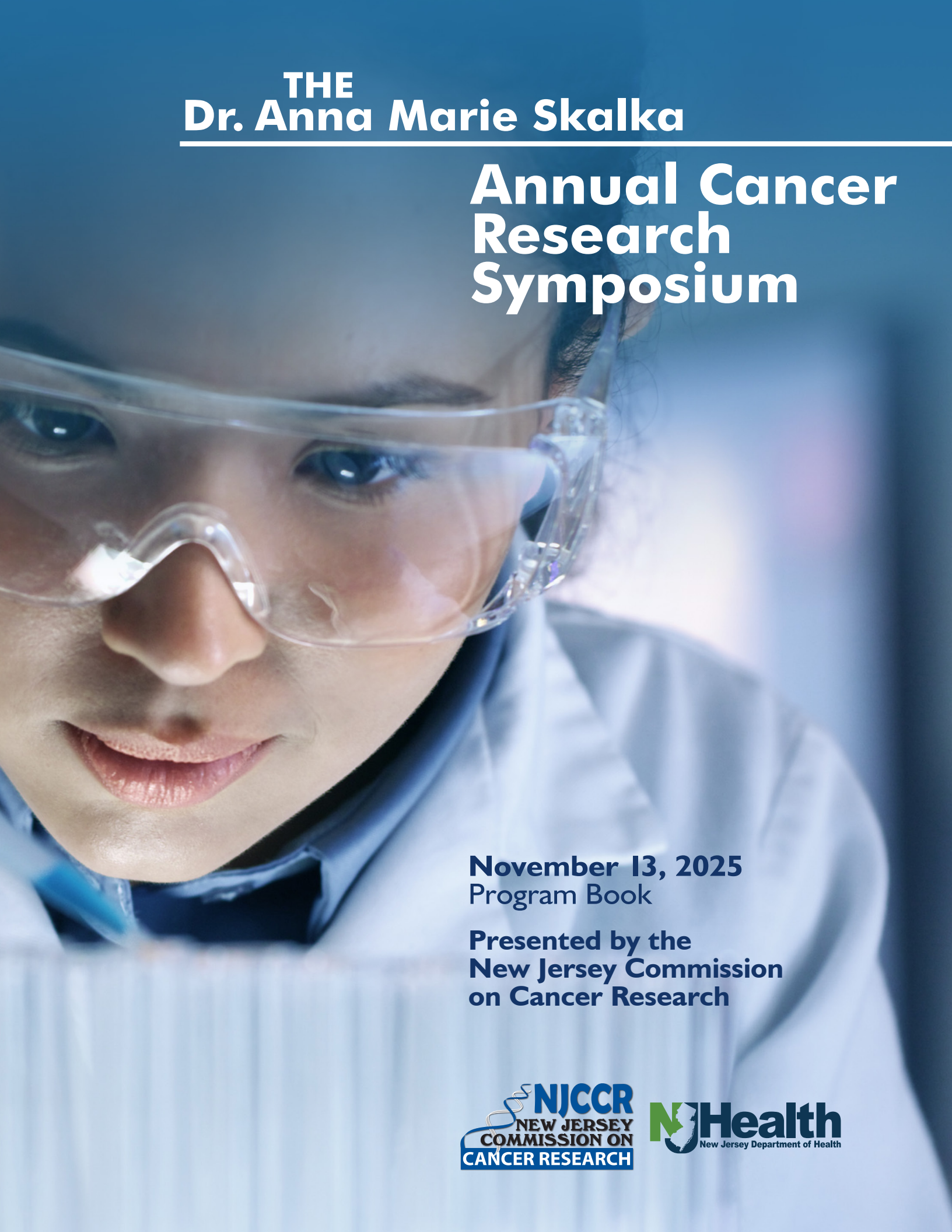


THE Dr. Anna Marie Skalka Annual Cancer Research Symposium

November 13, 2025
Program Book

Presented by the New Jersey Commission on Cancer Research

NJCCR NEW JERSEY COMMISSION ON CANCER RESEARCH

NJHealth New Jersey Department of Health

THE Dr. Anna Marie Skalka 9th Annual Cancer Research Symposium

NJCCR
NEW JERSEY COMMISSION ON CANCER RESEARCH

The New Jersey Commission on Cancer Research was ushered in by the Cancer Research Act in 1983, to support its activities. This Act resulted from the collaborative efforts of people with cancer and their families, clinicians, academicians, scientists, public officials, and representatives of research, pharmaceutical industry, and non-profit organizations.

Symposium Agenda at-a-Glance

8:30 am — **Registration/Continental Breakfast**

9:00 am — **Welcome**
Lisa Cummings, Executive Director, *NJCCR*
Dr. Kenneth Adler, Chair, *NJCCR*
Dr. Generosa Grana, Vice Chair, *NJCCR*

9:10 am — **Keynote Address**
Medicaid & the Cancer Safety Net: Recent Findings & Growing Concerns
Joel Cantor, Sc.D.
Edward J. Bloustein School of Planning and Public Policy Distinguished Professor of Public Policy, Rutgers University; Director, Center for State Health Policy, Rutgers University

10:00 am — **2025 Grantee Presentations**

Dr. Estella Jacinto
Dr. Juan Liu
Dr. Minh Ma
Dr. Zhaomeng Niu
Dr. Benjamin Tycko
Dr. Travis Baker
Dr. Rebecca Burdine
Dr. Daniel Herranz
Dr. James Holaska
Dr. Wenwei Hu

11:30 am — **Networking Session**

12:30 am — **Lunch**

1:30 pm — **Panel Discussion**
Employment, Career Pathways & Trends in Cancer
Elisa Bandera, M.D. Ph.D. – *Rutgers CINJ*
Ian McLaughlin, Ph.D. – *BioNJ*
Antoinette Stroup, Ph.D. – *NJ State Cancer Registry & Rutgers School of Public Health*
Eileen White, Ph.D. – *Rutgers Cancer Institute and Princeton Ludwig Institute for Cancer Research*

2:15 pm — **Award Presentations**
Legislative Champion Award
Dr. Jonathan Yavelow Mentor Award of Commendation
Patient Advocate Award

2:30 pm — **Concluding Remarks**

3:00 pm — **NJCCR Business Meeting – Closed Session**

NJCCR NEW JERSEY COMMISSION ON CANCER RESEARCH

Mission of the Commision:

To ensure that the people of New Jersey receive the fullest benefit of our nation's fight against cancer through its promotion of research into the causes, prevention and treatment of this disease.

Vision of the Commission:

To promote significant, original research in New Jersey and to fund talented researchers exploring the causes of cancer.

NJCCR
NEW JERSEY COMMISSION ON CANCER RESEARCH

Table of Contents

Award Presentations 2

2025 Awardees 3

Grantee Presentations 5

Research Presentations: 2026 Grant Recipients 12

Keynote Speaker 31

Panel Members 32

The Commission Members 36

NJ Pediatric Cancer Research Advisory Group 40

Index of Authors 41

NJCCR Staff Contact Information 42

Photography/video will be taken at this event. If you do not want to be filmed or photographed, please speak with a staff member.

Award Presentations

- **Legislative Champion Award:** The Legislative Champion Award is given to a state legislator or an individual who has championed cancer research at the state level. Past recipients have championed the New Jersey Commission on Cancer Research's work in funding state cancer research including: support for restoring state funding cuts to cancer research, introducing state legislation that would create a dedicated funding source for cancer research, and additional resources for cancer researchers at the state level in New Jersey.

- **Patient Advocate Award:** The Patient Advocate Award is dedicated to honoring advocates in the community who work to support cancer patients, whether pushing for early detection of cancer, better treatment options for cancer patients, or advocating for cancer research. Past recipients include cancer survivors and zealous advocates who have used their strength to continue the fight to defeat cancer long after treatment is done.

- **Dr. Jonathan Yavelow Mentor Award:** The Dr. Jonathan Yavelow Mentor Award was created to honor the work of Dr. Yavelow, a longtime member of the New Jersey Commission on Cancer Research who dedicated his work to mentor students over his storied career. Dr. Yavelow was a Professor of Biology at Rider University for 35 years and a member of the Commission since 1984. He was a dedicated researcher and beloved by his students, many of whom he mentored throughout the years. Mentorship plays a key role in supporting a successful career in cancer research. Therefore, outstanding mentors in cancer research are eligible for this award.

2025 Awardees

Legislative Champion Award:
The Honorable Aura K. Dunn
Assemblywoman, New Jersey 25th Legislative District

The Honorable Aura K. Dunn represents New Jersey's 25th Legislative District, where she serves on the Assembly Budget Committee and the Children, Families & Food Security Committee. A strong champion for cancer research, she authored the legislation establishing the New Jersey State Commission on Cancer Research Charitable Contribution Check-Off Fund and helped increase dedicated cancer research funding through the state's cigarette tax. She has also co-sponsored key measures supporting pediatric cancer research and expanding access to school- and college-based mental health services.

Assemblywoman Dunn is the bipartisan co-chair of the Legislative Disability Caucus and a committed advocate for veterans, families, and children. Before joining the New Jersey Legislature, she served on Capitol Hill with the U.S. Senate Appropriations Committee and the House Veterans Affairs Committee. Her public service has been recognized with multiple honors, including the Domestic Shelters' Purple Ribbon Award, the Arc of Morris Legislator of the Year Award, and the NJBIA Paul L. Troast Award.

Dr. Jonathan Yavelow Mentor Award:
Susy C. Kohout, Ph.D.
Associate Professor in the Biomedical Science Department at Cooper Medical School of Rowan University

Dr. Susy C. Kohout views cells and their critical communication from a molecular point of view. After earning her BS in Organic Chemistry from the California Institute of Technology, she branched out to study how proteins interact with calcium and with the lipid membrane, earning a Ph.D. in Chemistry and Biochemistry with a certificate in Biophysics from the University of Colorado, Boulder. Following her developing love of cell communication, she started her postdoctoral research at the University of California, Berkeley where she studied biophysics and neuroscience, discovering how electrical signaling is as fundamental as chemical signaling in cells. After her postdoc, Dr. Kohout started her own laboratory at Montana State University where she continued her research into neuroscience and the electrical signaling of cells while expanding to include cell biology and physiology. She recently joined the Biomedical Science faculty at Cooper Medical School of Rowan University where she will take advantage of the medical school to expand her research into more medically relevant directions.

2025 Awardees

Dr. Kohout has 9 years of experience teaching both undergraduates and graduate students in a classroom setting covering cell biology, molecular biology and molecular neuroscience. She has more than 20 years of experience teaching and mentoring students in the laboratory. She is looking forward to contributing to the Active Learning Groups (ALGs) as well as the lectures at CMSRU.

Dr. Kohout also strongly believes in serving her community. She has served on departmental, college and university wide committees. She also participates in outreach, particularly advancing and encouraging women and underrepresented minorities in STEM fields.

Patient Advocate Award:
John Bowlin

John and Barbara Bowlin Patient Assistance Fund at John Theurer Cancer Center

John Bowlin was first treated at Hackensack University Medical Center (HUMC) almost 10 years ago. While there, he noticed that many patients around him whether in the waiting room or in infusion chairs didn't have access to the same resources he did. He wanted to help. With support from John and his wife, the John and Barbara Bowlin Patient Assistance Fund was established at John Theurer Cancer Center (JTCC) to help patients cover the cost of transportation, food cards, lodging near the hospital, and other expenses that can make treatment difficult. Although John and Barbara now spend most of the year in Florida, they continue to generously support patient assistance at JTCC.

Patient Advocate Award:
Tracy Waits

Patient Navigator, MD Anderson Cancer Center at Cooper

A cancer survivor and tireless champion for patients, Tracy Waits brings empathy, strength, and unwavering dedication to her role as Gynecologic Oncology Patient Navigator at MD Anderson Cancer Center at Cooper. Her ability to connect with patients on both a clinical and deeply personal level makes her a beacon of hope and support. Tracy's own cancer journey inspired a profound calling to help others, leading her to change career paths and devote herself fully to patient advocacy. Her work exemplifies the heart of our mission, and we celebrate her as a true champion in our cancer community.

Travis Baker, Ph.D.
Affiliation: *Rutgers Biomedical and Health Sciences*

Project Title: ***Understanding Brain Adaptation After Pediatric Cancer Treatment: At the Intersection of Brain, Cognition, and Computation***

Intensive chemotherapy for pediatric cancer can affect brain function during treatment and recovery, creating challenges that extend beyond the medical treatment itself. Converging evidence demonstrates that 40-70% of childhood cancer survivors treated with central nervous system (CNS) chemotherapy show differences in attention, working memory, and processing speed. However, the specific neural mechanisms underlying these patterns remain poorly understood, limiting our ability to develop targeted support strategies. Our research examined how chemotherapy affects reward processing, cognitive control, and decision-making in pediatric cancer survivors, focusing on lymphoblastic leukemia and non-CNS solid tumor survivors aged 6-17 years. Using a multi-modal approach combining neural imaging, cognitive assessments, and computational modeling, we investigated these cognitive domains to better understand individual variation in post-treatment recovery. This talk will present our preliminary findings, which provide new insights into how the brain's reward processing systems adapt following cancer treatment. Electrophysiological results showed differences in reward-related brain activity patterns between cancer survivors and age-matched peers, with survivors showing altered neural responses during reward processing tasks. Additionally, computational modeling revealed that survivors used different learning strategies when processing positive feedback, suggesting the brain develops alternative pathways for processing reward information. Our multi-system approach enabled us to address questions spanning basic neuroscience, translational research, and clinical applications. These findings contribute to our understanding of cognitive recovery patterns and have potential implications for developing personalized support strategies and optimizing treatment approaches to better preserve cognitive function during cancer care.

Rebecca D Burdine, Ph.D.
Affiliation: *Princeton University*

Project Title: ***The role of Nodal signaling in promoting partial EMT migration***

The heart is one of the first organs to form during development, and it must be placed on the correct side of the body to work properly. In zebrafish embryos, the cells that will become the heart need to move as a group toward the left side before the heart takes shape. This movement is called “jogging.” If this process goes wrong, the heart can end up in the wrong place or develop incorrectly. Our research shows that the heart cell moves with

Grantee Presentations

characteristics that are similar to metastasis. We demonstrate that two important cell communication signals, called Nodal and FGF, work together to control how fast these heart-forming cells move. When FGF is missing, the cells move slowly. When both Nodal and FGF are missing, the cells do not move at all. Using advanced gene analysis, we discovered that heart-forming cells delay their response to Nodal signals until just before they start moving. This response leads to an increase in expression of genes that can promote migration. Overall, our research provides clues into the mechanisms that drive cancer metastasis and identified explored genes in this process for further study.

Daniel Herranz, Pharm.D., Ph.D.
Affiliation: *Rutgers Biomedical and Health Sciences*

Project Title: ***ACLY as a novel therapeutic target in T-cell leukemia***

T-cell acute lymphoblastic leukemia (T-ALL) is an aggressive blood cancer driven by a gene called NOTCH1. Despite recent progress in clinical outcomes in this disease, 25% of children and over 50% of adult T-ALL cases show primary resistant leukemia or respond only transiently to chemotherapy, and ultimately die, highlighting the need to discover novel and improved treatments. In this work, we found that ACLY (a multi-faceted enzyme controlling both metabolism and gene expression) plays an important role in this disease. Indeed, inhibiting ACLY with genetic tricks or with drugs led to extended survival in leukemic mice. Thus, ACLY is a novel target for the treatment of T-ALL.

James Holaska, Ph.D.
Affiliation: *Cooper Medical School of Rowan University*

Project Title: ***Emerin dysregulation compromises nuclear integrity during metastatic transformation***

The overarching goal of this research is to identify and characterize what drives breast cancer growth and metastases, and how to stop it. Breast cancer metastasis remains the most lethal event in the disease course and the current lack of treatment options for patients with metastatic disease drives negative patient outcomes. Therefore, ascertaining the mechanisms that enable metastatic spread can be used for development of preventative or therapeutic interventions, or for identifying biomarkers of tumor progression. For a tumor to metastasize, cancer cells need to enter and exit the nearby vessels by squeezing through extremely small gaps in the endothelium. The major physical barrier to cells moving through these narrow gaps is the size and stiffness of the nucleus. During transformation the nucleus changes from

a rigid structure, like that of a golf ball, to a more pliable structure, like that of a water balloon. These significant changes in nuclear size and structure allow the cancer cells to now pass through these narrow endothelial gaps with ease, and to metastasize. We found these nuclear properties are governed by the nuclear envelope protein emerin. First, we found invasive breast cancer cell lines had less emerin protein and that these cells had greater nuclear deformation and faster migration through tight spaces. Downregulation or inactivation of emerin was sufficient to enable cancer cell malleability. Second, analysis of more than 300 breast cancer samples from patients showed emerin levels inversely correlate with invasiveness. Third, we showed emerin expression in invasive breast cancer cells inhibits their invasion and metastasis in mouse models of breast cancer. To address a potential mechanism for emerin downregulation during tumorigenesis, we focused on the observation that as tumors grow, they become stiffer, and their nuclei become more malleable. This suggested tumor stiffness drives nuclear malleability. We found this increased stiffness caused emerin protein reduction and more malleable nuclei. This supports a model in which emerin senses mechanical signals from outside the cell and responds by reducing its interactions with the nucleoskeleton to make nuclei more malleable. Collectively, our results support emerin performing a central role in pathogenic transformation of malignant tissues.

Wenwei Hu, Ph.D.
Affiliation: *Rutgers Cancer Institute of New Jersey*

Project Title: ***The protective role of LIF in graft-versus-host disease***

Bone marrow transplantation, also known as allogeneic hematopoietic stem cell transplantation (allo-HSCT), can offer a potential cure for children facing certain blood cancers and genetic conditions. Yet, its benefits are often limited by a serious complication called graft-versus-host disease (GVHD). This condition arises due to antigen incompatibility between the transplant donor and recipient, which affects up to 70% of all allo-HSCT patients, and has the mortality rates between 20% and 75%. The mechanism of GVHD involves the donor T cell-induced damage to the vital organs of the recipient, including the gastrointestinal (GI) tract, skin, and liver. The severity and lethality of GVHD are primarily determined by the extent of damage to the GI tract. It has been shown that donor immune cells migrate to the GI tract, preferentially damage intestinal stem cells, which leads to GI injury and initiates GVHD. Leukemia inhibitory factor (LIF) is a multi-functional cytokine that plays many important roles in different physiological and pathological processes. Our study, supported by NJCCR, reveals that LIF decreases the infiltration and activation of donor immune cells in the GI tract and protects intestinal stem cells to ameliorate GVHD. Administering recombinant LIF protein protects against GVHD while preserving the anti-tumor efficacy of allo-HSCT in mice. This finding suggests that LIF could become a promising treatment

Grant programs are designed to provide scientific opportunities to attract young and seasoned research scientists.

Grantee Presentations

to protect healthy tissues from GVHD during bone marrow transplantation, without interfering with its anti-tumor benefit. If accomplished successfully, this proposed study will a) establish the protective role of LIF in GVHD and reveal its underlying molecular mechanisms; b) significantly increase our understanding of the molecular mechanism of GVHD; and c) have the direct potential to develop LIF as an effective therapeutic strategy to limit tissue damage without affecting the anti-leukemic efficacy of BMT. We expect results from this study will significantly impact clinical treatments for children with hematologic malignancies as well as non-malignant conditions who need BMT.

Estela Jacinto, Ph.D.
Affiliation: *Rutgers Biomedical and Health Sciences*

Project Title: *Targeting mTOR and metabolism in lymphoma*

Leukemias are the most common type of childhood cancer, accounting for about 30% of all cancers in children. In New Jersey, there is about 3-10% incidence (per 100,000) and although mortality rate has decreased due to improved treatments, there remains a need to devise more effective strategies since survival rates after relapse remain low. Importantly, there is also considerable toxicity with current treatment regimen that generates long-term undesirable consequences such as damage to other tissues. Most therapeutic strategies that are currently used in the clinic or undergoing pre-clinical/clinical trials exploit the vulnerability of the tumors to "addiction" or demand for particular nutrients by starving them or inhibiting their ability to acquire such nutrient(s). While initially effective in some cases, remaining abnormal cells acquire additional mutations that enable them to satisfy their voracious appetite for nutrients, often becoming highly metastatic tumors. The goal of our studies was to understand how a developing tumor, given a specific oncogenic driver mutation, reprograms its nutrient metabolism and how we can restore normal metabolism by dietary approaches to prevent their uncontrolled proliferation. Our lab generated mice wherein we turned off the expression of a tumor suppressor, PTEN, in cells from the thymus (thymocytes) that normally develop into T cells. When PTEN was turned off, the mice developed lymphoma around 15 weeks after birth (roughly equivalent to 12 year old in humans). We tracked the changes in metabolism and cell signals that promote growth in these mutant mice. We found that as they age, the thymocytes consume specific nutrients insatiably, as if continuously starved. Consequently, they turn on their "nutrient stress response" machinery to facilitate nutrient acquisition. Over time, this nutrient stress response becomes amplified, culminating in malignancy. We reasoned that feeding the mutant mice shortly after birth with the nutrients they crave for may then mitigate this stress response, thus delaying or preventing malignancy. Indeed, we obtained evidence supporting that diet supplementation significantly delayed malignancy up to 24 weeks (equivalent roughly to 60 years old in humans). With the advent of personalized medicine, tailoring dietary strategies that are specific

but less toxic and invasive could prove beneficial in cancer prevention particularly for individuals with known mutations that predispose them to malignancy. An effective diet manipulation could also serve to improve pharmacological therapies to treat cancer or prevent relapse.

Juan Liu, M.D., Ph.D.
Affiliation: *Rutgers Biomedical and Health Sciences*

Project Title: ***The role of mutant p53 accumulation and Gain of Function in colorectal cancer***

p53 is a key tumor suppressor. Loss of tumor-suppressive function of p53 leads to cancer development. The p53 gene is the most frequently mutated gene in human cancers; p53 mutations occur in >50% of all human cancers and in almost every type of cancer. Many p53 mutations in cancers not only lead to the loss of the tumor-suppressive function of p53 but also acquire new oncogenic activities to promote cancer progression, which is termed gain-of-function of mutant p53. Mutant p53 protein often accumulates to very high levels in cancer cells, which is critical for its gain-of-function activity to promote tumor development. Given the high mutation frequency of the p53 gene and the gain-of-function activities of mutant p53 in cancer, mutant p53 has become an attractive target for cancer therapy. A better understanding of the mechanisms underlying mutp53 protein accumulation and gain-of-function will help develop effective therapies treating human cancers containing mutant p53. With support from NJCCR, we investigated the mechanisms of mutant p53 protein accumulation and gain-of-function in human cancers, as well as potential therapeutic strategies targeting cancers carrying mutant p53.

Zhaomeng Niu, Ph.D.
Affiliation: *Rutgers Biomedical and Health Sciences*

Project Title: ***Understanding sun protection and skin examination practices among Hispanicsn***

Skin cancer is one of the fastest-growing cancers in the United States, and melanoma rates have increased by more than 22% among Hispanic individuals over the past two decades. Hispanic adults often face higher risks due to lower awareness, limited access to prevention resources, and less frequent use of sun protection. To better understand what influences these behaviors, we analyzed survey data from more than 175 Hispanic adults (first wave of a national survey). We examined how personal factors, cultural values,

Grantee Presentations

NJCCR was established in 1983 to promote and fund cancer research projects to scientists at qualified research institutions in New Jersey. Throughout its 40-year history, the NJCCR has awarded over $52 million for research grants and student fellowships.

Grantee Presentations

and beliefs shaped people's willingness to practice sun safety. Our results showed that participants who felt confident in their ability to protect themselves from the sun (self-efficacy) and those who already practiced sun-safe behaviors related to tanning were much more likely to engage in protection. Cultural values such as familism (the importance of family) also encouraged healthy sun practices. We also asked participants about their acceptance of new technologies for skin cancer prevention. Augmented Reality (AR), which overlays digital information onto the real world, shows promise as a prevention tool by making health information interactive and engaging. Participants were more likely to adopt AR for skin cancer prevention if they had higher education, enjoyed the AR experience, and felt that its use was supported by their social circles. These results suggest that combining culturally tailored strategies with engaging technologies like AR may improve sun protection and reduce skin cancer risk among Hispanic adults.

Minh Ma
Affiliation: *Rutgers Biomedical and Health Sciences*

Project Title: ***Evaluation of combination mTOR agonist and CD147-IL15-CAR-NK cell therapy in transgenic human CD147 Hepatocellular Carcinoma models***

Liver cancer is the second most common cause of cancer-related death worldwide. Liver cancer is caused by hepatitis B virus (HBV) and/or HCV infections; though, high fat diets and excessive alcohol consumption increases the risk of liver cancer developing. The worldwide burden of liver cancer is projected to be over 1 million cases by 2030. Liver cancer ranks fifth in terms of global cases and second in terms of death for men. Approximately 80% of liver cancer patients die within 12 months post-diagnosis due to limited effective treatments. It is becoming more evident that an individual's immune system is capable of naturally eliminating tumor cells. However, cancer cells develop different methods to evade the immune system, resulting in uncontrolled tumor growth. Chimeric antigen receptors (CARs) are a new promising approach used in cancer therapy where either the patient's or the donor's blood is used for the genetic modification of the immune cells. The engineered cells are grown in the lab and infused into a patient in order to destroy cancer cells, but not healthy cells. The biomarker that our lab focuses on is called CD147 which is shown to be abundantly present in liver cancer tissues but not healthy tissues. Leveraging this genetic modification technology, our lab has successfully engineered CD147-CAR-natural killer (NK) cells derived from healthy donors' blood to specifically target liver cancer cells. Our data show that CD147-CAR-NK cells significantly control liver cancer progression. Though the results are promising, we

continue to improve the functionality of CD147-CAR-NK cells in this proposal to further equip the patient's immune system. Completion of the proposed project will accelerate the bench-to-bedside of cancer immunotherapy in treating liver cancer and other solid tumors.

Benjamin Tycko, M.D., Ph.D.
Affiliation: *Hackensack Meridian Health School of Medicine*

Project Title: *Cell-cell interactions and epigenetic changes in multiple myeloma*

Multiple myeloma is a cancer of adults that arises in the bone marrow. There are approximately 35,000 new cases in the USA each year, and while there have been major advances in treatment cures have been elusive, with the 5-year survival rate currently about 60 percent. Our research on this important disease is based on two biological hypotheses, which we have been investigating in parallel. First, we posit that malignant MM cells are supported by other cells in the bone marrow, including osteoblasts, that are not malignant but are co-opted by the MM cells to help these cancer cells survive and proliferate. To better understand these cell-cell interactions, we have used a co-culture system in which non-malignant osteoblasts enhance MM cell growth and confer significant drug resistance to these malignant cells. Our transcriptomic analysis, after purifying the two cell types from the co-cultures, revealed widespread upregulation of interferon signaling pathway genes in the MM cells and increased expression of pro-inflammatory chemokines CXCL1 and CXCL8 in the osteoblasts. These findings, which we are now pursuing functionally with gene knock-down experiments, suggest possibilities for new therapeutic approaches targeting these pathways. Our second main hypothesis is that patterns of CpG methylation in the DNA of MM cells can reveal mechanisms by which these malignant cells arise and proliferate. Thus, we posit that analyzing such patterns can shed light both on the inherited risk of developing the disease and, when the overt MM arises, the risk of disease progression and treatment resistance. Our findings from whole-genome profiling applied to more than 50 primary MM cases suggest that risk of cancer formation and risk of cancer progression are mechanistically linked through altered net and allele-specific DNA methylation of some of the same key genes. Among these genes is CDKN2A, which is a regulator of cell proliferation that is a known inherited susceptibility gene for MM formation and which our profiling data nominate as a candidate single-gene marker for clinically aggressive treatment-resistant MM cases.

Bridge Grant Recipients

Bridge Grant Recipients

Type of Research: Basic
Project Title: *The mechanism and therapeutic potential of Parkin in Colorectal Cancer. COCR26RBG002*

Zhaohui Feng, Ph.D., fengzh@cinj.rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

Colorectal cancer (CRC) is the third most commonly diagnosed cancer and the second leading cause of cancer-related deaths in the United States. A better understanding of the mechanisms underlying CRC will provide novel and effective strategies for its treatment, which are urgently needed. Recent studies have suggested that the Parkin protein plays an important role in the suppression of CRC. Parkin protein levels are often decreased in CRC, and this decrease is associated with poor clinical outcomes in CRC patients. However, the precise mechanisms by which Parkin suppresses CRC remain unclear, hindering the development of effective therapeutic strategies for Parkin-deficient CRC. Metabolic reprogramming is a hallmark of cancer and a key contributor to cancer progression, making it an attractive target for cancer treatment, including CRC. To uncover new and important tumor-suppressive mechanisms of Parkin in CRC and develop new therapeutic strategies, we conducted preliminary studies. The results from our preliminary studies strongly suggest that Parkin regulates metabolism in cells, and Parkin deficiency in CRC drives metabolic reprogramming, which in turn promotes CRC. Furthermore, our preliminary results suggest that targeting the metabolic reprogramming induced by Parkin deficiency can effectively suppress CRC. Based on these preliminary results, we hypothesize that Parkin plays a critical role in metabolic regulation to suppress CRC, and furthermore, Parkin deficiency in CRC drives metabolic reprogramming to promote CRC progression, which is a potential therapeutic target. In this proposed study, we will determine the tumor-suppressive mechanisms of Parkin in CRC and test potential therapeutic strategies for Parkin-deficient CRC. The goal of this study is to uncover new molecular mechanisms and treatments for CRC. We expect that this study will significantly deepen our understanding of CRC mechanisms and provide a strong foundation for developing new and effective treatments for CRC, which are urgently needed in clinical practice.

Type of Research: Translational
Project Title: ***Mechanisms and Targeting Tumor-stromal/immune Signaling in Metastatic Gastric Cancer. COCR26RBG006***

Shumei Song, M.D., Ph.D., ssong@coriell.org
Affiliation: *Coriell Institute for Medical Research*

Gastric cancer (also called stomach cancer) is one of the most serious health problems worldwide. Nearly half of patients with gastric cancer develop a condition called peritoneal metastasis (PM), where the cancer spreads to the lining of the abdomen. This causes painful fluid buildup (ascites), blockages, weight loss, and weakness. Sadly, survival is usually less than six months, and treatment options are very limited. This is partly because we don't fully understand the biology of how these metastases grow and suppress the body's immune defenses. Our research focuses on a type of cell in tumors called cancer-associated fibroblasts (CAFs). These cells don't become cancer themselves, but they surround tumors and can help them grow, spread, and escape the immune system. We have discovered that a protein called YAP1 is highly active in CAFs. When YAP1 is active, CAFs produce a signal (a molecule called IL-6) that weakens immune cells, especially T cells that normally fight cancer. This creates a "safe space" for cancer cells to grow and spread in the abdomen. When we block YAP1 in these fibroblasts, tumors grow more slowly, and immune cells become stronger.

Grant recipients continued on next page

Bridge Grant Recipients

The purpose of the Bridge Grants is to enhance cancer-related research at New Jersey Institutions by providing funding to promising and productive investigators who anticipate a short-term interruption in funding for research projects focused on cancer prevention, diagnosis, treatment, and survivorship.

Pilot Grant Recipients

Type of Research: Basic Research
Project Title: ***Development of AI-designed molecular glues targeting NRF2Mut protein for cancer therapy. COCR26PPR001***

Zhaohui Xiong, Ph.D., zxiong@coriell.org
Affiliation: *Coriell Institute for Medical Research*

Our research endeavors have centered on NRF2, a protein frequently subject to mutations in human cancer, in particular, esophageal squamous cell carcinoma (ESCC). These mutations confer resistance to conventional treatments such as chemotherapy and radiation therapy. Our previous investigations led to one NRF2 inhibitor (pyrimethamine) which has moved into Phase I clinical trial. However, this drug also acts as an inhibitor of dihydrofolate reductase which may generate significant toxicities for cancer patients. When we further explore the mechanisms of action of pyrimethamine, we found this drug also acts as a "molecular glue," facilitating the tight binding of NRF2 to its inhibitory protein, KEAP1, and thus promotes NRF2 degradation. Using a series of chemical compounds similar to pyrimethamine, we found that pyrimethamine acts by fitting into a pocket of KEAP1 protein. In a pilot study, our collaborator at an AI drug design company (Insilico Medicine) helped us design several compounds which fit in this pocket but do not inhibit DHFR. One of the compounds was validated as a molecular glue in cell culture studies. In the proposed studies, we will synthesize 10 AI-designed small molecule degraders of mutant NRF2 protein, and test their mechanisms of action, and efficacy in ESCC cells and mouse models. We anticipate that this study will yield NRF2 inhibitors capable of effectively treating ESCC characterized by NRF2 mutations. Furthermore, we envision that the success of this research will pave the way for securing substantial NIH research grants, enabling us to further evaluate the efficacy and mechanism of additional NRF2 inhibitors and translate our findings into tangible clinical therapies of human cancer carrying NRF2 mutations in the future.

Pilot Grant Recipients

Type of Research: Psychosocial and Behavioral
Project Title: *Costs of Smoking: Economic Analyses and Modeling to Inform Policy. COCR26PPR006*

Carolyn Heckman, Ph.D., ch842@rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

The costs and taxes associated with purchasing cigarettes significantly influence the decisions to start, continue, or quit smoking, particularly among certain groups such as young adults and those with lower incomes. One specific behavior of interest is "relighting," which refers to the practice of extinguishing, saving, and later relighting unfinished cigarettes. Approximately half of cigarette smokers engage in this behavior, but little is known of its implications for quitting smoking and overall health. Preliminary evidence suggests that relighting may be linked to demographic and economic disparities, nicotine addiction, challenges in quitting, increased exposure to harmful chemicals, and tobacco-related diseases such as bronchitis and lung cancer. One common motivation for relighting is to save money by extending the use of a cigarette over multiple smoking sessions rather than smoking it all at once. The proposed project aims to further the understanding of relighting by utilizing nationally representative survey data collected from 2,000 individuals as part of a grant from the National Cancer Institute (Investigators: Heckman, Steinberg, Stepanov) that focused on cigarette relighting. The project will also compile publicly available data, including the Tax Burden on Tobacco Reports and the Tobacco Supplement to the Current Population Survey. Additionally, it will acquire and utilize NielsenIQ Consumer Panel and Retail Scanner Data.

Type of Research: Basic Research, Epidemiological (Population) Research
Project Title: *Cancer Health Disparities in the Weiss Cohort of Persons with Opioid Use Disorder. COCR26PPR013*

Stanley H. Weiss, M.D., weiss@rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

Cancer's impact is exacerbated by racial/ethnic disparities. Reducing these disparities will require quantitative analyses of cancer incidence, treatment, & mortality across types and racial/ethnic groups, including people with opioid use disorder (OUD) who have complex, unmet social and medical needs. To address this, since 1984 we have assembled and followed a cohort of ~3000 NJ OUD patients with specimens, data, and written

Pilot Grant Recipients

consent for database matching and indefinite follow-up, and laboratory research studies. The cohort includes 35% Blacks, 13% Latinos, 41% women, and 30% HIV-infected (at enrollment or follow-up). We completed genomic association analyses with cocaine use with state-of-the-art technology and will now apply this to cancer outcomes with our match to the National Death Index and now to the NJ State Cancer Registry that includes initial cancer treatment as well as diagnoses and deaths. These genomic and outcome data will be further compared to our rich data set on drug use, behaviors, and cancer-associated infections. Preliminary analyses have shown liver & lung cancer are the two most common in our cohort, not surprisingly, given high rates of smoking and liver infections. We will identify racial/ethnic differences in the impact of such characteristics on cancer risk and mortality. For example, why do Blacks have higher lung cancer rates than Whites despite smoking less on average? And what role does cannabis use (in ~50% of our subjects) play? Our research is committed to improving the medical care of persons with OUD by understanding the factors contributing to racial/ethnic disparities in cancer risk, treatment, & mortality, thereby providing actionable policy insights into the care of all those who are medically underserved. Many results will have much broader applicability, such as our examination as to whether the risk for lung cancer is increased by smoking cannabis, and if so by how much.

Type of Research: Clinical Research
Project Title: *TELEhealth Shared decision-making COaching and navigation for lung cancer screening in Primary carE (TELESCOPE) for Hispanics. COCR26PPR014*

Evelyn Arana, Dr.PH., earana@cinj.rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

Lung cancer is a major health concern in New Jersey (NJ), particularly for Hispanics, who make up 22% of the state's population. While Hispanic people in NJ tend to smoke less than non-Hispanic White individuals, they remain at high risk for lung cancer due to greater exposure to secondhand smoke and harmful environmental toxins. Lung cancer is the leading cause of cancer-related deaths among Hispanic men and the second leading cause among Hispanic women. By 2030, Hispanics are expected to make up 27% of NJ's population, yet they continue to face disparities in access to lung cancer screening (LCS). To detect lung cancer early, a screening test called low-dose computed tomography (LDCT) is recommended for people at high risk, particularly those aged 50 – 80 with a long history of heavy smoking. Before undergoing this test, doctors and patients are encouraged to have a conversation, known as shared decision-making (SDM), to discuss the benefits and risks of screening. However, many Hispanic individuals miss these screenings due to challenges such as language barriers, cultural beliefs,

family stigma, time constraints, and concerns about the healthcare system. This proposed study aims to address these challenges by adapting a lung cancer screening and patient navigation program to better serve Hispanic communities. The study aims to 1) Culturally adapt the program for Hispanic communities, 2) Test if it is feasible and acceptable for Hispanic patients and doctors, and 3) Compare it to usual care to see if it improves LDCT screening rates and quality of SDM for LCS. This approach could provide an efficient way to improve lung cancer screening for Hispanic communities in NJ, reducing health disparities and supporting better outcomes.

Type of Research: **Psychosocial and Behavioral**
Project Title: ***Development of a brief social connectedness and communication intervention for young adult couples with cancer: An application of the ORBIT model. COCR26PPR016***

Katie Darabos, Ph.D., katie.darabos@rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

For young adult couples, managing the diagnosis and treatment of cancer in one partner is a profoundly stressful experience. Relative to older adult couples coping with cancer (aged ≥40), young adult (YA) couples coping with cancer (aged 18-39) are at elevated risk for poor psychosocial outcomes. They encounter the loss of goals and dreams that older couples may have already achieved (e.g., starting or growing their family), experience more pronounced challenges (e.g., communication, loss of social networks), and are less resilient to cancer-related difficulties. Among older adult couples with cancer, interventions that focus on improving dyadic (couple) communication represent a promising approach to enhancing relationship satisfaction and well-being. Unfortunately, extremely few interventions have been developed among this young adult couple population. To ensure that the unique needs and life experiences of YA couples are met, intervention development must be optimized for them. Guided by the ORBIT model for behavioral intervention development, our formative qualitative work guides the development of intervention content focused on three areas: 1) information and resources tailored for YA couples; 2) challenges around communication and changes within their couple relationship; and 3) challenges in maintaining disruptions in their relationships with members of their social support networks (e.g., family, friends). Based on this work, the current project will develop, refine, and test the feasibility and acceptability of a novel web-based intervention for YA couples named Together We Thrive. Upon completion of this study, we expect to have a highly feasible and acceptable young adult couples-based intervention to move towards larger studies that will test effectiveness of Together We Thrive in improving relationship satisfaction and well-being among YA couples coping with cancer.

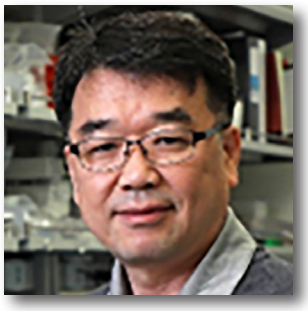
Pilot Grant Recipients

The purpose of the Pilot Grants is to provide seed funding for fundamentally sound research projects that address current priority areas.

Pilot Grant Recipients

Type of Research: Translational Research
Project Title: *A Novel Combinatorial Strategy for Enhanced CAR T Cell Therapy. COCR26PPR020*

Byungwoo Ryu, Ph.D., byungwoo.ryu@hmh-cdi.org
Affiliation: *HMH Hospitals Corporation*

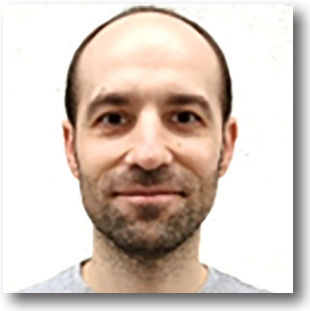


Chimeric Antigen Receptor (CAR) T cell therapy is an innovative cancer treatment that uses a patient's own immune cells to fight cancer. While it has been successful in treating blood cancers, it has not worked as well for solid tumors like pancreatic cancer. This is because solid tumors create a difficult to penetrate environment around tumor cells, called the tumor microenvironment (TME), which makes it hard for CAR T cells to reach and destroy the cancer. TME includes physical barriers of fibroblastic stromal tissues, lack of nutrients, substances, and molecules that suppress the immune system, all of which make treatment difficult. Additionally, there is a risk of adverse side effects, harming healthy tissues during therapy, a phenomenon known as on-target, off-tumor toxicity. This proposed study focuses on improving CAR T cell immunotherapy by manipulating a protein called MYC. MYC is often abnormally over-expressed in most human cancers while also contributing to their growth making the TME more hostile to tumor-killing immune cells and resistant to standard immunotherapy. Interestingly, MYC is also important for the activity and survival of T cells. Our recent study discovered a novel way to stabilize MYC using a protein complex called CoREST-complex, which prevents MYC from breaking down too quickly. Early experiments have shown that T cells engineered with a stabilized version of MYC exhibit enhanced tumor-killing capabilities. Building on these findings, we intend to test whether blocking MYC in cancer cells will slow their growth and reprogram the TME while boosting MYC in CAR T cells to enhance their strength and effectiveness against solid cancers. This project will evaluate whether this approach improves CAR T cell performance in pancreatic cancer, an aggressive solid cancer with limited treatment options, and assess whether combining these engineered CAR T cells with MYC-blocking drugs improves treatment outcomes. If successful, this strategy could help CAR T cells survive longer, penetrate solid tumors more effectively, and kill cancer cells more efficiently, offering new hope for patients with hard-to-treat cancers like pancreatic cancer.

Postdoctoral Research Fellowship Recipients

Type of Research: Basic Research; Translational Research
Project Title: *Modelling Hepatosplenic T-cell Lymphoma in vivo. COCR26PDF003*

Rodrigo Prieto Bermejo, Ph.D., rp1397@cinj.rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*



Hepatosplenic T-cell Lymphoma (HSTL) is a very rare but fatal disease without known cures. In order to find potential treatments for this disease, we need to have better models that allow us to investigate HSTL in its normal context in vivo. Thus, here we propose to develop the first-ever model of primary HSTL in mice by using a novel and complex mouse model. If we succeed in generating these models, they could be used to dramatically advance basic research in HSTL, which would lead to better treatments in the future.

Type of Research: Basic Research, Translational Research
Project Title: *Impact of physical activity on cancer progression and response to checkpoint inhibitor-based immunotherapy. COCR26PDF004*

Gabriela Reyes Castellanos, Ph.D., gr488@cinj.rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

Besides general health benefits, it has been shown that regular physical activity (PA) can lower the risk of cancer, improve survival rates, and reduce the probabilities of recurrence. Likewise, research in mouse models further support these findings, showing that PA can slow tumor growth and dissemination by several mechanisms, mainly effects on cancer metabolism and the host immune system. However, a deeper understanding of the effects of PA on cancer is needed, as well as its impact across different cancer types. Our aim is to investigate the impact of regular PA on cancer progression. Specifically, we seek to study the adaptive responses induced by PA, with a focus on the immune system. We hypothesize that PA can enhance the immune system's ability to detect and attack cancer cells. Moreover, PA could improve immunotherapy efficacy. To test our hypothesis, we work with genetically modified mouse models that mimic human cancers, mainly lymphoma and lung cancer, and we have implemented voluntary wheel running, which is a non-stress activity that closely resembles wild-life conditions, thus allowing us to perform long-term studies. Using cutting

Postdoctoral Research Fellowship Recipients

NJCCR offers Pre- and Post-Doctoral Fellowships to trainees at New Jersey non-profit research institutions with formally established and active graduate research programs.

Postdoctoral Research Fellowship Recipients

edge techniques for the analysis of immune cells, inflammatory markers and metabolites, we seek to unveil the molecular mechanisms underlying the PA-tumor-immune system interactions. This research has the potential to redefine the role of PA in cancer, both in a preventive context or as a complementary strategy to improve cancer treatments. In addition, these findings could be important for populations with genetic predispositions to cancer, providing a non-invasive and accessible intervention to reduce risk. Ultimately, this research opens the door to more personalized cancer treatments, where PA could play an important part in raising the efficiency of the standard of care.

Type of Research: **Translational Research**
Project Title: ***Elucidating the Mechanisms by Which the Ketogenic Diet Regulates Tumor Growth and Metastasis in KRAS-Driven NSCLC. COCR26PDF009***

Hyungsin Kim, Ph.D., hk970@cinj.rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

Lung cancer is one of the most prevalent cancers in the United States and a leading cause of cancer-related deaths. Due to its aggressive nature, the 5-year survival rate was reported to be only 19% as of 2021. Non-small cell lung cancer (NSCLC) with KRAS mutations is a deadly disease, and its behavior differs depending on co mutations such as TP53 (KP) or LKB1 (KL). We studied the effects of the ketogenic diet, which reduces carbohydrate intake and uses fat as the main energy source, on these cancer types. We found that the diet slowed tumor growth and reduced metastasis in KP tumors but unexpectedly promoted tumor progression and metastasis in KL tumors. In KP tumors, the ketogenic diet reshaped the tumor microenvironment by increasing tumor fighting M1 macrophages and reducing tumor promoting M2 macrophages, and this effect was independent of T cells and natural killer cells. Moreover, the ketogenic diet improved the response of KP tumors to anti PD1 immunotherapy, suggesting it could be used as a supportive treatment. These findings show that the ketogenic diet does not have the same effect in all KRAS mutant lung cancers and may benefit KP patients while posing risks for KL patients, highlighting the need for precision approaches based on tumor genetics. Since this work was performed in mouse models, further clinical research is needed before applying it to patients.

Postdoctoral Research Fellowship Recipients

Type of Research: Basic Research
Project Title: ***Effects of dietary amino acid perturbations on cancer progression and susceptibility to immune checkpoint blockade. COCR26PDF010***

Edisa Pirani, Ph.D., ep815@cinj.rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

Cancer is one of the leading causes of death in New Jersey, and while new immune-based treatments have helped many patients, they do not work for everyone. One possible reason is the way cancer cells use nutrients. Tumors need a constant supply of building blocks, such as the amino acids tryptophan and arginine, to grow and avoid attack by the immune system. These same nutrients are also essential for immune cells, especially T cells, which are key in fighting cancer. This project will study how changes in the availability of these amino acids in the diet affect both cancer growth and the body's response to immunotherapy. Using models of aggressive cancers, we will test whether restricting or adding these nutrients can make treatments more effective. The findings may point to new, non-invasive strategies, such as dietary changes, that improve cancer therapy. In addition, this research may help address cancer-related malnutrition and weight loss, conditions that weaken patients and reduce their ability to tolerate treatment. By understanding how tumors compete with the body for nutrients, we aim to find better ways to support patients' health during therapy and improve their quality of life.

Type of Research: Basic Research
Project Title: ***Age related DNA methylation changes cause epigenetic mosaicism resulting in pre neoplasia in colorectal cancer. COCR26PDF022***

Himani Vaidya, Ph.D., hvaidya@coriell.org
Affiliation: *Coriell Institute for Medical Research*

Colorectal cancer (CRC) develops due to genetic alterations and changes in gene regulation within colon cells, influenced by both aging and specific mutations. Our study seeks to unravel the relationship between aging, genetic mutations, and the early stages of CRC development. To explore these connections, we employ advanced techniques such as single-cell Assay for Transposase-Accessible Chromatin using sequencing (scATAC-seq) and single-cell RNA sequencing (scRNA-seq), allowing us to investigate how DNA structure and gene activity evolve as

Postdoctoral Research Fellowship Recipients

mice age. Analyzing colorectal patient samples helps us identify genes undergoing specific alterations linked to colon cancer. By comparing datasets, we gain insights into how genes age in mice and which ones undergo changes in CRC. Additionally, we use mouse models and cutting-edge CRISPR technology, including organoid cultures, to replicate and study the gradual process leading to tumor formation. This comprehensive approach enhances our understanding of the complex interplay between aging, genetic mutations, and CRC development, ultimately aiming to advance knowledge of this disease.

Type of Research: **Basic Research**
Project Title: ***Developing Therapeutic Approaches for Chemobrain. COCR26PDF037***

Sang Hoon Kim, Ph.D., sanghoon.kim l@rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

Chemotherapy saves lives, but many survivors struggle with "chemobrain," which causes problems with memory, focus, and daily life. Unfortunately, there are currently no effective medications to treat this condition. Exercise has been shown to help protect the brain, but not all patients are able to be physically active because of pain or other health issues. My research seeks to understand how exercise benefits the brain and to identify key molecules that could be targeted with safe, existing drugs. This work aims to develop new treatment options that mimic the protective effects of exercise, ultimately improving the quality of life for cancer survivors.

Pre-doctoral Research Fellowship Recipients

Type of Research: Basic Research
Project Title: ***Human cytomegalovirus microenvironment as a determinant of viral spread and pathogenesis. COCR26PRF001***

James Kostas, jk5605@princeton.edu
Affiliation: *The Trustees of Princeton University*

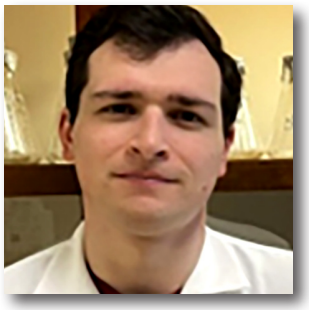
Scientists estimate that viral infections cause up to 20% of human cancers. One of the viruses linked to cancer is the herpesvirus human cytomegalovirus (HCMV), from the family of viruses that causes cold sores, mono, and chicken pox. HCMV is so widespread that it infects 80% of people worldwide. And once a person is infected, the virus stays in the body for life. While HCMV usually stays dormant and causes few issues for people with healthy immune systems, researchers are finding increasingly that HCMV infection can support the development of cancer in all people. Some researchers even propose that HCMV can directly make a cell cancerous, but exactly how the virus can carry out these roles are active areas of research. Research from my colleagues and myself has shown that uninfected cells next to sites of HCMV infection look very strange. Despite being uninfected themselves, these nearby cells have abnormal growth cycles, causing them to become stuck in a fragile state. My results show that this fragile state makes them easier to be infected by the virus, which I believe may enhance HCMV's ability to spread within the body. These cells near an infection also show defects with how their DNA is organized. This could lead to DNA damage–a key starting point for the formation of cancer and possibly explaining how HCMV can support cancer development or even cause cancer itself. My ongoing research aims to determine the finer details of how these changes caused by HCMV infection might help the virus spread and how they may contribute to cancer development. Understanding these processes is important because of how many people are infected with HCMV and because it could reveal new ways to prevent or treat cancers associated with HCMV.

Pre-doctoral Research Fellowships continued on next page

Pre-doctoral Research Fellowship Recipients

Type of Research: Basic Research
Project Title: ***Investigation of BTNL9 as a novel regulator of T cells in ovarian cancer. COCR26PRF004***

Christopher Alcott, ca674@gsbs.rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*



Ovarian cancer is the deadliest gynecologic cancer and is among the top five deadliest of all cancers for women in the US. The American Cancer Society estimates that approximately 20,000 women in the US will be diagnosed with ovarian cancer in 2025 and that over half of that number will die from the disease. In 2020, the last year in which state-level incidence data are available from the CDC, New Jersey specifically had nearly double the national rate of ovarian cancer diagnoses as well as an above-average number of ovarian cancer deaths. As devastating as ovarian cancer is for many women in the US, and even more so for women in New Jersey, effective and comprehensive treatments are vital. Surgery is generally performed to remove the tumor, and chemotherapy is administered to kill any remaining cells, however ovarian cancer patients often relapse and show overall poor five-year survival. This relapse is usually attributed to a portion of cancer cells that are resistant to chemotherapy and continue to grow despite intense treatments. There is, however, a newer class of treatments that holds great potential in cancer treatment: immunotherapy. Immunotherapy helps guide the immune system to kill tumors and comes without the harsh side effects of chemotherapy. A type of immune cell called a T cell is known to be especially good at recognizing and killing cancer cells, and immunotherapies that enhance T cells have been effective in treating malignancies like lung and skin cancer. Unfortunately, these same immunotherapies have not achieved success yet in ovarian cancer patients. Our bodies naturally possess many signals to regulate T cells and ovarian tumors are particularly adept at using these signals to prevent T cell activity. This proposal aims to better understand what signals decide whether or not a T cell infiltrates and kills ovarian tumors, and develop novel immunotherapies based on these signals.

Pre-doctoral Research Fellowship Recipients

Type of Research: Basic Research
Project Title: ***Tripartite Motif-Containing Protein 6 in colorectal cancer. COCR26PRF006***

Yiyun Shen, ys941@gsbs.rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

Colorectal cancer is the third most commonly diagnosed cancer and the second leading cancer-related deaths in the United States. A better understanding of the molecular mechanisms of colorectal cancer will provide novel targets and strategies to effectively treat colorectal cancer, which are urgently needed in clinical practice. Previous studies and our preliminary studies suggest that TRIM6 protein plays a critical role in regulating the progression of colorectal cancer and affecting the clinical outcomes of cancer patients. However, the precise role and mechanism of TRIM6 in colorectal cancer remain poorly understood, which hinders the development of effective therapeutic strategies for colorectal cancer. Based on our preliminary results, we hypothesize that TRIM6 plays a critical role in colorectal cancer, which can be targeted for colorectal cancer treatment. To test our hypothesis, in this proposed study, we will determine the role and mechanism of TRIM6 in colorectal cancer using various mouse colorectal tumor models and test the new therapeutic targets and strategies to treat colorectal cancer. The goal of this proposed study is to reveal the novel role and mechanism of TRIM6 in colorectal cancer and yield novel strategies to treat CRC. We anticipate that this study will establish the role of TRIM6 in colorectal cancer and reveal the novel and critical underlying mechanism of TRIM6 in colorectal cancer. We anticipate that the results from this proposed project will deepen our understanding of the molecular mechanism of colorectal cancer and provide a strong rationale and foundation to develop novel and effective therapeutic targets and strategies for colorectal cancer.

Pre-doctoral Research Fellowship Recipients

Type of Research: **Basic Research**
Project Title: ***Targeting taurine-induced cisplatin resistance. COCR26PRF007***

Emily Hyde, erh111@gsbs.rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

Epithelial ovarian cancer (OC) is the deadliest type of gynecological cancer. While most patients initially respond to standard treatments, including surgery and chemotherapy, up to 80% will experience relapse that is unaffected by treatment. This recurrence is the main cause of the high mortality rate of ovarian cancer and remains a major clinical problem. Despite efforts to find better treatments, survival rates have not improved significantly in decades, highlighting the need for new approaches. Taurine is a naturally occurring substance found in all human tissues, including in ovarian cancer cells. In our previous work, we found that taurine levels are high in ovarian cancer cells, particularly those that have spread into the abdomen. In our lab models, increasing taurine levels helped cancer cells become resistant to the chemotherapy drug cisplatin, reducing its effectiveness in causing cancer cell death. These data suggest that taurine might help protect cancer cells from the cisplatin, which could explain how the cells develop drug resistance. The exact way taurine helps ovarian cancer cells survive chemotherapy is still unclear. We believe that increased taurine levels may change how these cells utilize different biomolecules within the cells to make themselves more resistant. In other words, taurine may "reprogram" the cell to become less sensitive to chemotherapy. We plan to test existing FDA-approved drugs to reverse this "reprogramming," hoping to develop new strategies to overcome drug resistance in ovarian cancer. Ultimately, this will offer real hope for ovarian cancer patients, making the disease treatable, not terminal.

Type of Research: **Basic Research; Translational Research**
Project Title: ***Investigating the Role of CAR-NK Immunological Synapse in Immunotherapy. COCR26PRF011***

Jong Hyun Cho, jc2674@gsbs.rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

Chimeric Antigen Receptor (CAR) immunotherapy, which involves engineering immune cells to recognize and attack cancer, has shown great success in treating blood cancers. However, reliable methods to predict and measure the effectiveness of these modified cells are still lacking. Our lab has developed novel CAR-engineered natural killer (CAR-NK) cells that target the CD147 antigen in hepatocellular carcinoma (HCC), a common and

aggressive form of liver cancer. While these CAR-NK cells have shown promising tumor reduction in mouse models, the mechanisms of cancer cell killing and ways to evaluate their effectiveness remain unclear. Recent studies suggest that the quality of the immunological synapse (IS), the contact point between CAR-NK cells and tumor cells, is critical for their antitumor activity. In this study, we aim to determine whether IS quality can serve as a biomarker for CAR-NK cell efficacy. We will test CAR constructs with different activation strengths, analyze IS formation and molecule distribution, and assess how tumor-related suppressive factors impact IS quality and CAR-NK cell function. These findings may offer new insights into CAR-NK cell mechanisms and provide novel tools for evaluating and improving cancer immunotherapy.

Type of Research: **Basic Research; Translational Research**
Project Title: ***Role of CD103 in Reactivation and Maintenance of Small Intestine CD8+ Trm. COCR26PRF012***

Corey Gallen cg961@gsbs.rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

The body's barrier surfaces, including the intestine, are home to a large number of immune cells that are integral in protecting the body from outside harms. One important type of immune cell, called tissue-resident memory T cells (Trm), resides predominately in these tissues where they help maintain health and rapidly respond to infection. The cell surface receptor CD103 is often found on Trm cells, but its role in Trm function has not been fully elucidated. Using a mouse model of intestinal infection with Yersinia pseudotuberculosis (Yptb), our preliminary results indicated Trm subsets with or without CD103 differ in their activity, suggesting CD103 may influence how Trm cells respond to threats. Our data revealed that CD103 interactions with its partner E-cadherin strengthened signaling in Trm cells and boosts immune responses, including cytokine production. Blocking this interaction in mice reduced Trm activity, further supporting the hypothesis that CD103 positively regulates Trm cells. To explore this in more detail, we used cells lacking CD103 to study its impact on Trm cells. Without CD103, Trm cells showed reduced activity in key signaling pathways, suggesting this protein influences their differentiation and long-term function. We also developed a new mouse model that allows CD103 to be deleted at specific time points, helping us determine whether CD103 is required for the initial establishment of Trm or for their maintenance over time. Together, this work shows that CD103 plays an important role in shaping Trm responses in the intestine. Understanding how CD103 controls immune cells will help us learn how to better harness them to fight not only infections but also other diseases affecting barrier tissues, such as cancer.

Pre-doctoral Research Fellowship Recipients

Pre-doctoral Research Fellowship Recipients

Type of Research: Basic Research
Project Title: ***Genomic and Epigenetic Effects of Tai Chi Qigong in Older Male Cancer Survivors with Fatigue. COCR26PRF015***

Samuel Tundealao, st1220@sph.rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

Fatigue is a common and persistent symptom reported by many older male cancer survivors, yet it remains poorly understood by patients and healthcare providers. Tai Chi Qigong (TCQ), a gentle mind-body exercise that combines slow movements, focused breathing, and meditation, has shown promise in helping to reduce fatigue and improve overall well-being. This study aims to uncover how TCQ might influence biological processes associated with fatigue by analyzing changes in gene expression among cancer survivors. Specifically, the study will compare the effects of TCQ to an intensity-matched form of exercise (EIM) and usual care, looking at how these interventions impact gene expression patterns related to fatigue. By examining the connections between fatigue and epigenetic changes related to TCQ and EIM, this research can provide new insights into the underlying mechanisms of fatigue and how mind-body therapies like TCQ may modulate it. These findings could lead to better, more targeted strategies for improving the quality of life for older male cancer survivors.

Type of Research: Basic Research
Project Title: ***Characterization of the human voltage sensing phosphatase specificity and regulation of the AKT/MTOR pathway. COCR26PRF016***

Brandi Sweet, sweetb95@rowan.edu
Affiliation: *Rowan University*

Metastatic melanoma is the deadliest form of skin cancer. Since current treatments are becoming less effective, the search for new therapeutic strategies is critical. Cancer develops when cells grow uncontrollably and spread throughout the body, a process known as metastasis. Under normal conditions, our bodies maintain strict control over cell behavior through internal signals that keep cellular growth and movement in balance. These signaling pathways are regulated by small molecules, such as proteins, that each have a specific role. My research focuses on a protein called the voltage-sensing phosphatase

(VSP), which helps regulate these internal signals to maintain cellular balance. What makes VSP unique is that it responds to electrical changes in the cellular environment, translating electrical signals into chemical communication within the cell. This exceptional function could represent an unexplored pathway for controlling cancer cell behavior. Interestingly, clinical studies have shown elevated levels of VSP in many forms of cancer, including melanoma, pointing to a potential role in the disruption of normal signaling that keeps cancers in check. These studies also suggests that human VSP may promote the growth and spread of melanoma cells. Yet when VSP is introduced into cancer-like breast tissue cells, it appears to suppress their abnormal growth and movement, suggesting a complex role in cancer biology that we don't fully understand. We still have much to learn about how electrical signals influence cell growth in melanomas, and studying VSP may open the door to an entirely new class of cancer treatments.

Type of Research: **Basic Research**
Project Title: ***Exploring the structure and function of miRNA binding sites regulating the PACER/COX-2/PGE2 signaling axis in lung adenocarcinoma. COCR26PRF019***

Samira Bell, sb2258@gsbs.rutgers.edu

Affiliation: *Rutgers Biomedical and Health Sciences*

Cancer treatments often come with harmful side effects, and scientists are working to find better, more targeted therapies. One promising approach involves studying molecules called RNA, which play important roles in controlling cell behavior. In particular, we're interested in specific types of RNA called long noncoding RNAs (lncRNAs) and microRNAs (miRNAs), which are involved in regulating genes and cell processes. Cyclooxygenase-2 (COX-2) is a protein that's linked to inflammation and cancer, and understanding how it's controlled by lncRNAs and miRNAs could lead to new cancer treatments.

Our research focuses on a specific lncRNA called PACER and a miRNA called miR-423-3p, which are both involved in controlling COX-2 in lung cancer. We've found that PACER is overactive in lung cancer cells, promoting their growth and spread. Using advanced tools, we've identified a part of PACER where miR-423-3p binds, potentially affecting its activity. We also found that there is a version of PACER in mice, which could be helpful for establishing an animal model to study this inflammatory network in. Our goal is to understand how miR-423-3p and PACER interact and how this interaction affects lung cancer. To do this, we'll perform experiments in lung cancer cells to see how miR-423-3p influences PACER and COX-2. We'll also investigate the structure of PACER in humans and in mice to understand how it works at a molecular level. By uncovering these details, we hope

Pre-doctoral Research Fellowship Recipients

to develop new treatments that target the specific molecules involved in lung cancer, potentially leading to more effective and less harmful therapies. Our research could pave the way for personalized treatments tailored to each patient's unique genetic makeup, and for the production of more efficient and targeted drug therapies with reduced harmful side-effects.

Type of Research: **Basic Research; Translational Research**
Project Title: ***Investigating Phosphatidylserine Signaling as Cancer Immune Evasion Strategy. COCR26PRF022***

Fnu Ahmed Aquib, fa453@gsbs.rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

Macrophages are immune cells that act as the body's garbage disposers and defenders. Previous work has shown that when macrophages clear away dead cells, the result is often immune suppression. This is necessary because the body must silently handle the turnover of millions of dying cells each day without triggering severe inflammation. In healthy tissues, this quiet process, known as efferocytosis, keeps the immune system calm and prevents damage. However, cancers—well known for blocking immune surveillance—are now believed to exploit efferocytosis as one of their strategies for immune evasion. Efferocytosis relies on molecular "flags" that mark cells for clearance. One of the most important of these is phosphatidylserine (PS). Normally hidden inside healthy cells, PS flips outward when a cell is dying, signaling macrophages to remove it without triggering alarm. Cancer cells hijack this pathway by displaying PS on their surface, whether in stressed live cells or dying ones. This deceptive signal tricks macrophages into tolerating the tumor instead of attacking it, reprogramming them into supporters of cancer growth. In our studies, we are investigating how efferocytosis and PS contribute to immune suppression in macrophages. We show that contact with PS-exposing cancer cells dampens macrophage inflammatory activity and shifts them toward a pro-tumor state. By uncovering how efferocytosis and PS signals suppress immunity, our work highlights strategies to block these deceptive cues, restore macrophage function, and re-arm the immune system against cancer.

Joel C. Cantor, Sc.D.

Keynote Speaker

Joel C. Cantor (Sc.D., Johns Hopkins University) is a Distinguished Professor of Public Policy and the founding Director of the Center for State Health Policy at Rutgers University in New Brunswick, New Jersey. Established 1999, the Center is a leader in health policy in research and development nationally, with a special focus on informing policy in New Jersey. Dr. Cantor has published widely in the health services and policy literature on innovations in health service delivery for high-need populations and the regulation of health insurance markets. He serves frequently as an advisor on health policy matters to New Jersey state government and was the 2006 recipient of the Rutgers University President's Award for Research in Service to New Jersey.

Dr. Cantor currently leads a major study funded by the National Institutes of Health (R01MD015261) examining the contribution of homelessness to racial/ethnic and geographic disparities in health services outcomes and how permanent supportive housing can mitigate those disparities. He also is co-principal investigator of the New Jersey Population Health Cohort Study, a major new investigation of the effects of stress and resilience on population health and health equity.

Prior to joining Rutgers in 1999, Dr. Cantor served as director of research at the United Hospital Fund of New York and director of evaluation research at the Robert Wood Johnson Foundation. He received his doctorate in health policy and management from the Johns Hopkins University, School of Public Health in 1988, and was elected a fellow of the National Academy of Social Insurance in 2019 and of Academy Health in 1996.

Panel Members

Panel Discussion:

Employment Opportunities, Career Pathways & Trends in Cancer Research

Elisa Bandera, M.D., Ph.D.

Professor & Chief of Cancer Epidemiology and Health Outcomes, CINJ

Dr. Elisa Bandera is Professor and Chief of Cancer Epidemiology and Health Outcomes, Co-Leader of the Cancer Prevention and Control Program and Director of the Cancer Prevention and Outcomes Data Support (CPODS) Shared Resource at the Rutgers Cancer Institute of New Jersey. She is also the Unilever Endowed Chair in Nutrition and Cancer Prevention and Professor of Medicine, Robert Wood Johnson Medical School, Rutgers University. Her major research interests include the impact of obesity and body composition and related factors on breast and ovarian cancer risk, treatment outcomes, survivorship and prognosis after a cancer diagnosis, with a focus on cancer health disparities.

Dr. Bandera has served as Principal Investigator in several epidemiologic cohort studies, including the Women's Circle of Health Follow-up Study and the New Jersey Breast Cancer Survivors Study, both studies aim to evaluate multi-level factors impacting survivorship and prognosis in minoritized and medically underserved women, including Black/African American and Hispanic women. She has also served in numerous advisory boards and expert panels for several organizations, including the American Cancer Society, the National Cancer Institute, the International Agency for Research on Cancer, the American Institute for Cancer Research (AICR) and the World Cancer Research Fund International (WCRF). Dr. Bandera has served as ad hoc reviewer for multiple grant review panels for NIH and other agencies, and a former standing member of the NIH Study Section Cancer Heart, and Sleep Epidemiology Panel B (CHSB) and the National Cancer Institute Initial Review Group Subcommittee J for Population and Patient-Oriented Training. She has also been a member of the Breast Cancer Prevention Partners' Science Advisory Panel since 2013 and contributed substantially to public policy by playing a major role in the development of nutritional guidelines for cancer prevention and survival at a national and global scale.

For over 10 years she served as a member of the International Expert Panel for the WCRF/AICR Continuous Update Project and the WCRF/AICR Third Expert Report on Diet, Nutrition, Physical Activity and Cancer: A Global Perspective, released in May 2018. This publication provided the most comprehensive review of the epidemiologic literature relating nutrition, physical activity and cancer published to date and issued dietary guidelines for cancer prevention.

Dr. Bandera has been involved with the American Cancer Society's Guidelines on Nutrition and Physical Activity for Cancer Prevention and Survival Advisory Committees since 2006. She has also served as Chair of the Lifestyle Behaviors, Energy Balance and Chemoprevention Special Interest Group of the American Society of Preventive Oncology (ASPO) and co-chaired the 2021 ASPO Annual Meeting: Health Equity, Culture, and Cancer. Dr. Bandera has continuously devoted significant effort to mentoring the next generation of cancer researchers, including graduate students, postdoctoral fellows, and junior faculty, and has served as mentor in many NIH career development (K) grants over the years. Her commitment was recognized with the Rutgers Biomedical Health Sciences Chancellor Distinguished Mentor Award in 2023. Dr. Bandera has also continuously devoted considerable efforts to mentoring the next generation of cancer researchers, including graduate students, postdoctoral fellows, and junior faculty, serving as mentor in many NIH K grants (career development grants) over the years. Her track record was recognized with Rutgers Biomedical Health Sciences Chancellor Distinguished Mentor Award in 2023.

Ian McLaughlin, Ph.D.

Vice President, Government Affairs, BioNJ

A life scientist by training, Ian McLaughlin most recently served as a Policy Analyst in the New Jersey Assembly Majority Office, where he served as a Committee Aide to the Assembly Health and Aging & Senior Services Committee. During his tenure with the New Jersey Assembly Majority Office, he was responsible for analyzing legislation, department budgets and regulations; preparing briefing documents for legislative leadership; and working closely with advocates, lobbyists and stakeholders in preparing and delivering testimony before Assembly committees. Prior to joining the Assembly Majority Office, Ian was a Science and Politics Fellow at the Eagleton Institute of Politics. His scientific research focused on understanding the neurobiology of addiction and psychiatric conditions with the goal of identifying novel druggable CNS targets. While in graduate school at the University of Pennsylvania, he served as a Vice President of the Penn Biotech Group, which enables teams of MBA, M.D. and Ph.D. candidates to consult for companies in biotech, pharma, health care analytics and venture capital. As Vice President of Government Affairs, Ian uses his scientific acumen, analytical mind and experience in working with the New Jersey Legislature to lead BioNJ's advocacy and public policy efforts to help our members help patients.

Panel Members

Antoinette M. Stroup, Ph.D.
Director of Cancer Epidemiology Services (CES)
New Jersey State Cancer Registry

Antoinette Stroup earned her B.S. and M.S. degrees from the University of Utah in Salt Lake City. She went on to earn her Ph.D. from the University of California, Berkeley in Epidemiology. Dr. Stroup is a Professor of Cancer Epidemiology in the Department of Biostatistics and Epidemiology at the Rutgers School of Public Health. She is the Director of the New Jersey State Cancer Registry at the New Jersey Department of Health, overseeing all administrative, operational, and research of the State's population-based cancer surveillance system; and the Principal Investigator for New Jersey's NCI SEER Program contract. Her current research projects include multi-registry collaborations studying risk and outcomes among Latino and Asian race and ethnic subgroups with breast, colorectal and cervical cancer and African American men with prostate cancer. Dr. Stroup is also the Assistant Director of Research and Catchment Data at the Rutgers Cancer Institute Cancer Health Equity Center of Excellence, where she oversees a team charged with evaluating the Center's catchment area cancer burden and promoting the integration of catchment area priorities into research. Finally, Dr. Stroup teaches an undergraduate course in Cancer Surveillance at the Edward J. Bloustein School of Planning and Public Policy, which is a core requirement for their new undergraduate certificate in Cancer Surveillance and Epidemiology.

Panel Members

Eileen White, Ph.D.
Deputy Director and Chief Scientific Officer at the Rutgers Cancer Institute at Rutgers University
Associate Director of the Ludwig Princeton Branch of the Ludwig Institute for Cancer Research at Princeton University

Eileen White, Ph.D., is a cancer biologist known for her work establishing that a DNA tumor virus oncogene functions by inhibiting programmed cell death by apoptosis and is a homologue of the human BCL-2 oncogene. She is also known for establishing that tumor cells induce intracellular nutrient scavenging by autophagy, which promotes their metabolism, growth, survival, and malignancy. These findings informed the means to target the apoptosis and autophagy pathways for cancer therapy. Eileen is Deputy Director and Chief Scientific Officer at the Rutgers Cancer Institute at Rutgers University, and Associate Director of the Ludwig Princeton Branch of the Ludwig Institute for Cancer Research at Princeton University. She is also the Lead PI for the Cancer Research United Kingdom/United States National Cancer Institute Cancer Grand Challenge grant to address the mechanisms causing cancer cachexia through the Cancer Cachexia Action Network (Team CANCAN). Amongst Eileen's honors are election to the US National Academy of Sciences and the American Academy of Arts and Sciences, and she is an elected fellow of the American Association for the Advancement of Science, the American Academy of Microbiology, and the American Association for Cancer Research Academy.

Meet the Commission Members

Kenneth Adler, M.D.

Chair

Dr. Adler specializes in Hematology/Oncology, with a special interest in benign and malignant hematology and geriatric oncology. He is an attending physician at Morristown Medical Center. He serves as Co-chair of the American Society of Hematology Practice Partnership and is a fellow of the American College of Physicians, a member of the American Society of Clinical Oncology and the American Society of Hematology. Dr. Adler has been awarded several outstanding honors throughout his career. In 2014 he received the prestigious Augustus Stone Award for his voluntary service to the Morristown Medical Center, and in 2017 he was the Medical Honoree of the American Cancer Society for Northwest New Jersey. Most recently in 2019, he was honored by the Summit Medical Group at their Annual Gala for his community service.

Generosa Grana, M.D., F.A.C.P.

Vice-Chair

Dr. Grana is the Director of the MD Anderson Cancer Center at Cooper. She is also a Professor of Medicine at Cooper Medical School of Rowan University and an adjunct professor of cancer Medicine at the University of Texas MD Anderson Cancer Center. Dr. Grana completed her fellowship in Hematology and Oncology at Fox Chase Cancer Center and Temple University in Philadelphia where she also completed a Postdoctoral Fellowship in Preventive Oncology. Dr. Grana's clinical and research endeavors at Cooper have focused on breast cancer, cancer genetics, and community outreach interventions aimed at underserved populations. She has received numerous awards including the American Cancer Society Silver Chalice Award and the Susan G. Komen for the cure "Light of Life" Award.

Kathleen Scotto, Ph.D.

Dr. Scotto is currently Vice-Chancellor for Research and Research Training, Rutgers Biomedical and Health Sciences, and Dean for the School of Graduate Studies, Rutgers, The State University of New Jersey. She received her Ph.D. from the Cornell Graduate School of Medical Sciences. Prior to joining Rutgers, she served as an Associate Professor of Molecular Pharmacology and Experimental Therapeutics at Memorial Sloan Kettering Cancer Center and a Professor with tenure at the Fox Chase Cancer Center. In addition to her administrative roles, Dr. Scotto maintains an active laboratory at Rutgers studying the Role of ABC Transporters in Tumor Survival and Treatment Response.

Wendy Budin, Ph.D., R.N.-B.C., F.A.A.N.

Dr. Budin is Professor and Associate Dean for Faculty Affairs in the School of Nursing, Rutgers University. Previously, she was the Director of Nursing Research at NYU Langone Medical Center and faculty at NYU College of Nursing. Dr. Budin is involved in an ongoing program of research on psychosocial adjustment to breast cancer. In 2019, she coauthored a book chapter entitled "Theoretical Frameworks and Philosophies of Care," in Current Trends in Oncology Nursing— Second Edition. Dr. Budin is a Fellow in the American Academy of Nursing and the New York Academy of Medicine (NYAM) for her achievements. She received the NJ Governor's Award for Nursing Research and Distinguished Alumnae Awards from the NYU College of Nursing and Seton Hall University, and in 2018 she received the March of Dimes, Nurse of the Year Award for Research.

Michele Lyne Donato, M.D., F.A.C.P., C.P.E., M.B.A.

Dr. Donato is a leader in the care of patients who need stem cell transplantation for cancerous and noncancerous diseases as well as those receiving immunotherapy. She was at the forefront of the development of photopheresis, a technique used to treat patients with chronic graft-versus-host disease (a potential complication of bone marrow and stem cell transplantation in which cells from the donor attack tissues of the recipient). At John Theurer Cancer Center Hackensack University Medical Center, she leads one of the region's largest bone marrow and stem cell transplant programs and one of the world's largest photopheresis centers.

Dr. Donato is presently the Chief of the Stem Cell Transplantation and Cellular Therapy Program, at Hackensack University Medical Center, and is affiliated with Jersey Shore University Medical Center at Hackensack Meridian Health. She received her medical degree from McGill University Faculty of Medicine and has been in practice for more than 20 years. She is actively involved in blood stem cell transplantation research. She has been awarded the Medal of the Governor General of Canada for the Highest Academic Standing, the Denis Dussiaume Award for Academic Excellence, and the McGill University J.W. McConnell Entrance Award

Li Li, Ph.D.

Dr. Li is currently the Executive Director at the Novartis Institute for Biomedical Research, where he has worked for over 17 years. He received his Ph.D., in Toxicology from the University of Texas-Houston School of Public Health. He is a member of the Society of Toxicology and a Board-certified Toxicologist. He is a recipient of numerous awards, most recently the Team Innovation Award for Novartis. In addition, he has co-authored many articles on toxicology innovation in research journals.

Meet the Commission Members

NJCCR consists of dedicated volunteer members that are involved, both statewide and nationally, in the field of cancer.

Meet the Commission Members

Jane Flint, Ph.D.

Dr. Flint is a Professor Emerita of Molecular Biology at Princeton University. Dr. Flint's research focused on investigation of the molecular mechanism by which viral gene products modulate host cell pathways and antiviral defenses to allow efficient reproduction in normal human cells of adenoviruses, viruses that are widely used in such therapeutic applications as gene transfer and cancer treatment. Her service to the scientific community includes membership on various editorial boards, several NIH study sections and the NIH Recombinant DNA Advisory Committee. She also is a founding author of the acclaimed textbook "Principles of Virology," now in its fifth edition.

Uta Steinhauser, M.P.H.

NJDOH Commissioner's Designee

Uta Steinhauser is a career public health professional with over 20 years of experience in the field. Ms. Steinhauser has served as Community Health and Wellness Unit's (CHWU) evaluation and surveillance lead since 2010. She provides oversight to all CHWU evaluation and surveillance deliverables, including the completion of evaluation and performance measurement plans and their subsequent federal and public reports. She serves as an SME for chronic disease data, has guided the planning of multiple chronic disease related plans and provides direction to chronic disease programs in selection and monitoring of appropriate data sources, evidence-based interventions and overall measurement of outcomes. She holds a Master of Public Health degree from Rutgers University, School of Public Health with a dual concentration in epidemiology and health education.

Meet the Commission Members

Christine Schell, M.P.A.

NJDEP Commissioner's Designee

Christine Schell is a 30+ year veteran of the New Jersey Department of Environmental Protection (NJDEP) and currently manages the NJDEP's Office of Environmental and Public Health Analysis. In partnership with the New Jersey Department of Health, Christine jointly coordinates New Jersey's Environmental Public Health Tracking Program through a CDC grant, and oversees the development, implementation and dissemination of data analyses and visualizations tools designed to integrate public health concerns into local and state decision making, planning, and the implementation of meaning actions on a broad array of issues, including environmental justice, climate resilience, and sustainability. In addition to representing the NJDEP's Commissioner on the NJCCR, she serves as the Commissioner's designee as ex-officio on the NJ One Health Taskforce. Ms. Schell participates on the Health Equity Workgroup of the NJ State Cancer Coalition and leads the Extreme Heat Coordinated Communications Workgroup for the NJ Interagency Council on Climate Resilience. Christine holds a Bachelor of Science in Environmental Science and a Master's in Public Administration, both from Rutgers University. She is a Certified Public Manager and member of the Pi Alpha National Honor Society for Public Administration

NJCCR Pediatric Cancer Research Advisory Group

Aubrey Reichard-Eline, *Chair*
Rutgers Cancer Institute of New Jersey

Peter Cole, M.D., *Co-Chair*
Rutgers Cancer Institute of New Jersey

Daniel Notterman, M.A., M.D.
Princeton University

Paulette Forbes, Ph.D., M.P.H., A.P.N.-B.C.
Cooper Medical School of Rowan University

Rafat Ahmed, M.D.
Rutgers Cancer Institute of New Jersey

Alfred Gillio, M.D.
Hackensack University Medical Center

Index of Authors

Alcott, Christopher 24
Aquib, Fnu Ahmed 30
Arana, Evelyn 16
Bell, Samira 29
Bermejo, Rodrigo Prieto 19
Castellanos, Gabriela Reyes 19
Cho, Jong Hyun 26
Darabos, Katie 17
Feng, Zhaohui 12
Gallen, Corey 27
Heckman, Carolyn 15
Hoon Kim, Sang 22
Hyde, Emily 26
Kim, Hyungsin 20
Kostas, James 23
Pirani, Edisa 21
Ryu, Byungwoo 18
Shen, Yiyun 25
Song, Shumei 13
Sweet, Brandi 28
Tundealao, Samuel 28
Vaidya, Himani 22
Weiss, Stanley H 15
Xiong, Zhaohui 14

NJCCR Staff Contact Information

New Jersey Commission on Cancer Research

New Jersey Department of Health Office of Research Initiatives

25 South Stockton St.
2nd Floor

PO Box 364
Trenton, NJ 08625-0364
nj.gov/health/ces/njccr

Acknowledgements

The New Jersey Commission on Cancer Research would like to express its sincere appreciation to all present and past Commission members and the New Jersey Department of Health staff.

Lisa H. Cummings, R.N., M.S.
Executive Director
Senior Policy Advisor, New Jersey Department of Health
Office of Research Initiatives
(609) 913-5008
Lisa.Cummings@doh.nj.gov

Candido A. Africa III, M.D., C.P.M.
Program Management Officer - Consultant
Office of Research Initiatives
(609) 913-5011
Candido.Africa@doh.nj.gov

Amir Bhochhibhoya, Ph.D., M.B.A., M.C.H.E.S.
Program Management Officer
Office of Research Initiatives
(609) 913-5327
Amir.Bhochhibhoya@doh.nj.gov

Anita Madavane, M.S.F.
Grants Management Officer
Office of Research Initiatives
(609) 913-5004
Anita.Madavane@doh.nj.gov

Jitendra Jagasia, M.B.A.
Grants Management Officer
Office of Research Initiatives
(609) 913-5012
Jitendra.Jagasia@doh.nj.gov

NJCCR
NEW JERSEY
COMMISSION ON
CANCER RESEARCH

There's always hope beyonad what you see.

Cora Connor
Caregiver

NJCCR
NEW JERSEY COMMISSION ON CANCER RESEARCH

Dedicated to conquering cancer through scientific research

NJHealth
New Jersey Department of Health

C50989