professor
University of Texas Health Science Center at Houston
Houston, TX 77030
Email: Irene.Tami@uth.tmc.edu

Ana Tergas, M.D.
Assistant Professor
City of Hope
Duarte, CA 91010
Email: atergas@gmail.com

Courtney Thomas, Ph.D.
Assistant Professor
South Carolina State University
Orangeburg, SC 29117
Email: cthoma17@scsu.edu

Shahrooz Vahedi, Ph.D.
Program Director
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: shahrooz.vahedi@nih.gov

Bryan Valcarcel, M.D.
Postdoctoral Fellow
Division of Cancer Epidemology & Genetics
National Cancer Institute
Rockville, MD 20850
Email: bryan.valcarcel@nih.gov

Suzanne S. Vang, Ph.D.
Assistant Professor
Grossman School of Medicine
New York University
New York, NY 10016
Email: suzanne.vang@nyulangone.org

Sophia Varriano, Ph.D.
Postdoctoral Fellow
National Cancer Institute
Bethesda, MD 20892
Email: sophia.varriano@nih.gov

Juan Vasquez, M.D.
Assistant Professor
Yale School of Medicine
New Haven, CT 06520
Email: juan.vasquez@yale.edu

Anil Wali, Ph.D.
Program Director
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: walia@mail.nih.gov

Tiffany Wallace, Ph.D.
Program Director
Center to Reduce Cancer Health Disparities
National Cancer Institute
Rockville, MD 20892
Email: wallaceti@mail.nih.gov

Clayton C. Yates, Ph.D.
John R. Lewis Professor of Pathology
Johns Hopkins University School of Medicine
Baltimore, MD 21205
Email: cyates10@jhmi.edu

Corey Young, Ph.D.
Postdoctoral Fellow
National Cancer Institute
Rockville, MD 20892
Email: corey.young@nih.gov

Nastaran Zahir, Ph.D.
Branch Director
Center for Cancer Training
National Cancer Institute
Rockville, MD 20850
Email: nas.zahir@nih.gov

Jelani C. Zarif, Ph.D.
Robert E. Meyerhoff Endowed Professor/
Assistant Professor
Johns Hopkins University School of Medicine
Baltimore, MD 21287
Email: jzarif1@jhmi.edu

CRCHD Resources

CRCHD Resources

Overview

- Website
- Contact Information and Staff Bios
- Twitter: @NCICRCHD
- LinkedIn: NCI Center to Reduce Cancer Health Disparities (CRCHD)

CRCHD Funding Opportunities

- **Disparities Research**
  - **Basic Research in Cancer Health Disparities (R01 Clinical Trial Not Allowed)**
    Tiffany Wallace, Ph.D.
    Program Director
    tiffany.wallace@nih.gov
  - **Exploratory/Developmental Grants Program for Basic Research in Cancer Health Disparities (R21 Clinical Trial Not Allowed)**
    Tiffany Wallace, Ph.D.
    Program Director
    tiffany.wallace@nih.gov
  - **Basic Research in Cancer Health Disparities (R03 Clinical Trial Not Allowed)**
    Tiffany Wallace, Ph.D.
    Program Director
    tiffany.wallace@nih.gov
  - **Exploratory Grant Award to Promote Workforce Diversity in Basic Cancer Research (R21 Clinical Trial Not Allowed)**
    Laritza M. Rodriguez, M.D., Ph.D.
    Program Director
    laritza.rodriguez@nih.gov

    Sangeeta Ghosh, Ph.D.
    Program Director
    sangeeta.ghosh@nih.gov
  - **Patient Derived Xenograft (PDX) Development and Trial Centers (PDTCs) Network (U54 Clinical Trial Not Allowed)**
    Tiffany Wallace, Ph.D.
    Program Director
    tiffany.wallace@nih.gov
  - **Feasibility and Planning Studies for Development of Specialized Programs of Research Excellence (SPOREs) to Investigate Cancer Health Disparities (P20 Clinical Trial Optional)**
    Tiffany Wallace, Ph.D.
    Program Director
    tiffany.wallace@nih.gov

CRCHD Resources

- **Diversity Training**

  - **Mentored Career Development Awards:**
    - **NCI Mentored Research Scientist Development Award to Promote Diversity (K01 Independent Clinical Trial Not Allowed) and (K01 Clinical Trial Required)**
      Shahrooz Vahedi, Ph.D.
      Program Director
      shahrooz.vahedi@nih.gov

    - **NCI Mentored Clinical Scientist Research Career Development Award to Promote Diversity (K08 - Independent Clinical Trial Not Allowed) and (K08 Clinical Trial Required)**
      Laritza M. Rodriguez, M.D., Ph.D.
      Program Director
      laritza.rodriguez@nih.gov

  - **Non-Mentored Career Development Award:**
    - **NCI Transition Career Development Award to Promote Diversity (K22 Independent Clinical Trial Not Allowed) and (K22 Clinical Trial Required)**
      Shahrooz Vahedi, Ph.D.
      Program Director
      shahrooz.vahedi@nih.gov

  - **Ruth L. Kirschstein National Research Service Award (NRSA) Individual Predoctoral Fellowship to Promote Diversity in Health-Related Research (Parent F31-Diversity)**
    Anthony DiBello, Ph.D.
    Program Director
    anthony.dibello@nih.gov

    Sangeeta Ghosh, Ph.D.
    Program Director
    sangeeta.ghosh@nih.gov

  - **Notice of Special Interest (NOSI): Research Supplements to Promote Re-Entry and Re-Integration into Health-Related Research Careers (Admin Supp - Clinical Trial Not Allowed)**
    Belem López, Ph.D.
    Program Director
    belem.lopez@nih.gov

CRCHD Resources

- **Research Supplements to Promote Diversity in Health-Related Research (Admin Supp - Clinical Trial Not Allowed)**
  Belem López, Ph.D.
  Program Director
  belem.lopez@nih.gov

  Fulera Salami, M.P.H.
  Health Specialist
  fulera.salami@nih.gov

  JoBeth McCarthy, D.H.Sc.(c), M.P.H., CPH (contractor)
  Program Director
  jobeth.mccarthy-jean@nih.gov

  Jiexin (Jason) Liu, Ph.D., M.B.A., M.S.
  Program Director
  liuj9@mail.nih.gov

Workforce Diversity

- **Cancer Moonshot Scholars Program**
  LeeAnn Bailey, M.B.B.S., Ph.D., M.S.
  Chief, Integrated Networks Branch
  leeann.bailey@nih.gov

  Tiffany Wallace, Ph.D.
  Program Director
  tiffany.wallace@nih.gov

- **Early Investigator Advancement Program (EIAP)**
  JoBeth McCarthy, D.H.Sc.(c), M.P.H., CPH (contractor)
  Program Director
  jobeth.mccarthy-jean@nih.gov

  Maria Jamela (Jay) R. Revilleza, Ph.D., M.Sc.
  Program Director
  mariajamela.revilleza@nih.gov

CRCHD Resources

CRCHD Programs and Initiatives

- **Intramural Continuing Umbrella of Research Experiences (iCURE)**
  Jessica Calzola, Ph.D., P.M.P.
  Program Director
  iCURE@nih.gov

  Gregory Adams, Jr., M.S., Ph.D.
  Program Director
  iCURE@nih.gov

- **Administrative Supplement for Strengthening Research, Training, and Outreach Capacity of the Geographic Management of Cancer Health Disparities Program (GMaP)**
  Anil Wali, Ph.D.
  Program Director
  walia@mail.nih.gov

- **Administrative Supplements to Strengthen NCI-Supported Community Outreach Capacity through Community Health Educators (CHEs) of the National Outreach Network (NON)**
  Sandra L. San Miguel-Majors, Dr.P.H., M.S.
  Program Director
  sandra.sanmiguel@nih.gov

- **Comprehensive Partnerships to Advance Cancer Health Equity (CPACHE) (U54 Clinical Trial Optional)**
  H. Nelson Aguila, D.V.M.
  Deputy Director
  aguilah@mail.nih.gov

  Behrous Davani, Ph.D.
  Chief, Diversity Training Branch
  behrous.davani@nih.gov

- **Training Navigation (TN)**
  Anil Wali, Ph.D.
  Program Director
  walia@nih.gov

- **Transformative Educational Advancement and Mentoring Network (TEAM)**
  Whitney Barfield Steward, Ph.D.
  Program Director
  whitney.barfield@nih.gov

CRCHD Resources

- **Youth Enjoy Science (YES) Research Education Program (R25)**
  Belem López, Ph.D.
  Program Director
  belem.lopez@nih.gov

- **Administrative Supplements to Support Cancer Disparity Collaborative Research (Clinical Trial Optional)**
  Maria Jamela (Jay) R. Revilleza, Ph.D., M.Sc.
  Program Director
  mariajamela.revilleza@nih.gov

- **Connecting Underrepresented Populations to Clinical Trials (CUSP2CT)**
  Maria Jamela (Jay) R. Revilleza, Ph.D., M.Sc.
  Program Director
  mariajamela.revilleza@nih.gov

  - **A Multilevel Approach to Connecting Underrepresented Populations to Clinical Trials (CUSP2CT; U01 Clinical Trial Optional)**
    Sandra L. San Miguel-Majors, Dr.P.H., M.S.
    Program Director
    sandra.sanmiguel@nih.gov

    Whitney Barfield Steward, Ph.D.
    Program Director
    whitney.barfield@nih.gov

  - **Data, Evaluation and Coordinating Center for: A Multilevel Approach to Connecting Underrepresented Populations to Clinical Trials (CUSP2CT) (U24 Clinical Trial Not Allowed)**
    Whitney Barfield Steward, Ph.D.
    Program Director
    whitney.barfield@nih.gov

    Sandra L. San Miguel-Majors, Dr.P.H., M.S.
    Program Director
    sandra.sanmiguel@nih.gov

- **Feasibility Studies to Build Collaborative Partnerships in Cancer Research (P20 Clinical Trial Not Allowed)**
  Behrous Davani, Ph.D.
  Chief, Diversity Training Branch
  behrous.davani@nih.gov

CRCHD Resources

- **Notice of Special Interest in Research on the Health of Sexual and Gender Minority (SGM) Populations**
  LeeAnn Bailey, M.B.B.S., Ph.D., M.S.
  Chief, Integrated Networks Branch
  leeann.bailey@nih.gov

  Whitney Barfield Steward, Ph.D.
  Program Director
  whitney.barfield@nih.gov

- **Research on the Health of Transgender and Gender Nonconforming Populations: R21, R01**
  LeeAnn Bailey, M.B.B.S., Ph.D., M.S.
  Chief, Integrated Networks Branch
  leeann.bailey@nih.gov

  Whitney Barfield Steward, Ph.D.
  Program Director
  whitney.barfield@nih.gov

NIH Resources and Tools

NIH Grant Application Resources

- NIH Grants and Funding Information: grants.nih.gov/
  - Find funding
  - How to apply
  - Explore NIH-funded research (RePORT)
- Center for Scientific Review: public.csr.nih.gov/
- Early Stage Investigator Policies: grants.nih.gov/policy/early-stage/index.htm

NIH/NCI Training Resources

NCI Resources for Researchers: cancer.gov/research/resources

Extramural

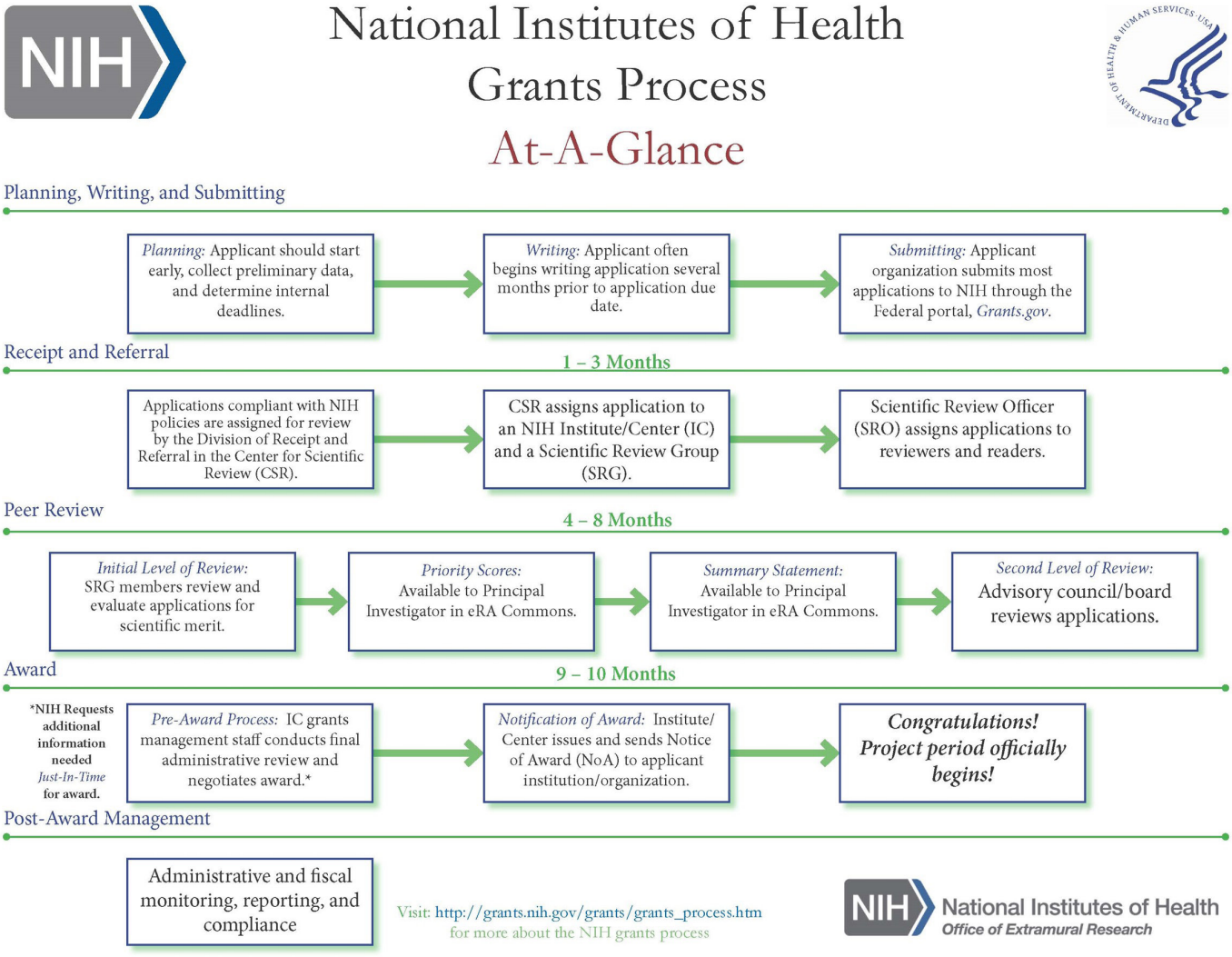
- NIH Extramural Diversity: extramural-diversity.nih.gov
- Center for Cancer Training: cancer.gov/grants-training/training/about
- Continuing Umbrella of Research Experiences (CURE): cancer.gov/about-nci/organization/crchd/diversity-training/cure

Intramural

- Office of Intramural Training and Education: training.nih.gov/
- NIH Intramural Research Program: irp.nih.gov/
- NCI Center for Cancer Research: ccr.cancer.gov
- NCI Division of Cancer Epidemiology and Genetics: dceg.cancer.gov
- Cancer Prevention Fellowship Program: cpfp.cancer.gov
- Intramural Continuing Umbrella of Research Experiences (iCURE): cancer.gov/about-nci/organization/crchd/diversity-training/icure
- Lasker Clinical Research Scholars: nih.gov/research-training/lasker-clinical-research-scholars
- Stadtman Tenure Track Investigators: irp.nih.gov/careers/trans-nih-scientific-recruitments/stadtman-tenure-track-investigators

See our fact sheets for additional information about CRCHD programs, initiatives, and funding opportunities.

NIH Grants Process



Acknowledgments

Acknowledgments

CRCHD would like to give a special thanks to the Planning Committee members. We appreciate their contributions, commitment, and dedication in organizing the 2023 Professional Development Workshop.

NCI Center to Reduce Cancer Health Disparities

Director

Dr. Sanya A. Springfield

Co-Chairs

Dr. Laritza M. Rodriguez
Dr. Anil Wali

Members

Dr. Gregory Adams, Jr.
Dr. H. Nelson Aguila
Dr. LeeAnn Bailey
Ms. Dionne Burt
Dr. Behrous Davani
Mr. Brian Davis
Dr. Anthony Dibello
Dr. Chantel F. Fuqua
Ms. Katelyn Garfinkel
Dr. Sangeeta Ghosh
Dr. Shadab Hussain
Ms. Rita LaPointe
Dr. Sylvia E. Long
Dr. JoBeth McCarthy
Ms. Fulera Salami
Dr. Whitney Barfield Steward

Lumina Corps

Ms. Michelle Murray
Mr. Ben Neal


DEPARTMENT OF HEALTH & HUMAN SERVICES · USA
NIH
NATIONAL
CANCER
INSTITUTE