

THE Dr. Anna Marie Skalka Annual Cancer Research Symposium

November 4, 2026
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
Annual Cancer Research Symposium



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, RN, MS, Executive Director, *NJCCR*
Generosa Grana, MD, Co-Chair, *NJCCR*

9:10 am **Keynote Address**
Epigenetic Regulation by CDK12 and the Cellular Stress Response
Jean-Pierre Issa, MD
President and Chief Executive Officer of the Coriell Institute for Medical Research

9:50 am **New Jersey State Cancer Registry Updates by Jie Li, MPH, PhD**

10:00 am **Grantee Presentations Facilitated by Peter Cole, MD**
Jian Cao, PhD
Rebecca Risman, PhD
Moamen Elmassry, PhD
Maria Ibrahim, PhD
Svetlana Bagdasarov, PhD

11:00 am **Networking Session on Scientific Posters**

12:00 am **Lunch**

1:00 am **Grantee Presentations Facilitated by Daniel Notterman, MD**
Gary Kwok, PhD
Shengkan Victor Jin, PhD
Carol Lutz, PhD
Coral Omene, MD
Dongfang Liu, PhD

2:00 pm **Panel Discussion Facilitated by Generosa Grana, MD**
Career Pathways and Employment Opportunities
Daniel Herranz, PharmD, PhD – *Rutgers CINJ*
Zhaohui Feng, PhD – *Rutgers CINJ*
Katie Devine, PhD, MPH - *Rutgers CINJ*
Binfeng Lu, PhD – *Hackensack Meridian Center for Discovery and Innovation*
Antoinette Stroup, PhD – *NJ State Cancer Registry & Rutgers School of Public Health*
Raymond Birge, PhD - *Rutgers Health, New Jersey Medical School*
Tessa Bergsbaken, PhD - *Rutgers Health, Infectious & Inflammatory Diseases*

3:00 pm **Award Presentations**
Legislative Champion Award
Dr. Jonathan Yavelow Mentor Award of Commendation
Patient Advocate Award

3:15 pm **Special Guest Speaker: Candido Africa III, MD**
Return on Investment in Cancer Research

3:30 pm **Concluding Remarks: Kenneth Adler, MD**

NJCCR NEW JERSEY COMMISSION ON CANCER RESEARCH



Drive Hope Forward represents our commitment to moving cancer research forward by supporting scientists and innovators working today to create discoveries and treatments for tomorrow. Funding helps fuel and advance cancer research in New Jersey.

Mission of the Commision:

To ensure that New Jersey residents receive the fullest benefit of our nation's fight against cancer through the 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

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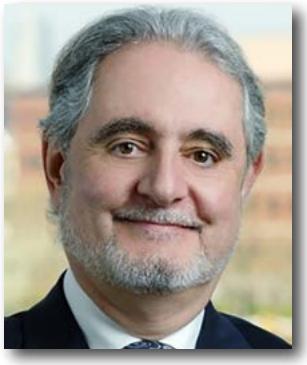
NJCCR Staff Contact Information 50

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Distinguished Keynote Speaker

Jean-Pierre Issa, MD

President and Chief Executive Officer of the Coriell Institute for Medical Research



Jean-Pierre Issa, MD, received undergraduate training and medical degree from the American University of Beirut followed by residency in internal medicine at Good Samaritan Hospital and fellowship in medical oncology at the Johns Hopkins Oncology Center in Baltimore. He was on the A faculty member at Johns Hopkins (1994-1999) and the University of Texas MD. Anderson Cancer Center (1999-2011), Dr. Issa was also. Professor of Medicine and Director, Fels Institute for Cancer Research from 2011 to 2019. He is currently President and Chief Executive Officer of the Coriell Institute for Medical Research in Camden, NJ.

Significant contributions to the understanding of epigenetics in cancer pathophysiology and treatment have emerged from Dr. Issa's laboratory. Discoveries include; (i) the first description of gene-specific DNA methylation changes with age (1994) which led to the eventual development of methylation-based aging clocks; (ii) the discovery of the CpG Island Methylator Phenotype (1999); (iii) the description of methylation-independent gene silencing in cancer by Polycomb group proteins (2008); (iv) the finding of a link between epigenetics and the microbiome in colon cancer (2014); and (v), the identification of a link between Cyclin Dependent Kinases and epigenetic silencing (2018). Starting in 2000, his group-initiated laboratory research directed clinical trials that showed that low doses of hypomethylating drugs specifically target DNA methylation and are optimal in the treatment of leukemias. This work contributed to the FDA approval of decitabine in 2006 and led to many epigenetic therapy clinical trials in different malignancies.

Dr. Issa's current research focuses on mechanisms of epigenetic alterations in aging and cancer, translation of epigenomic studies for precision medicine, development of drugs for reprogramming the epigenome, and clinical trials of epigenetic therapy in cancer. Additionally, he leads a group that was awarded a SPORE grant in Epigenetics from the NCI in 2021.

His research highlights include several recognition awards including a Sidney Kimmell Foundation Scholar Award in 1997, election to the American Society of Clinical Investigation in 2003, the Faculty Achievement Award in Basic Research from MD Anderson in 2007, the Rosenthal Award from the American Association for Cancer Research in 2011, election to the American Association of Physicians in 2015, and the Temple University Faculty Research Award in 2018.

Dr. Issa was also selected as an American Cancer Society Research Professor in 2007. As CEO of The Coriell Institute, he steered the center towards an unprecedented expansion, including the creation of the Camden Cancer Research Center in 2023, a collaboration with the Cooper Medical School at Rowan University and the Cooper University Hospital. In October 2025, the Institute broke ground for a new Health Sciences Campus in Camden, and a new building that will serve as headquarters for Coriell as well as for a Strategic Innovation Center in collaboration with the NJEDA.

Distinguished Keynote Speaker

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 principal investigator in cancer research.

Legislative Champion Award Recipients:
Marisa Sweeney, DCN RDN CSSD E-RYT 500
Assemblywoman, New Jersey 25th Legislative District



The Honorable Dr. Marisa Sweeney DCN, RDN, CSSD, E-RYT is an Assemblywomen representing New Jerseys 25th Legislative District. She is also a Doctor of Clinical Nutrition, Registered Dietitian Nutritionist, Certified Specialist in Sports Dietetics and Experienced Yoga Teacher. She is the Founder of Be Well Integrative Health Services and The New Jersey VegFest: The Garden State's Official Vegan Food Festival.

In addition to her business ventures, Dr. Sweeney does extensive volunteer and community work. She was the 2022 President of the New Jersey Academy of Nutrition and Dietetics, and New Jersey Representative in the House of Delegates for the national Academy of Nutrition and Dietetics from 2023-2025. Dr. Sweeney founded the Morristown Mayors' Wellness Campaign, is an Advisory Board Member for the Food and Nutrition Department of University of Saint Elizabeth, serves on Morristown's Planning Board, and is a Board Member of Grow it Green Morristown. She has also served on Atlantic Health's Community Advisory Board, as a Board Member of United Way North Jersey, and President of the Morristown Women in Business.

Dr. Sweeney has taught graduate classes at Northeastern University, and has been a guest lecturer at Montclair State University, Rutgers University, Rowan University, Saint Elizabeth University, Drew University, Drexel University, New York University and Columbia University.

Dr. Sweeney has been happy to support bills supporting cancer research and testing. She has co-sponsored bills that establish a pancreatic cancer awareness campaign and expand prostate cancer screening requirements for insurers. She's also used her vote and voice to increase the amount of tobacco product taxes provided to the NJCCR to $10 million.

Quinton Law
New Jersey Government Relations Director for the American Cancer Society Cancer Action Network (ACS, CAN)



Quinton Law serves as the New Jersey Government Relations Director for the American Cancer Society Cancer Action Network (ACS, CAN), where he leads advocacy efforts to advance public policies that prevent cancer, improve access to lifesaving treatments, reduce the financial burden of disease, and strengthen cancer research in New Jersey.

During his time with ACS, CAN, Quinton has helped advance several major bipartisan policy victories that have been signed

into law. These include expanding insurance coverage for biomarker testing, giving more patients access to precision medicine and treatments tailored to their cancer; passage of the Louisa Carman Medical Debt Relief Act, which established new protections for patients and families facing medical debt; and legislation protecting the value of copay assistance for patients managing high prescription drug costs.

Quinton is currently leading ACS CAN's advocacy to strengthen New Jersey's investment in cancer research by increasing annual funding for the New Jersey Commission on Cancer Research from $4 million to $10 million and establishing a dedicated, non-lapsing Cancer Research Fund. As part of this effort, he founded the Cancer Research Advocacy Coalition, which since January 2026 meets regularly to bring advocates, researchers, patients, and other stakeholders together in support of greater state investment in cancer research. Through the coalition, Quinton has worked to build sustained, bipartisan support for strengthening New Jersey's cancer research infrastructure and ensuring the state remains a leader in research and innovation.

In addition to his cancer advocacy, Quinton serves as Mayor of Moorestown, New Jersey, becoming the youngest actively serving mayor in the state when he assumed the role. He previously served on the staff of U.S. Senator Cory Booker and worked in economic development in the City of Camden. Across his work in state advocacy and local government, Quinton is committed to using public policy to improve lives and make government work for all of us.

Dr. Jonathan Yavelow Mentor Award Recipient: Suzie Chen, PhD

Distinguished Professor, Rutgers University nominated by Minatulah Elzawawy



Dr. Suzie Chen and her team have been studying the involvement of glutamatergic signaling in cutaneous melanoma using a genetic engineered mouse model. Her team identified the ectopic expression of a normal neuronal receptor, metabotropic glutamate receptor (GRM1/mGluR1), drives the initiation and progression of metastatic melanoma. They showed the aberrant expression of GRM1 is also detected in melanoma patients. Using an FDA approved drug, riluzole, for the treatment of Amyotrophic lateral sclerosis (ALS), they demonstrated reduced tumor progression in subsets of melanoma patients. As with other chemotherapy drugs, riluzole also led to a decrease in responsiveness with time. Recently, using cell lines, they were able to identify candidate proteins that may be involved in the onset of resistance to riluzole. Further studies are underway to validate these observations.

Dr. Chen and her group recently initiated studies to identify the different gene expression between normal and metastatic tumor cells by taking advantage of sequencing at the single cell level. The goal is to design approaches to diminish expression of these putative proteins that are involved in the metastasis process and reduce melanoma metastases.

Patient Advocate Award Recipient:
Evelyn Robles-Rodriguez, DNP, APN, AOCN

Cooper University Health Care – Breast Medical Oncology, Hematology Oncology

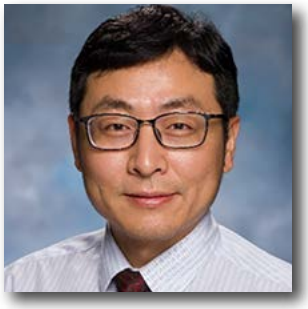


For more than 25 years, Dr. Robles-Rodriguez has been a tireless advocate for cancer patients and families throughout South Jersey, dedicating her career to expanding access to cancer prevention, early detection, treatment, and survivorship care. Through innovative screening, patient navigation, outreach, and survivorship programs, she has helped thousands of underserved and vulnerable individuals overcome barriers to care and has facilitated more than 1,500 cancer screenings annually. A respected clinician, educator, mentor, and community leader, Dr. Robles-Rodriguez has championed health equity, culturally responsive care, and patient empowerment while developing programs that improve quality of life throughout the cancer journey. Her clinical excellence, compassion, resilience, and sustained commitment to underserved communities have earned numerous honors and made her an exceptional leader and advocate for cancer care.

Grantee Presentations

Jian Cao, PhD

Affiliation: *Cancer Metabolism and Immunology Research Program at Rutgers Cancer Institute*



Project Title: *Oncoviral Integrations as Drivers and Therapeutic Vulnerabilities in Cancer*

Some viruses can cause cancer. Seven known human cancer-causing viruses are linked to about 12% of cancers worldwide. Two of the most important are hepatitis B virus, or HBV, and human papillomavirus, or HPV. HBV causes about half of liver cancers worldwide. HPV causes nearly all cervical cancers and many oropharyngeal and genital cancers. One shared feature of HBV- and HPV-related cancers is that viral DNA often becomes inserted into the DNA of human cells. This is called viral integration. These insertions can change how cells behave and help cancer develop. However, HBV and HPV integrations appear to promote cancer in different ways. In HBV-related liver cancer, viral DNA can insert into specific human genes. One important example is a gene called KMT2B. HBV insertions in KMT2B are found in about 10% of HBV-positive liver tumors. Our study showed that these insertions produce shortened forms of the KMT2B protein. When we introduced these shortened proteins into mouse livers, they helped drive liver cancer development. This cancer-promoting effect depended on their ability to bind to another protein called MENIN, which normally acts as a tumor suppressor. These findings show that HBV integration can create new cancer-driving changes in human genes. HPV-related cancers are different. Although HPV DNA also often integrates into human DNA, it usually does not insert into the same human gene again and again. Instead, HPV-related tumors almost always keep the part of the viral genome that contains two cancer-causing genes, E6 and E7. These genes are often expressed at very high levels and help cancer cells grow and survive. We are studying how HPV integration helps keep E6 and E7 highly active. We are also testing whether drugs that control gene activity can reduce E6 and E7 expression. By lowering E6 and E7, these drugs may help restore the cell's natural tumor-suppressing systems and make HPV-related cancers easier to treat. Together, these studies show that viral DNA insertions can help drive cancer, but they may also create weaknesses that can be targeted with therapy. By understanding how viral DNA changes cancer cells, we may find new ways to treat virus-associated cancers.

Rebecca Risman, PhD
Affiliation: *Rutgers University – Biomedical Engineering*



Project Title: ***Clot formation, structure, and fibrinolysis from plasma from pancreatic cancer patients***

Pancreatic cancer is one of the leading causes of cancer-related death with a five-year prognosis of 5-10%. Contributing to this high rate of morbidity and mortality is the fact that pancreatic cancer also has the highest prevalence of all cancers of venous thromboembolisms (VTE), which is the second leading cause of death in cancer patients. In the case of injury, you want your blood to clot in order for bleeding to be stemmed. However, if a blood clot forms when or where you do not want it this results in unwanted blood clotting (thrombosis). This occurs when the balance between clot formation and clot resolution (fibrinolysis) is shifted toward formation and away from resolution. It has been established that pancreatic cancer can lead to excessive blood clotting, but the mechanisms behind this are still unknown. Tumor cells release factors that cause overexpression of blood clotting agents and limit those that assist in resolution or the breakdown of clots. Furthermore, it is known that chemotherapeutic agents are prothrombotic, causing an elevated risk of getting a blood clot; however, not all cancer drugs take into account the need for medications that counteract the elevated clotting. Cisplatin, one of the common drugs for pancreatic cancer, is known to lead to procoagulation. Unfortunately, the mechanism of cisplatin-based chemotherapy leading to thrombosis has not been fully identified. Recurrence of thrombosis is common, despite delivery of blood thinning treatments; cancer and anticoagulant therapy can lead to an increase in bleeding, which complicates the approach of treatment. There is a need to understand the mechanisms of cancer related thrombosis and the thrombogenicity of cancer therapies to help optimize treatments. Our proposed research will explore these mechanisms using a high-throughput assay and structural studies to analyze different combinations the main four factors involved in the blood clotting process that are over- or under- expressed in pancreatic cancer plasma. Likewise, the development of a mathematical model of cancer-related thrombosis and fibrinolysis could predict the response to chemotherapy to choose the optimal treatment for thrombosis.

Presentations continued on next page

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

Grantee Presentations

Moamen M. Elmassry, PhD
Affiliation: *Princeton University, Department of Molecular Biology*



Project Title: *Discovery of novel microbiome-derived small molecules and their impact on colorectal cancer*

The trillions of bacteria living in our gut have long been linked to colorectal cancer, but the specific chemicals these bacteria produce that may drive this connection are still largely unknown. Most research has focused on only a handful of bacterial species and a narrow set of already-familiar chemicals, leaving much of this chemical landscape unexplored. In this study, we grew 30 different gut bacterial species previously linked to colorectal cancer in large-scale population studies. We collected the chemicals each species produced, sorting them into a library of 240 samples. We then tested these samples against three biological processes known to be important in colorectal cancer. This screening led to several key discoveries: a protein from one bacterial species that can block a family of enzymes involved in cell death, new chemicals from another species that can activate a receptor involved in cell growth regulation, and previously unrecognized compounds from a third species that activate a receptor connecting gut bacteria to the immune system. Together, this work establishes a new, scalable approach for discovering bacterial chemicals connected to colorectal cancer, laying the groundwork for future studies into how the microbiome influences cancer and pointing toward potential microbiome-based strategies for prevention or treatment.

Maria Ibrahim, PhD
Affiliation: *Rutgers University*



Project Title: *Autophagy Suppresses CCL2 to Preserve Appetite and Prevent Lethal Cachexia*

Macroautophagy (autophagy hereafter) captures intracellular components and delivers them to lysosomes for degradation and recycling. In adult mice, autophagy sustains metabolism to prevent cachexia, survive fasting and suppresses inflammation, liver steatosis, neurodegeneration, and lethality through unclear mechanisms. Here we profiled metabolism and inflammation in adult mice with conditional, whole-body deficiency in an essential autophagy gene and found that autophagy deficiency altered fuel usage; reduced ambulatory activity, energy expenditure, and food intake; and elevated circulating GDF15, CXCL10, and CCL2. While deletion of GDF15 or CXCL10 provided no or mild benefit, deletion of Ccl2 restored food intake, suppressed cachexia, and rescued lethality of autophagy-deficient mice. Neutralization of CCL2 using a monoclonal antibody similarly prevented

weight loss and prolonged survival of these mice. Single nucleus RNA sequencing and histology of the hypothalamus revealed that autophagy deficiency was specifically toxic to hypocretin/orexin and pro-MCH neurons that produce orexigenic neuropeptides that promote food intake, which was rescued by CCL2 deficiency. Restoration of food intake via leptin deficiency also prevented lethal cachexia in autophagy-deficient mice. Our findings demonstrate that autophagy prevents induction of a cachexia factor, CCL2, which damages appetite-maintaining neurons, and identify CCL2 blockade as a potential therapeutic strategy for degenerative wasting conditions.

Svetlana Bagdasarov, PhD
Affiliation: *Libutti laboratory at the Cancer Institute of New Jersey, and Rutgers New Jersey Medical School*



Project Title: *Octreotide-targeted α-galactosyltransferase gene therapy using hybrid AAV phage for antitumor immune response*

Cancer immunotherapy is a treatment that involves a patient's own immune system to fight cancer. The immune system protects the body from infections and other diseases including cancer by attacking any new substance that is not normally found in the body. When normal cells become cancerous, they are altered and can produce certain proteins that the immune system does not recognize. Antibody binding or targeting by special immune cells raises an alarm to destroy these foreign cells. In many people who develop cancer, the immune system does not work because some types of cancer are not different enough from normal cells to be recognized as foreign. To overcome this, our strategy for this research project is to modify a prokaryotic virus to infect only cancer cells to improve immune recognition and elimination of cancer cells. Prokaryotic viruses infect and replicate within bacteria alone and lack intrinsic ability to infect mammalian cells. These viruses have a long history of safe delivery to humans to treat bacterial infections. In addition, they can be modified to target mammalian cells that have specific receptors, a property our group will use to deliver an immune-stimulating gene to tumors. This gene, called α−1,3-galactosyltransferase (Ggta1), makes a protein which transfers a Gal carbohydrate to the surface of cells. Ggta1 is found in large amounts in most mammals, however, is absent in humans. As a result, humans lack Gal carbohydrate and antibodies to Gal are the most abundant natural antibodies in human blood. Pig organs transplanted in humans are rapidly rejected because anti-Gal antibodies interact with the Gal carbohydrate on pig tissue and destroy the transplanted organ. Delivery of the Ggta1 gene specifically to cancer cells will enable them to express this carbohydrate that will bind to anti-Gal antibodies to attack the tumor by the same mechanism.

Continued on next page

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.

Development of this new virus-based targeted gene therapy may enhance the immune response to identify and attack tumor cells and directly benefit cancer patients. In this study, the described treatment approach was probed on a mouse model of pancreatic neuroendocrine tumors. However, this targeted treatment methodology is quite versatile with potential application to a wide variety of cancers.

Gary Kwok, PhD
Affiliation: *Center for Discovery & Innovation, Hackensack Meridian Health*



Project Title: ***Advancing Implementation Science: Integrating Mindfulness Apps into AYA Cancer Survivorship Care***

Young people who survive cancer often face ongoing stress, anxiety, and challenges as they return to school, work, and everyday life. Mindfulness—learning to focus on the present moment in a calm and supportive way—can help reduce stress and improve emotional well-being. Mobile apps make it possible to practice mindfulness anywhere, but many young survivors struggle to use these tools consistently. They may forget, feel unsure about how to begin, or feel disconnected from the app without support from their care team. Healthcare providers can play an important role by recommending mindfulness apps and offering simple check-ins to encourage use. However, providers also face real challenges, such as limited time, unclear steps for introducing apps, or uncertainty about which tools are most helpful for young people.

This project aims to understand these real-world challenges from both survivors and providers so we can identify practical ways to bring mindfulness apps into routine cancer care. Through interviews with AYA survivors and healthcare providers, we found that providers are most concerned about whether they have enough time, resources, and clear processes to introduce and support app use. Survivors, on the other hand, emphasized that encouragement from people they trust and clear information about how the apps work strongly influence whether they use them.

By learning what helps—and what gets in the way—we can develop strategies that make it easier for young survivors to access and sustain mindfulness practices that support their well-being during and after treatment. As a next step, we are exploring engagement boosters such as brief clinician-led check-ins and peer support groups for AYA cancer survivors to help them stay connected to mindfulness apps and continue practicing regularly. We will design and test these strategies in real clinical settings, with the goal of preparing for larger clinical trials. These trials will help determine how mindfulness apps can be successfully integrated and sustained in cancer care, ultimately improving the emotional and psychological health of adolescent and young adult cancer survivors.

Victor Shengkan Jin, PhD, *Professor*
Affiliation: *Rutgers, The State University*



Project Title: *Developing Off-the-shelf CAR-T for treating Acute Lymphocytic Leukemia with a New Base Editing Technology*

Acute lymphoblastic leukemia (ALL) is the most common cancer type in children and the second most common acute leukemia in adults. B- cell type ALL (B-ALL) accounts for the majority of pediatric ALL and 75% of adult ALL. While the initial response rate to frontline therapies of B-ALL is high (85%), the relapsed and refractory B-ALL is accompanied by a dismal prognosis and accounts for a large percentage of cancer-related mortality, specifically in the pediatric population. Improving B-ALL treatment is of great significance to pediatric cancer research and healthcare in general.

In recent years, chimeric antigen receptor T cells (CAR-T cells) have emerged as effective immunotherapy to treat advanced B-ALL cancers. The CAR-T cells are engineered from a patient's own T cells to produce an artificial T cell receptor that can recognize the patient's cancer cells and act as a "living drug" to attack these tumor cells. CAR-T cell therapy has achieved outstanding clinical outcomes, with complete response (CR) rates of 65–90% across clinical trials spanning institutions. However, the process to engineer these cells is currently highly complex, lengthy, and very costly. The root cause of these issues is that the current CAR-Ts are engineered individually from the patient's own T cells in a laboratory setting. It is highly desirable to develop off-the-shelf CAR-T therapies, where T cells from healthy donors are engineered to create a bank of cells that are available for treating patients on-demand. Dr. Jin's Lab developed a novel Base Editing technology (Pin-pointTM Base Editing), providing a safe way to do a "surgical operation" on DNA by avoiding the oncogenic damages to DNA. Funded by the New Jersey Commission on Cancer Research, the base editing technology was successfully applied to simultaneous inactivate multiple immune genes and introduce the CAR gene in human primary T cells, creating a new approach of engineering and manufacturing the off-the-shelf CAR-T cells. This proof-of-concept research, later published in Molecular Medicine, has contributed to the subsequent sub-license of the technology to global pharma company AstraZeneca for developing off-the-shelf CAR-T and other cell therapies.

Grantee Presentations

Carol Lutz, PhD

Affiliation: *Rutgers Biomedical and Health Sciences*



Project Title: *Regulation of Arachidonic Acid Metabolism and Gene Expression by RNA Mediated Mechanisms*

Long noncoding RNAs regulate gene expression; however, in many cases, the mechanism of this regulation is unknown. One novel lncRNA relevant to inflammation and arachidonic acid (AA) metabolism is the p50-associated COX-2 extragenic RNA (PACER). PACER is a key regulator of COX-2. COX-2 is a major enzyme in the AA pathway, an important inflammatory signaling network. PACER is known to play a role in inflammation-associated conditions, yet the mechanisms responsible for the regulation of PACER are not understood. Our recent research focuses on the regulation of PACER in lung cancer. Our data suggest PACER is a key regulator of COX-2 expression and dysregulated expression in lung cancer cells. Bioinformatic analysis of TCGA data has identified that PACER is overexpressed in lung cancer patients. Our experimental results identified previously undescribed transcription factors may play a role in PACER regulation in lung cancer cells. We hypothesize that Inhibition of PACER expression decreases lung cancer cell proliferation and migration and has regulatory effects on the arachidonic acid metabolic pathway and beyond.

Coral Omene, MD, PhD

Affiliation: *Program Director, Breast Cancer Disparities Research, Robert Wood Johnson Medical School, Rutgers Cancer Institute*



Project Title: *Evaluating the Gene Rearrangement Landscape and Response to Neoadjuvant Chemotherapy in Triple Negative Breast Cancer and its impact on Cancer Health Disparities*

A significant number of women with early-stage breast cancer receive chemotherapy before surgery (“neoadjuvant chemotherapy”), and those whose tumors disappear completely (a "pathological complete response") tend to survive very well. This is especially true for triple-negative breast cancer (TNBC), an aggressive form of the disease that's more common in Black women. Studying tumor genetics can help doctors predict how well a patient will respond to treatment, choose the best therapies, and understand why some cancers resist treatment. Obesity, which causes chronic inflammation in the body, is a known contributor to worse breast cancer outcomes and may also influence these genetic patterns, making it an important research area to study. In this study, Black patients with early-stage TNBC treated with neoadjuvant

chemotherapy were identified from the tumor registry at Rutgers Cancer Institute/ RWJBarnabas Health System. Clinical information was extracted from the health records and gene sequencing performed on the tumors obtained before treatment and on any remaining tumor left after treatment. We analyzed the genetic changes and gene expression activity of the tumors to see how they related to treatment response and obesity. We found differences between tumors from obese and non-obese patients, including in genes already linked to aggressive TNBC in Black women. There were differences in tumor gene expression activity before versus after treatment in patients whose cancer didn't fully respond to treatment so did not achieve a pathologic complete response to chemotherapy prior to surgery.

These findings lay the groundwork for larger studies aimed at a better understanding of TNBC biology in the setting of obesity and the development of treatment strategies for patients who don't respond well to treatment, to ultimately improve survival and reduce health disparities.

Dongfang Liu, PhD
Affiliation: *Department of Pathology, Immunology and Laboratory Medicine, Center for Immunity and Inflammation, Rutgers New Jersey Medical School*



Project Title: ***HSP90 Inhibition Drives GRP78 Relocalization to Potentiate CAR-Based Immunotherapy during Tumor Evolutions***

For patients whose cancer no longer responds to treatment, new therapies cannot help unless they can be produced reliably. This project will advance an experimental treatment made from natural killer cells, immune cells that help the body fight cancer. Our Rutgers team engineers these cells to recognize CD147, a protein found on cancer cells. The resulting cells, called CD147-CAR-NK cells, could potentially be prepared from donated blood and stored for future use as an "off-the-shelf" treatment. A major challenge is growing enough cells while preserving their cancer-fighting activity and consistent quality. We have developed a method that uses specially designed support cells to help these immune cells grow. Rather than replace this established method, we will adapt it for tightly controlled manufacturing at Rutgers' specialized cell and gene therapy facility. Working with manufacturing experts and cancer physicians, we will test whether the process produces consistent batches, assess cell purity and cancer-killing ability, and determine how well the cells retain their quality during storage. We will also establish safety checks and prepare the manufacturing records needed to support a future application to the U.S. Food and Drug Administration for testing in people. This project develops the manufacturing foundation; it does not treat patients. By connecting New Jersey research, specialized manufacturing, and clinical expertise, the work could expand future treatment options while strengthening the state's biotechnology workforce, industry partnerships, and cell-therapy innovation.

2027 Grant Recipients

Category: Postdoctoral, Type of Cancer: General
Grant *#COCR26RBG002*

Arif Satria Wira Kusuma, PhD, ak1695@gsbs.rutgers.edu
Affiliation: *Rutgers University*



Project Title: *Investigating the Role of the p53-mTOR Pathway in Autism Pediatric Cancers*

Autism is a disorder that affects how the brain develops, and children with autism often face other health challenges. For severe autism, there is an increased risk of cancer (Liu, 2022). For example, children with autism and birth defects or intellectual disability (ID) have an increased risk for cancer. In addition, children with a specific deletion of chromosome 16 (16p11.2) is one of the greatest contributors to autism and are about 14 times more likely to develop neuroblastoma. The Millonig laboratory uses neural progenitor cells (NPCs) derived from induced human pluripotent stem cells (iPSCs) to study the molecular genetic basis of autism. Previous data have demonstrated that the 16p11.2 deletion reduces the activity of the well-studied onco-protective gene p53. P53 is often described as the "guardian" against cancer because it helps prevent damaged or overgrown cells from becoming tumors. With less p53, the NPCs grow too quickly. We do not yet know whether the same thing happens in the neural crest cells (NCCs) that give rise to neuroblastoma. In this project, we will grow NCCs from children with autism and test whether lower p53 causes them to multiply too fast, cope poorly with stress, and accumulate more damage in their genetic material. Our results may help explain why autism and childhood cancer are linked, an important but understudied question, and could eventually guide better ways to monitor and protect the health of children with autism.

Category: Postdoctoral, Type of Cancer: Liver
Grant *#COCR27PDF006*

Samer Ayoub, PhD, samer.ayoub@princeton.edu
Affiliation: *Trustees of Princeton University*



Project Title: *Developing a Zebra Fish-Based In-Vivo Model to Study HBV Driven-Liver Carcinogenesis*

Liver cancer is one of the deadliest cancers worldwide. A major cause of this disease is long-term infection with hepatitis B virus (HBV), which is responsible for about 60% of all liver cancer cases. Although billions of people have been exposed to HBV, nearly 300 million people live with chronic infection. Many of them, up to two-thirds, do not

know they are infected and therefore do not receive medical care. Over time, chronic HBV infection can severely damage the liver and greatly increase the risk of liver cancer, leading to hundreds of thousands of deaths each year. While effective vaccines can prevent HBV infection, and existing treatments can slow disease progression, there is currently no cure for HBV, and lifelong treatment is required. Despite the strong connection between HBV and liver cancer, we still do not fully understand how the virus causes cancer. One major challenge is the lack of suitable animal models that allow researchers to study the earliest steps of cancer development. In addition, recent studies suggest that a person's genetic makeup plays an important role in determining who develops liver cancer, but how these genetic differences interact with HBV to drive cancer remains largely unknown. To overcome these challenges, this project will develop a new and innovative animal model using zebrafish. Zebrafish are widely used in cancer research because they develop quickly, are easy to study genetically, and have livers that function similarly to human livers. Unlike traditional cancer models that rely on artificially inducing cancer-causing mutations, this project uses viruses closely related to human HBV that naturally infect fish. Importantly, some of these HBV-like viruses are already linked to liver cancer in their natural hosts, making them an ideal system to study virus-driven cancer from its earliest stages. My preliminary work has already shown that several of these fish viruses can function inside zebrafish liver cells. This proposal will examine how these viruses replicate, how their viral components behave, and whether they can trigger liver disease and cancer in zebrafish. By creating this new model, the proposed research will provide valuable insight into how HBV causes liver cancer and why some individuals are more vulnerable than others. Ultimately, this work may help guide the development of better strategies to identify people at highest risk, improve early cancer detection, and inform future approaches to prevent and treat HBV-associated liver cancer.

Category: Bridge, Type of Cancer: Colon & Rectum
Grant *#COCR27RBG004*

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Project Title: *Gain-of-function mutant p53 and metabolic reprogramming in colorectal cancer*

Colorectal cancer is a leading cause of cancer-related death in the United States. Chronic inflammation of the intestine, including that associated with inflammatory bowel disease, can greatly increase the risk of developing a particularly aggressive form of colorectal cancer known as colitis-associated cancer. Compared with more common

forms of colorectal cancer, colitis-associated cancer often develops at a younger age and has poorer outcomes, highlighting the need for better approaches to prevention and treatment. Our research focuses on tumor suppressor p53, an important gene that normally protects cells from becoming cancerous but is frequently altered in human cancers. Notably, p53 alterations occur particularly early and frequently in colitis-associated cancer, suggesting that loss of p53 may play an important role in allowing inflammation to promote tumor development. As a transcription factor, p53 senses a variety of stress signals and regulates the expression of its target genes to exert its tumor-suppressive function. While early studies have established p53's functions in cell cycle arrest, repair and apoptosis in cancer cells as important tumor-suppressive mechanisms, recent studies have strongly suggested that p53's function in regulating the tumor microenvironment contributes significantly to its role as a tumor suppressor. This project will investigate how p53 regulates the immune environment and its contribution to suppressing inflammation-associated cancer. By uncovering how p53 connects chronic inflammation and colorectal cancer development, this research may reveal new understanding on colorectal cancer, and to provide a foundation for developing more effective strategies to prevent and treat colitis-associated colorectal cancer.

Category: Predoctoral, **Type of Cancer:** HPV-associated cancers
Grant *#COCR27PRF020*

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Project Title: ***Identification of HPV-16 E6/E7-Reactive TCR for Broader Application TCR-T Cell Therapy***

Human papillomavirus (HPV) is a sexually transmitted infection and a major cause of several epithelial cancers, including cervical, anal, and some head and neck cancers. Advanced stages of these HPV-associated cancers remain difficult to treat with currently available therapies. A promising new approach is T cell receptor (TCR)-T cell therapy, which equips a patient's own immune cells with the ability to recognize and kill cancer cells by targeting viral proteins made by HPV. An ongoing clinical trial at Rutgers Cancer Institute is evaluating a TCR-T therapy targeting one of these HPV-16 viral proteins, E7. This therapy was developed by our group. Early results have been encouraging. In an initial group of ten patients whose cancers had not responded to multiple prior treatments, six experienced significant tumor shrinkage, and the therapy was generally well tolerated. Notably, two patients had all detectable metastatic tumors disappear after one treatment and have remained cancer-free for more than one year. This advance brings new hope to patients who would otherwise be considered incurable.

Despite this breakthrough, not all patients will be eligible for this treatment because TCR-T therapies depend on a patient's specific human leukocyte antigen (HLA) proteins. Similar to blood types, HLA varies from person to person and can differ in frequency across ethnic backgrounds. The current E7 TCR-T therapy is restricted to a single HLA type, HLA-A*02:01, which is required to display the E7 fragment so the engineered T cells can recognize and kill tumor cells. As a result, approximately two thirds of patients in the state of New Jersey, who carry other HLAs would not be expected to benefit from the current E7 TCR-T approach. Therefore, it is important to discover additional TCRs that work with different HLA types so that more people can benefit from this therapy. To accomplish this, I will use two approaches to discover new TCRs specific to two common HLA types, C*07:02 and A*11:01, by (1) isolating HPV-reactive T cells from healthy donor blood, and (2) using a mouse model to develop new TCRs. T ogether with our ongoing laboratory strategy of identifying TCRs directly from patient tumor samples, these parallel approaches allow us to pursue almost every feasible path to expand HPV-targeted TCR-T therapies to a broader patient population. By finding new TCRs compatible with additional HLA types, this work aims to expand the treatment options for patients across diverse populations.

Category: Pilot, Type of Cancer: Liver
Grant *#COCR27PPR010*

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Project Title: ***Clinical-Grade Manufacturing of CD147-CAR-NK Cells for IND-Enabling Studies Targeting Cancers***

For many patients in New Jersey diagnosed with aggressive cancers such as acute myeloid leukemia (AML), treatment options become extremely limited once standard therapies fail. These patients often face toxic side effects, prolonged hospital stays, and uncertain outcomes. While recent advances in CAR-T cell therapy have transformed cancer care, these treatments can cause life-threatening complications and are expensive and difficult to manufacture. There remains a critical unmet need for safer, more accessible immune-based therapies. Our project aims to bring forward a next-generation treatment known as CAR-NK cell therapy. Natural killer (NK) cells are part of the body's natural immune defense and can destroy cancer cells with a lower risk of severe inflammatory side effects compared to CAR-T therapy. Researchers at Rutgers have developed an innovative method to grow and engineer NK cells in large numbers while preserving their strength, stability, and safety. This breakthrough makes it possible to produce high-quality immune cells that could be delivered as an "off-the-

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

shelf" therapy for patients. With support from the New Jersey Commission on Cancer Research, we will work with the Rutgers Immune, Cell and Gene Therapy GMP Facility and clinical leaders at Rutgers Cancer Institute to manufacture clinical-grade CD147-CAR-NK cells under strict FDA-compliant standards. The goal of this project is to generate the essential safety, quality, and manufacturing data required to seek federal approval to begin first-in-human clinical trials. If successful, this work could accelerate access to a safer and more scalable immunotherapy for patients with leukemia and other high-risk cancers in New Jersey. Beyond patient benefit, the project strengthens New Jersey's position as a national leader in advanced cell therapy manufacturing. By building GMP-ready infrastructure, training specialized personnel, and advancing homegrown intellectual property, this initiative supports local biotech innovation, attracts industry partnerships, and contributes to the state's growing life sciences economy.

Category: Pilot, Type of Cancer: Hematologic
Grant *#COCR27PPR029*

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Project Title: *Cellular Bioenergetics, the Achilles' Heel of Adult T-Cell Leukemia/ Lymphoma*

Adult T-cell Leukemia/Lymphoma (ATLL) is a rare and aggressive cancer of the white blood cells caused by a virus called HTLV-1. In the United States, patients survive a median of less than seven months after diagnosis, and there are currently no treatments that reliably work. Recent studies found that many North American ATLL patients carry damaging mutations in a gene called EP300, which normally helps cells control which genes are turned on and off, but it is not known why losing this gene makes the cancer so aggressive. Our laboratory discovered that ATLL cells lacking EP300 have damaged mitochondria, the tiny "power plants" that supply a cell's energy. To survive, these cancer cells appear to rely on a backup process called autophagy, in which a cell breaks down its own worn-out parts for fuel. We believe this dependence is the cancer's Achilles' heel. In this project, we will first measure how losing EP300 changes the way ATLL cells produce and use energy. We will then test whether blocking the backup fuel process with hydroxychloroquine, an inexpensive drug already widely used in patients, makes the cancer cells more vulnerable to standard chemotherapy. If successful, this work will explain what drives this aggressive cancer and point to a new treatment combination using existing medicines, which could move quickly toward patients who currently have few options.

Category: Predoctoral, Type of Cancer: Liver
Grant *#COCR27PRF011*

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Project Title: *Understanding Virally Induced Hepatocarcinogenesis Using Molecular Recorders*

Hepatocellular carcinoma is a leading cause of cancer-related mortality worldwide and frequently develops in the setting of chronic hepatitis B or C virus infection. Although antiviral therapies can cure hepatitis C and suppress hepatitis B, liver cancer risk can persist after treatment, particularly in patients with metabolic conditions such as obesity, diabetes, and fatty liver disease. This persistent risk suggests that viral infection may induce durable changes in hepatocyte function that are compounded by metabolic stress, but the underlying cell-specific mechanisms remain poorly understood. We have developed a molecular recording system that permanently labels hepatocytes infected with rodent hepacivirus (RHV), a hepacivirus related to HCV that enables controlled studies of infection in mice. This system allows previously infected hepatocytes to be identified after viral clearance and their transcriptional states to be analyzed at single-cell resolution. We will use this approach to compare hepatocytes during acute infection, chronic infection, and after antiviral cure, and determine how diet-induced fatty liver modifies these states. Defining how viral history and metabolic stress interact within hepatocytes may reveal mechanisms that sustain liver cancer risk after antiviral treatment and identify opportunities for improved risk stratification and prevention.

Category: Predoctoral, Type of Cancer: Ovarian
Grant *#COCR27PRF018*

Adam Robinson, adam.robinson@rutgers.edu
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Project Title: *Evaluating Cellular Stress in T Cell-Mediated Humoral Immunity in Ovarian Cancer*

High-grade serous ovarian cancer is one of the deadliest forms of ovarian cancer. Although treatments such as chemotherapy and newer immunotherapies - drugs that help the immune system attack cancer - have helped some patients, many tumors still escape the body's

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

defenses. Understanding why this happens is essential to developing better treatments. Our research focuses on T cells, a type of white blood cell that helps the body find and destroy cancer. In ovarian tumors, T cells are often present, but they do not always work well together. Tumors create stressful conditions, such as low oxygen and limited nutrients, that weaken these immune cells. We are studying a protein called BLTP3A that becomes more active in T cells under stress. Our early findings suggest that BLTP3A acts like a brake, limiting the immune system's ability to mount a strong and lasting attack. Patients tend to do better when T cells form organized groups inside tumors. A specialized type of T cell, called T follicular helper (Tfh) cells, helps coordinate these responses. We believe BLTP3A interferes with this process. Using patient samples and laboratory models, we will test whether reducing BLTP3A's effects can strengthen T cell coordination, improve tumor control, and increase the benefit of immunotherapy. By understanding how tumors weaken T cells, this research may lead to new strategies that help the immune system fight ovarian cancer more effectively.

Category: Pilot, Type of Cancer: Ovarian
Grant *#COCR27PPR042*

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Project Title: *Assessing the Efficacy and Safety of BLTP3A-Targeted amiR Engineered T Cell in Cancer*

Ovarian cancer remains one of the deadliest cancers affecting women, and current immunotherapies have had limited success in this disease. One promising approach is adoptive T cell therapy, in which a patient's immune cells are engineered to recognize and kill tumor cells. However, these engineered T cells often lose function after entering the stressful tumor microenvironment. The T cell receptor (TCR) is a critical surface protein that allows T cells to recognize cancer cells and activate their killing program. Our studies show that tumor-associated stress disrupts TCR stability and recycling, weakening T cell function. We have identified BLTP3A as an important regulator of this process. In preclinical models, loss of BLTP3A preserves TCR expression and allows T cells to remain active under tumor-associated stress. We have also developed an approach to engineer human tumor-reactive T cells in which BLTP3A is silenced while the cells are programmed to recognize ovarian cancer. However, it remains unclear whether BLTP3A-silenced T cells can maintain durable antitumor activity and safely control ovarian cancer in vivo. This project will define how BLTP3A regulates TCR function under tumor-associated stress and test whether targeting BLTP3A can improve the activity of engineered T cell therapies for ovarian cancer.

Category: Pilot, Type of Cancer: Psychosocial/Behavioral
Grant *#COCR27PPR071*

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Project Title: *Empower Patients with Breast Cancer in New Jersey with a Co-Designed AI Assistant: A Pilot Project*

In New Jersey, where breast cancer rates are higher than the national average, there is a large and growing community of survivors. Many of these survivors struggle with ongoing symptoms after treatment, which can lower their quality of life. While digital health tools can help, they are often confusing, especially for those who are not tech-savvy or find medical information overwhelming. To solve this problem, we are creating "AI-to-Cope," a friendly and easy-to-use artificial intelligence (AI) tool. AI-to-Cope works like a chatbot, providing reliable health information through simple messaging or voice conversations. It is designed to be safe, private, and easy to understand for everyone, regardless of their comfort level with technology. The goal is to help patients build the knowledge and confidence they need to effectively manage their symptoms. In this project, we will first work with patients and doctors to build an early version of AI-to-Cope. Then, we will engage patients—the future users of AI-to-Cope—to gather feedback and refine the tool for usability, acceptability, and practical value. This process will ensure AI-to-Cope is responsive to survivor needs and positioned to support health and well-being among breast cancer survivors.

Category: Pilot, Type of Cancer: Psychosocial/Breast
Grant *#COCR27PPR092*

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Project Title: *Investigating Phosphatidylserine Externalization on Activated Immune Cells for Novel Immunotherapy Approaches*

Cancer cells can avoid destruction by creating conditions that weaken the immune cells sent to eliminate them. Immune checkpoint inhibitors, a cornerstone of modern cancer immunotherapy, are designed to address this

tactic, but currently benefit only about one in five patients who receive them, in part because T cells within tumors often become "exhausted," losing their ability to attack cancer cells. One contributor to this immune evasion is a lipid called phosphatidylserine (PtdSer). Under healthy conditions, PtdSer stays on the inner surface of the cell membrane, but under stress it can move to the outside. Cells within tumors often display high levels of external PtdSer, which suppresses the immune response. Recent research from this laboratory and others has shown that healthy, activated T cells can also externalize PtdSer even when they are not dying, suggesting that this lipid signal on immune cells themselves may influence how effectively they function inside the tumor microenvironment. This project aims to uncover why activated T cells expose PtdSer and how this lipid shift affects their ability to fight cancer. Using advanced genetic and cellular tools, our team will identify the enzymes that control PtdSer externalization in T cells and test whether adjusting these pathways can restore or strengthen responses in these exhausted, underperforming subsets. By defining how membrane lipid dynamics shape immune behavior, this work may reveal new immunotherapy strategies that target the enzymes regulating PtdSer externalization on T cells

Category: Postdoctoral, Type of Cancer: Other
Grant *#COCR27PDF009*

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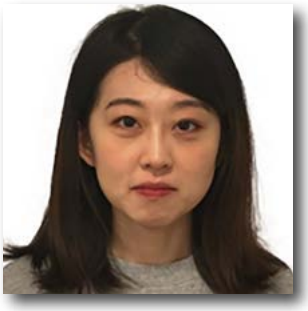
Project Title: *mTORC1 Control of Taurine Metabolism as a Potential Vulnerability in Cancer*

Most cancer cells grow uncontrollably with the help of a protein complex called mTOR Complex 1 (mTORC1), which acts like a master switch for cell metabolism. In most human cancers, this switch is permanently turned on, driving tumor growth. Because of this, there have been intense efforts to block mTORC1 in cancer. However, with over 1000 clinical trials to date, the drugs used to target mTORC1 have shown limited success: they can slow or halt cancer cell growth but rarely kill the cancer cells. Thus, the tumors remain, the cells are healthy and viable, and they resume their rapid growth once the treatment is discontinued. Our research takes a different approach. Instead of directly targeting mTORC1, we focus on the metabolic pathways it controls. Our previous work provided a proof-of-principle demonstration that tumor cells can be killed by blocking metabolic pathways that are controlled by mTORC1, without affecting mTORC1 itself, referred to as “metabolic vulnerabilities”. Thus, it is critical to determine exactly which metabolic pathways are controlled by mTORC1, not only as an essential step in understanding cancer growth, but also as a basis for identifying and exploiting new

metabolic vulnerabilities that could be broadly applicable in cancer. However, key metabolic functions of mTORC1 remain to be discovered, in part because of limitations of older drugs that either did not completely turn off mTORC1, or they non-specifically turn off other important proteins in the cells. To resolve this problem, we are using a new generation drug that fully blocks only mTORC1, giving us a clear understanding of how it regulates cancer cell metabolism. Using this new class of drugs, we discovered new metabolic pathways controlled by mTORC1, including taurine metabolism. It has recently been discovered that taurine helps cancer cells grow, but it was not known that mTORC1 is a major player that controls taurine. Based on our new data, we hypothesize that mTORC1 regulates taurine levels in cancer, which fuels cancer growth. In this project, we will figure out exactly how mTORC1 controls taurine in cancer, and whether blocking the cancer cells' ability to use taurine could be a new way to treat them. Ultimately, we hope this research will not only uncover a new way that cancer cells fuel themselves, but also lead to a new, effective treatment strategy.

Category: Postdoctoral, Type of Cancer: Prostate
Grant *#COCR27PDF016)*

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Project Title: *Non-Canonical AR Signaling in Prostate Cancer Pathogenesis and Therapy*

Prostate cancer affects about 1 in 8 men during their lifetime and is a major cause of cancer-related death. Most prostate cancers depend on male hormones, called androgens, to grow, so treatment often works by lowering androgen levels or blocking their effects. Although these treatments can be effective at first, many tumors eventually begin growing again and become much harder to treat. Our laboratory has found that a major cell-growth pathway called mTOR may help prostate cancer cells survive by activating the androgen receptor, a protein that normally responds to male hormones, even when androgen levels are very low. This may allow cancer cells to continue growing despite hormone-blocking treatment. In this study, we will investigate how mTOR activates the androgen receptor without hormones and how this process contributes to treatment resistance and tumor growth. By better understanding this alternative growth pathway, we hope to identify new treatment targets and develop more effective combination therapies for patients with advanced prostate cancer.

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Category: Pilot, Type of Cancer: Ovarian
Grant *#COCR27PPR030*

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Project Title: *Implementation of Opportunistic Salpingectomy into General Surgery; A Pilot Study*

Ovarian cancer remains a significant cause of cancer-related morbidity and mortality, and opportunistic salpingectomy (OS)—removal of both fallopian tubes during an unrelated abdominal surgical procedure—offers an important opportunity for prevention among women who have completed childbearing. Population-based studies suggest that OS is associated with a 50–60% reduction in future ovarian cancer risk, and it has increasingly been incorporated into gynecologic surgery. However, OS remains uncommon during non-gynecologic abdominal operations, representing an important gap in the implementation of an evidence-based cancer prevention strategy. This pilot study will examine the surgeon-, patient-, and health system-level factors that influence whether OS is considered and incorporated into General Surgery practice across the 12-hospital Rutgers RWJBarnabas Health system. Using established implementation science frameworks, we will identify barriers and facilitators to OS adoption through structured stakeholder engagement and interviews with surgeons, and use these findings to develop and refine a practical, stakeholder-informed workflow for implementation. We will also assess patient perspectives, including perceived risks and benefits, preferences regarding surgical decision-making, and whether there are disparities in which patients are considered for OS. The resulting data will provide preliminary evidence regarding the feasibility, acceptability, and equitable implementation of OS in non-gynecologic abdominal surgery and will inform the development of a future multi-site implementation trial aimed at expanding ovarian cancer prevention.

Category: Predoctoral, Type of Cancer: Psychosocial/Breast
Grant *#COCR27PRF004*

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Project Title: *Investigating the Effects of Obesity and Weight Loss Therapy in Early-Onset Breast Cancer Risk*

Breast cancer is increasingly being diagnosed in women under 40, often in aggressive forms and without a family history of the disease. At the same time, rates of obesity have risen dramatically in this same age group. More

than half of young women diagnosed with breast cancer are living with obesity, and obesity is consistently linked to worse treatment outcomes and higher rates of cancer returning. Despite this alarming overlap, we do not understand how obesity affects breast cancer in young women, or whether losing weight can reduce that risk. The newer class of weight loss drugs — including tirzepatide, has transformed obesity treatment and is now taken by millions of people, many of them young women. These drugs produce substantial, sustained weight loss, but whether they also lower cancer risk is completely unknown. In this project, I will use specially designed mouse models that develop breast cancer in adulthood, similar to how young women develop the disease, and feed them with a diet that produces obesity resembling the human condition. My preliminary results are striking: obesity dramatically accelerated breast cancer development, and weight loss with tirzepatide completely reversed this effect. When I examined the tissue at the earliest stage of cancer, I found that the drug changed the immune cells and surrounding tissue in ways that appeared to suppress tumor growth. Building on these findings, I will determine whether these results hold across different breast cancer types, identify which specific cells and biological signals are responsible, and test whether the drug's cancer-protective effects go beyond weight loss alone. This research will provide the first rigorous evidence of how obesity drives breast cancer in young women and whether the weight loss drugs already being prescribed to millions could also help prevent it.

Category: Predoctoral, Type of Cancer: Colon & Rectum, Melanoma of the skin, Grant *#COCR27PRF007)*

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Project Title: *The Role of Methionine Metabolism in NK Cell Anti-Tumor Activity*

Cancer cells consume nutrients from their surroundings to support their growth and expansion. This creates nutrient competition with immune cells, which rely on the same nutrients to function and kill cancer cells. Natural Killer (NK) cells are important immune cells that help identify and destroy cancer cells. However, they often become dysfunctional in the solid tumor environment, allowing tumor cells to escape. Methionine is an essential amino acid that cells use to control which genes are turned on or off. The goal of this proposal is to understand the mechanisms by which methionine deprivation contributes to NK cell dysfunction and impairs anti-tumor immunity. I will first investigate how methionine availability affects NK cell activity and genes that support cancer-fighting functions. I will then determine whether tumor cells weaken NK cell responses by consuming methionine and whether restoring methionine can improve NK

cell-mediated tumor control. Together, this work will establish methionine as an important regulator of NK cell anti-tumor immunity and will further advance the development of NK cell-based immunotherapies.

Category: Bridge, Type of Cancer: Lung
Grant *#COCR27RBG006*

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Project Title: *Elucidate the mechanism of targeting MTR to enhance anti-tumor immunity in KRAS-driven lung cancer*

Lung cancer caused by mutations in a gene called KRAS is one of the most difficult types of lung cancer to treat, and many patients do not respond well to current treatments, including immunotherapy. Immunotherapy helps the body's immune system recognize and attack cancer cells, but KRAS-driven tumors can develop ways to avoid immune attack. Cancer cells also change how they use nutrients to support their growth and survival. One important metabolic process, called one-carbon metabolism, provides materials needed for cancer cells to make DNA and grow. Although some chemotherapy drugs target this process, tumors often become resistant, creating an urgent need to identify new metabolic targets. Our preliminary studies have identified methionine synthase (MTR), an enzyme that plays an essential role in one-carbon metabolism, as a promising new target in KRAS-driven lung cancer. In this project, we will determine how targeting MTR strengthens the immune response against KRAS-driven lung cancer and how MTR helps tumors avoid immune detection. Ultimately, this research could establish MTR as a new treatment target and provide the foundation for combining MTR-targeted therapies with immunotherapy to improve treatment options and outcomes for patients with KRAS-driven lung cancer.

Category: Predoctoral, Type of Cancer: Kaposi's Sarcoma
Grant *#COCR27PRF009*

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Project Title: *Alternative Splicing of Notch Signaling Components in DNA Tumor Virus Infections*

Research on the virus infections that cause human cancers has been crucial for developing lifesaving interventions for both viral and non-viral cancers. We study infections by the tumor virus Kaposi's sarcoma-associated herpesvirus (KSHV) to uncover important interactions of virus proteins and DNA with those of its host cells. We identified a crucial interaction between a viral protein called Rta and members of a host protein signaling pathway called Notch. Abnormal activity of the Notch pathway is involved in driving many viral and non-viral cancers. By studying KSHV infection, we are uncovering molecular mechanisms that govern healthy and non-healthy functions of Notch signaling. In KSHV infections, the Rta protein is required for the virus to emerge from latency and amplify itself in a process called reactivation. We recently showed that Notch works together with Rta to promote reactivation. Both Rta and Notch interact with a second protein component of the Notch pathway, called RBP-Jk, that binds to DNA to determine the genes that Rta and Notch will activate. Our work has shown unique functions of Rta and Notch, and we have recently identified new, alternative forms of RBP-Jk that we propose distinguish the functions of Rta from Notch. One of these new RBP-Jk proteins, called P10, inhibits KSHV reactivation. We believe that P10 inhibits the ability of Notch to bind to DNA to activate genes. The overall goal of this application is to define how alternative forms of RBP-Jk regulate the basic interactions of Rta and Notch with viral and cellular DNA. We will gain insight into fundamental cellular processes that distinguish health from cancer. Our work has the potential to reveal new cancer biomarkers and therapeutic targets.

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Category: Predoctoral, Type of Cancer: Breast
Grant *#COCR27PRF019*

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Project Title: *Reduced Repair of Estrogen Metabolite-Induced DNA Damage Drives Genomic Instability in BRCA1 Hapoinsufficient Breast Epithelial Cells*

Inherited changes in the BRCA1 gene greatly increases a person's risk of developing breast cancer. BRCA1 normally helps cells repair damage to their DNA, which protects cells from changes that can lead to cancer. Scientists have traditionally thought that breast cells become especially vulnerable only after losing the function of both copies of BRCA1. However, recent work from my laboratory and others suggests that having just one working copy may already make cells less able to repair DNA damage. Estrogen is a hormone that helps breast cells grow, but as the body processes estrogen, it can also produce substances that damage DNA. This may make breast tissue particularly vulnerable in people who inherit a BRCA1 mutation, whose cells may be less able to repair this damage. My research will test whether breast cells with only one working copy of BRCA1 are more sensitive to this estrogen-related damage. I will study breast cells grown in the laboratory, including three-dimensional models made from patient tissue, and measure how much DNA damage and genetic change build up after exposure to these substances. By identifying the earliest changes that occur in breast cells of people who inherit a BRCA1 mutation, this research may help explain why they have such a high risk of breast cancer. It could also point to new ways to prevent cancer before a tumor develops, potentially reducing the need for major preventive procedures.

Category: Pilot, Type of Cancer: Hematologic
Grant *#COCR27PPR013*

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Project Title: *Targeting Ribonucleotide Reductase to Overcome Flt3 Resistance in Acute Myeloid Leukemia*

Acute myeloid leukemia (AML) is an aggressive blood cancer that can be difficult to treat. Although targeted drugs have significantly improved treatment for some patients, leukemia cells often become resistant, causing

the disease to return. Our research seeks to understand how AML cells develop this resistance and identify new ways to overcome it. We discovered that an enzyme called ribonucleotide reductase (RNR), which helps cells produce the building blocks needed to copy and repair DNA, plays an important role in resistance to a class of targeted drugs called FLT3 inhibitors. By blocking RNR with drugs that are already approved for cancer treatment, we were able to restore the effectiveness of FLT3 inhibitors in laboratory and animal models of resistant AML. These findings may provide a practical strategy for repurposing existing drugs and could ultimately lead to more effective treatments for patients with drug-resistant AML.

Category: Pilot, Type of Cancer: Colon & Rectum
Grant *#COCR27PPR039*

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Project Title: *Targeting the Metabolic Enzyme PHGDH in Cancer*

Metabolic reprogramming is a hallmark of cancer that drives tumor progression and represents a promising therapeutic target. The metabolic enzyme PHGDH is often highly expressed in cancer, where it promotes cancer progression and correlates with poor clinical outcomes in cancer patients. To date, the tumor-promoting effects of PHGDH have been mainly attributed to its canonical function in metabolism. PHGDH is being actively pursued as a promising target for cancer therapy. Colorectal cancer (CRC) is the second leading cause of cancer-related deaths in the United States, despite recent advances in chemotherapy and surgery. A better understanding of CRC mechanisms will provide novel therapeutic opportunities, which are urgently needed in the clinic. Our preliminary studies strongly suggest that PHGDH plays a critical role in promoting CRC progression through a novel, noncanonical mechanism independent of its well-established metabolic function. Importantly, our preliminary studies further suggest that this newly identified mechanism could be targeted to develop better treatments for CRC. In this project, we will investigate the role and mechanisms of this noncanonical function of PHGDH in CRC, and furthermore, will determine whether targeting this noncanonical function can improve therapeutic outcomes in CRC. The goal of this study is to discover new mechanisms and treatments for CRC, which are critically needed in clinical practice. We expect that this study will significantly advance our understanding of the mechanisms of CRC and provide a strong foundation for developing new and more effective treatments for patients with CRC.

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Category: Pilot, Type of Cancer: Liver
Grant *#COCR27PPR053*

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Affiliation: *Rowan University*



Project Title: *Diagnostic Biomarkers for Immune Checkpoint Inhibitor-Induced Liver Toxicity in Breast Cancer Patients*

Drug-induced liver injury is reported in up to 60% of cancer patients and can lead to life-threatening complications, treatment delays, and compromising patient care. Early identification of those at high risk for liver toxicity is a major challenge due to variability in patient responses. Although clinical assessments can measure ongoing organ damage, we are unable to predict liver toxicity before damage occurs. We propose to integrate machine learning approaches and pre-treatment biomarkers to predict the risk of liver toxicity in breast cancer patients using data from either biomedical repositories (Specific Aim 1) or from patients actively undergoing therapy (Specific Aim 2). These models could greatly improve patient outcomes as side effects of cancer medications are often more detrimental to the patient's quality of life than the cancer itself.

Category: Pilot, Type of Cancer: Hematologic
Grant *#COCR27PPR060*

Min-Jeong Yang, PhD, My277@ints.rutgers.edu
Affiliation: *Rutgers Institute for Nicotine & Tobacco Studies*



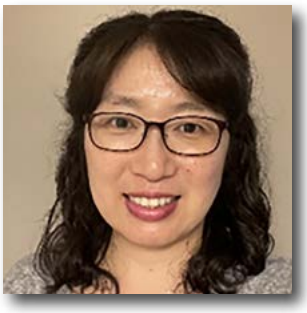
Project Title: *Mindfulness-Based Ecological Momentary Intervention for Non-Cigarette Smoking Cessation (mindEMI-NC) Addressing Cancer-Related Difficulties*

Continuing to smoke after a cancer diagnosis is linked to poorer health outcomes for cancer survivors. Most quitting smoking studies have focused extensively on helping people quit cigarette smoking. However, many cancer survivors also use non-cigarette (NC) combustible products (e.g., cigars, marijuana). These products contain harmful chemicals that can increase cancer risk, yet we have very limited information about their use among cancer survivors and how to support quitting. Therefore, there is a clear need to develop smoking cessation treatments specifically for NC smoking among cancer survivors.
Our team previously developed a smartphone application (app) to help cancer survivors quit cigarette smoking. This app uses ecological momentary assessments

to capture high-risk moments when cancer survivors are likely to smoke due to cancer-specific smoking vulnerabilities (e.g., fear of cancer recurrence). During these moments, the app delivers real-time, mindfulness-based skills to help them cope without smoking. This project aims to: (1) Conduct in-depth interviews with 30 survivors who smoke NC products to understand their experiences and needs; (2) Adapt our team's app to create a prototype app for NC smoking, based on the feedback from these interviews; and (3) Pilot-test the prototype app with the same 30 cancer survivors to evaluate usability and gather feedback. This project will produce a low-cost, scalable intervention ready to be tested in a large-scale clinical trial.

Category: Pilot, Type of Cancer: Breast
Grant *#COCR27PPR026)*

Mi-Hyeon Jang, PhD, mihyeon.jang@rutgers.edu
Affiliation: *Rutgers Institute for Nicotine & Tobacco Studies*



Project Title: *Health Disparities in Chemobrain: The Role of Obesity in Black Breast Cancer Survivors*

Chemotherapy can cause problems with memory, concentration, sleep, and mood, commonly known as "chemobrain," which can significantly affect cancer survivors' quality of life. Black breast cancer survivors often experience more frequent and severe symptoms, but the reasons remain unclear. Our previous research suggests that obesity, which is highly prevalent in this population, may contribute to these problems. Both obesity and chemotherapy can cause brain inflammation and damage brain cells, potentially worsening cognitive difficulties. Our studies have also shown that chemotherapy reduces levels of NAD+, a molecule essential for producing energy and maintaining healthy brain function. Obesity may further reduce NAD+ levels, making the brain more vulnerable to chemotherapy-related damage. We hypothesize that obesity worsens chemobrain by increasing NAD+ loss. To test this, we will examine blood NAD+ levels, obesity, physical activity, and cognitive symptoms in a group of Black breast cancer survivors. We will also use mouse models to investigate how obesity and chemotherapy together affect brain NAD+ levels, inflammation, and cognitive function. In addition, we will test whether veliparib, a drug that helps preserve NAD+ by blocking its excessive consumption, can reduce chemotherapy-related cognitive problems. This research will help us better understand why some cancer survivors experience more severe chemobrain and identify potential ways to prevent or treat it. Ultimately, our findings will provide the foundation for larger studies and the development of targeted strategies to improve brain health and quality of life among Black breast cancer survivors.

2027 Grant Recipients

Category: Bridge, Type of Cancer: Breast; Ovary Pancreas Prostate
Grant *#COCR27RBG001*

Bing Xia, PhD, xiabi@cinj.rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*



Project Title: *Role of PALB2 in the DNA Damage Response and Cancer Suppression*

This project focuses on a key protein called PALB2, which acts as a linker connecting two other important and well-known tumor suppressor proteins, BRCA1 and BRCA2. Together, these three proteins form a vital defense system that repairs broken DNA and ensures cells divide safely. When someone inherits a mutation, or fault, in the PALB2 gene, this defense system breaks down. This leads to a high risk of breast cancer, as well as increased risks for ovarian, pancreatic, and prostate cancers. While we know PALB2 works with BRCA1 to repair damages DNA, we don't fully understand how it signals a cell to stop dividing when its DNA is damaged (a safety feature called a "checkpoint") or how it interacts with other proteins like MRG15 to protect active genes and monitor the copying of DNA in preparation of cell division. In this study, our team will use two unique mouse models that have been genetically modified to have PALB2 disconnected from BRCA1 or MRG15. By studying these mice and the cells derived from these mice, we aim to: (1) Figure out how the connection between PALB2 and BRCA1 helps the cell stop dividing when DNA is broken; and (2) Determine how PALB2 works with MRG15 to influence the stability of our DNA when it is being copied in the cell and the how this affect cancer development. By solving these puzzles, we can provide better information for genetic counseling and develop smarter, more targeted therapies for patients with PALB2-related cancers.

Category: Pilot, Type of Cancer: Hematologic
Grant *#COCR27PPR083*

Renata Feinman-Siegel, PhD, rena.feinman@hmh-cdi.org
Affiliation: *HMH Hospitals Corporation Inc.*



Project Title: *The Systemic Role of Butyrate, a Gut Microbiota-Derived Metabolite, in Multiple Myeloma Progression and Outcome*

Multiple myeloma (MM) is a blood cancer affecting plasma cells in the bone marrow (BM), with over 35,000 new cases annually in the United States. Autologous stem cell transplantation (ASCT) remains a cornerstone therapy, but many patients relapse within 2-3 years. There is an unmet need to discover novel therapies that are both safe and curative. The gut microbiome, made of trillions of bacteria, plays a critical role in immune function and cancer outcomes. High-dose chemotherapy given to MM patients prior to ASCT often destroys "good"

bacteria that produce butyrate with powerful immune-regulating properties. Our preliminary clinical data shows that patients with higher levels of these butyrate-producing bacteria have better treatment responses and longer periods of remission. However, the precise biological pathways linking these gut signals to the BM microenvironment remain poorly understood. This study aims to bridge that gap by investigating how the natural restoration of these beneficial bacteria can stabilize the immune system and inhibit cancer progression after transplant. Using advanced technologies to map gene activity and measure bacterial products, we will track how these microbial signals travel from the gut to the BM to suppress tumor growth in MM mouse models. By uncovering the "Gut-Bone-Marrow axis", this research seeks to establish a scientific foundation for simple, safe dietary interventions that can be integrated into standard oncology care. Our goal is to transform the post-transplant period from a state of vulnerability into one of active defense, ultimately reducing the risk of relapses in newly diagnosed MM patients.

Category: Bridge, Type of Cancer: General (Radiation)
Grant *#COCR27RBG005*

Zhiyuan Shen, MD, PhD, shenzh@cinj.rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*



Project Title: *Radiation-carcinogenesis modulated by SETD4-mediated protein methylations*

Radiation is a double-edged sword. While it is widely used for clinical diagnosis and cancer treatment, it can also cause acute toxicities and long-term adverse effects, including secondary malignancies. To improve the safety of radiation-based applications, we seek to better understand the fundamental mechanisms underlying radiation-induced carcinogenesis and to identify biological factors that can mitigate cancer risk following radiation exposure. Our research focuses on a protein called SETD4, which helps regulate cellular functions by adding small chemical marks, known as methyl groups, to other proteins. In previous studies, we discovered that mice lacking SETD4 recovered more quickly from radiation damage to the blood-forming system and were much later to develop radiation-induced cancers. We subsequently identified two critical amino acid residues that are essential for SETD4 enzymatic activity. Remarkably, mice carrying mutations at these residues phenocopy SETD4 knockout animals, demonstrating that loss of SETD4 catalytic activity alone is sufficient to confer protection against radiation-associated carcinogenesis. These findings suggest that temporarily blocking SETD4 activity during radiation exposure could potentially lower the risk of secondary cancers after radiotherapy. To investigate this possibility, we have developed specialized mouse models that allow us to study how SETD4 influences cancer formation following radiation exposure. Our ongoing work aims to uncover the biological mechanisms through which SETD4 affects cancer risk and to determine whether targeting this pathway could become a new strategy for improving the long-term safety of radiation therapy.

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.

Presenter Biographies

Jian Cao, PhD

Affiliation: *Cancer Metabolism and Immunology Research Program at Rutgers Cancer Institute*

Jian Cao, PhD, is an Associate Professor in the Cancer Metabolism and Immunology Research Program at Rutgers Cancer Institute. He received his PhD. from the Chinese Academy of Sciences in 2009 and completed his postdoctoral training at Yale School of Medicine. He established his laboratory at Rutgers Cancer Institute in 2019. His research focuses on cancer epigenetics, tumor-intrinsic immune signaling, oncoviral mechanisms of cancer, and strategies to improve cancer immunotherapy. His laboratory investigates how viral integrations and epigenetic alterations drive tumor development and create therapeutic vulnerabilities, with ongoing studies in HBV-associated liver cancer, HPV-associated cancers, and engineered T cell therapies. Dr. Cao also collaborates on translational efforts to develop TCR-T cell therapies for virus-associated malignancies.

Rebecca Risman, PhD

Affiliation: *Rutgers University – Biomedical Engineering*

Rebecca Risman, PhD completed her doctorate in Biomedical Engineering at Rutgers University. Under the guidance of Dr. Valerie Tutwiler, she studied the role of blood clot structure in regulating clot degradation (fibrinolysis). The New Jersey Commission on Cancer Research funded the investigation of pancreatic cancer in altering clot structure and impairing fibrinolysis in a subset of patients, which was recently published in the Journal of Thrombosis and Thrombolysis as part of a collection of articles on "Thrombosis in patients with cancer: challenges and advances." She is currently a postdoctoral fellow mentored by Dr. John Weisel at the University of Pennsylvania studying clot mechanics as predictors of thrombosis. Her research is supported by the Hemostasis and Thrombosis Research Society's Seed Grant to study cancer-associated thrombosis. She serves as a member of the International Society of Thrombosis and Haemostasias Early Career Committee and the social media manager of the International Fibrinogen Research Society.

Moamen M. Elmassry, PhD

Affiliation: *Princeton University, Department of Molecular Biology*

Moamen Elmassry, PhD, received his Bachelor of Pharmaceutical Sciences from the Faculty of Pharmacy at Cairo University, Egypt, in 2012, followed by a PhD in Biology from Texas Tech University in 2020. He is currently completing his postdoctoral training in the Donia Lab at Princeton University, where his research focuses on gut microbiome, microbiome-derived small molecules, and their roles in human disease. In January 2027, he will join Texas Tech University as an Assistant Professor.

Maria Ibrahim, PhD

Affiliation: *Rutgers Institute for Nicotine & Tobacco Studies*

Maria Ibrahim, PhD, earned her doctorate from the Rutgers Cancer Institute of New Jersey, where she worked in Dr. Eileen White's lab. Her research focuses on nutrient pathways in cancer cachexia. She is currently continuing this work as a postdoctoral researcher in the White lab.

Svetlana Bagdasarov, PhD

Affiliation: *Libutti Laboratory at the Cancer Institute of New Jersey, and Rutgers New Jersey Medical School*

Svetlana Bagdasarov, PhD, is a fourth year medical student at Rutgers Robert Wood Johnson Medical School. She completed her PhD in the Libutti lab at the Cancer Institute of New Jersey where she worked on an antibody-based immunotherapy to treat pancreatic neuroendocrine tumors. Her work has been supported by funding from the NJCCR Predoctoral Fellowship and NJ ACTS CTSA TL1 Predoctoral Fellowship.

Gary Kwok, PhD

Affiliation: *Center for Discovery & Innovation, Hackensack Meridian Health*

Gary Kwok, PhD, is an Assistant Member at the Center for Discovery and Innovation. His research focuses on improving psychosocial outcomes and quality of life among adolescent and young adult (AYA) cancer survivors as they navigate cancer survivorship alongside critical developmental milestones. His work integrates behavioral science, digital health, and implementation science to develop scalable, accessible, and equitable supportive care interventions for this underserved population. Dr. Kwok's long-term research program centers on two overarching goals: (1) developing evidence-based digital health interventions that address the psychosocial needs of AYA cancer survivors and (2) identifying effective implementation strategies to integrate and sustain these interventions across healthcare and community settings. As a New Jersey Commission on Cancer Research (NJCCR) grantee, he examines the multilevel barriers and facilitators to implementing mindfulness-based mobile health (mHealth) interventions in AYA survivorship care. By identifying these factors, his research aims to develop and test implementation strategies that promote the adoption and long-term sustainability of mindfulness-based mHealth interventions among both healthcare providers and AYA cancer survivors.

Victor Shengkan Jin, PhD, Professor

Affiliation: *Rutgers, The State University*

Victor Shengkan Jin, PhD, received his BS degree in Biology from Tsinghua University, and his PhD in Pharmacology from Cornell Weill Medical School/Sloan Kettering Cancer Center. Dr. Jin continued his postdoctoral training at Rockefeller University in Cancer Biology. In 2003, Dr. joined the faculty of Pharmacology

Department at Rutgers University-Robert Wood Johnson Medical School, where he is now a full professor. Dr. Jin has made important contributions in diverse research areas including p53, autophagy, and cancer metabolism. Recently, Dr. Jin's lab shifted research focus to developing CRISPR gene editing technologies for gene and cell therapy applications. Dr. Jin invented the Pin-pointTM base editing platform, which is being used by global pharma and agriscience companies to support therapeutic development and crop improvement, respectively. Dr. Jin was awarded Thomas Edison Patent Award in 2022.

Carol Lutz, PhD

Affiliation: *Rutgers Biomedical and Health Sciences*

Carol Lutz, PhD, (BS Biology with Highest Honors UNC, PhD Duke University), is an Associate Professor in the Department of Microbiology, Biochemistry and Molecular Genetics at Rutgers Health-New Jersey Medical School. She is also an Associate Dean for Student Affairs in the Rutgers School of Graduate Studies. She teaches medical, dental, and graduate students, runs an extramurally funded research lab studying gene expression regulation in lung cancer, and works with more than 600 graduate students in administrative, academic, wellness and student affairs functions. Carol volunteers her time as a grant and fellowship reviewer for multiple local, regional, national and international agencies, and serves on the board of directors for the YWCA of Union County NJ, and the Central New Jersey Alumnae Panhellenic Foundation Board of Trustees.

Coral Omene, MD, PhD

Affiliation: *Rutgers Cancer Institute*

Dr. Coral Omene is a breast medical oncologist at Rutgers Cancer Institute, and she serves as the Program Director, Breast Cancer Disparities Research at Rutgers Cancer Institute. She has a passion for women's health and is dedicated to the care and management of a diverse pool of breast cancer patients. She began her career upon completion of a combined MD/PhD degree at Columbia University College of Physicians and Surgeons. This was followed by an Internal Medicine residency at New York Presbyterian Hospital-Columbia University Medical Center. After residency, she pursued a Heme/Onc fellowship at NYU School of Medicine. She conducts extensive translational and clinical research in all subtypes of breast cancer with particular areas of interest including triple negative breast cancer; the interplay with obesity, breast cancer and treatment outcomes; Metabolomics and immune cell metabolism in breast cancer, and in the area of breast cancer disparities. She is a Principal Investigator for numerous clinical trials and serves on multiple national committees including as the Co-Chair of the BIG Ten Cancer Research Consortium Breast Cancer Clinical Trial Working Group and Co-Chair of the BIG Ten CRC Health Equity Committee.

Dongfang Liu, PhD

Affiliation: *Department of Pathology, Immunology and Laboratory Medicine, Center for Immunity and Inflammation, Rutgers New Jersey Medical School*

Dongfang Liu, PhD, is a Professor at the Department of Pathology, Immunology, and Laboratory Medicine in Rutgers University - New Jersey Medical School (NJMS). He also serves as the Scientific Director of the Cellular Imaging & Histology Core at Rutgers-NJMS. In 2012, Dr. Liu was recruited to Baylor College of Medicine as a tenure-track Assistant Professor in the Departments of Pediatrics, and Pathology & Immunology. In 2015, he joined the Houston Methodist Research Institute (HMRI, affiliated with Weill Cornell Medical College (WCMC)/Cornell University) as a tenure-track Assistant Professor. He was promoted to Associate Professor in 2018 in HMRI. Dr. Liu earned his PhD in immunology from 2001 to 2005 in Huazhong University of Science and Technology (HUST), with a focus on natural killer (NK) biology and immunology, his medical training in Wuhan University School of Medicine in 2001. After completing his PhD training in HUST, he joined the National Institutes of Health (NIH) for his postdoctoral training from 2005 to 2011, specializing in the immunobiology of NK cells and immunological synapse. After completing his postdoctoral training at NIH, he joined the Ragon Institute of MGH, MIT, and Harvard in 2011 as a Senior Research Scientist, focusing on HIV-specific immune cell dysfunction and immunological synapse. Dr. Liu's current research focuses on the immunobiology of NK cells, Chimeric Antigen Receptor (CAR)-NK cells, and immunological synapse biology and application. With over 20 years of experience in NK cell research and > 10 patent applications, Dr. Liu has published extensively in top-tier journals, including Nature Immunology, Immunity, Nature Communications, JACI, and the Proceedings of the National Academy of Sciences (PNAS). He serves on several editorial boards and acts as a reviewer for numerous journals and grant agencies, including the NIH and DOD. Dr. Liu holds multiple federal research grants to study the basic mechanisms of NK cell biology and CAR-NK.

Jie Li, MPH, PhD

Affiliation: *New Jersey State Cancer Registry*

Jie Li, PhD earned her MPH and PhD from the Rutgers School of Public Health. She is a Research Scientist at the New Jersey State Cancer Registry, where she works on cancer surveillance research and population-based patient contact studies examining cancer incidence and trends, cancer risk factors, health disparities, and cancer survival.

Panel Members

Panel Discussion:

Career Pathways and Employment Opportunities

Daniel Herranz, PharmD, PhD

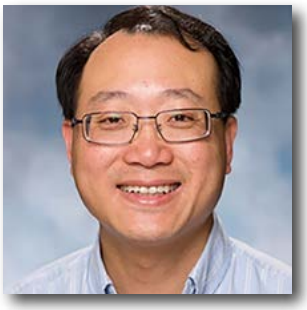
Associate Professor of Pharmacology and Pediatrics at Rutgers University and Associate Director for Shared Resources at Rutgers Cancer Institute



Daniel Herranz, PharmD, PhD, is a tenured Associate Professor and Associate Director for Shared Resources at Rutgers Cancer Institute. He is a translational scientist whose research focuses on the roles of metabolism, epigenetics, and non-coding enhancers in cancer, with a particular emphasis on T-cell acute lymphoblastic leukemia (T-ALL). His laboratory uses genetically engineered mouse models and other preclinical systems to uncover novel therapeutic targets, and he has maintained continuous NIH/NCI funding since 2015. Dr. Herranz earned his PhD. under the mentorship of Dr. Manuel Serrano, where he investigated the role of SIRT1 in metabolism and cancer, resulting in multiple high-impact publications, including Nature Communications, PNAS, and Nature Reviews Cancer. During his postdoctoral training with Dr. Adolfo Ferrando at Columbia University, he led pioneering studies identifying the oncogenic enhancer N-Me that regulates MYC expression in T-ALL and uncovered metabolic mechanisms underlying NOTCH1-driven leukemia, with findings published in Nature Medicine. Since establishing his independent laboratory at Rutgers Cancer Institute in 2017, Dr. Herranz has built a nationally recognized research program focused on enhancer biology and cancer metabolism. His work has been published in leading journals, including Blood, Blood Cancer Discovery, Leukemia, Molecular Systems Biology, and Trends in Cancer. An accomplished mentor, Dr. Herranz has guided trainees who have received numerous competitive awards, including multiple New Jersey Commission on Cancer Research fellowships, and several former postdoctoral fellows have advanced to independent faculty and industry leadership positions.

Zhaohui Feng, MD, PhD

Professor in the Department of Radiation Oncology and the Department of Pharmacology, Rutgers Cancer Institute, Rutgers University



Zhaohui Feng, MD, PhD, is a tenured Professor in the Department of Radiation Oncology and the Department of Pharmacology at Rutgers Cancer Institute, Rutgers University. Since joining the Rutgers Cancer Institute faculty in 2008, Dr. Feng has established a strong research program focused on the molecular mechanisms of tumor suppression, cancer metabolism, and therapeutic resistance. Dr. Feng has made significant contributions to understanding tumor suppressor p53 signaling and gain-of-function mutant p53 in cancer and other diseases. His laboratory has identified multiple novel regulators and regulatory mechanisms of p53 and mutant p53, including E3 ubiquitin ligases, microRNAs, and post-translational modifications such as SUMOylation and palmitoylation. More recently, his research has focused on metabolic reprogramming in cancer, elucidating how altered metabolism drives tumor progression and identifying novel metabolic vulnerabilities for therapeutic intervention. Dr. Feng has published more than 100 peer-reviewed articles. His research has been continuously supported by the National Cancer Institute (NCI), the U.S. Department of Defense (DoD), and the New Jersey Commission on Cancer Research.

Katie Devine, PhD

Associate Director of the New Jersey Pediatric Hematology and Oncology Research Center of Excellence, Rutgers Robert Wood Johnson Medical School and Rutgers Cancer Institute



Katie Devine is the Associate Director of the New Jersey Pediatric Hematology and Oncology Research Center of Excellence (NJPHORCE), Professor of Pediatrics, and Section Chief of Pediatric Population Science, Outcomes, and Disparities Research at Rutgers Robert Wood Johnson Medical School and Rutgers Cancer Institute. Her research focuses on the psychosocial aspects of pediatric, adolescent, and young adult cancer survivorship, including patient and family adaptation to illness, adherence to medical recommendations and survivorship care, transitions in care, and health promotion for survivors. Her work focuses on the unique needs of adolescent and young adult (AYA) survivors who must navigate the challenges of cancer care during normative developmental transitions. Her work has been funded by the National Institutes of Health since 2014.

Panel Members

Binfeng Lu, PhD

Professor at Hackensack Meridian School of Medicine



Binfeng Lu, PhD., is a Member at the Center for Discovery and Innovation at Hackensack Meridian Health and a Professor at Hackensack Meridian School of Medicine. His research focuses on cytokine biology, tumor immunology, and cancer immunotherapy. His laboratory has made pioneering contributions to defining how IL-33 and IL-36 regulate antitumor immunity and the tumor microenvironment. His earlier work helped establish TIM-3 as an important tumor-associated immune checkpoint, while more recent studies from his laboratory have identified regulatory T cell–cancer-associated fibroblast interactions as a mechanism of stromal immunosuppression and resistance to antitumor immunity. Supported by NIH and Department of Defense awards, his translational research seeks to harness cytokines for cancer therapy through tumor-targeted fusion proteins, oncolytic viruses, and nanoparticle-based delivery approaches. Dr. Lu has served as a reviewer and chair for NIH and Department of Defense cancer research panels and teaches immunology to medical and graduate students.

Antoinette M. Stroup, PhD

Director of Cancer Epidemiology Services (CES)
New Jersey State Cancer Registry



Antoinette M. Stroup, PhD, is the Director of Cancer Epidemiology Services and the New Jersey State Cancer Registry. She has been the Director of CES since 2013, overseeing all administrative, operational, and research activities of New Jersey's population-based cancer surveillance system. Dr. Stroup has a PhD in Epidemiology from the University of California, Berkeley. She is a Professor in the Department of Biostatistics and Epidemiology at the Rutgers School of Public Health, Principal Investigator for New Jersey's SEER Program contract funded by the National Cancer Institute, and Program Director for New Jersey's CDC National Program of Cancer Registries. In addition to her role as CES Director, 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.

Raymond Birge, PhD

Vice Chair, Department of Microbiology, Biochemistry, and Molecular Biology, Rutgers University



Raymond Birge, PhD, received a Bachelor of Science from the University of Connecticut in 1984, and a PhD in Biochemical Toxicology in 1989 from the University of Connecticut under the tutelage of Professor Edward Khairallah. He completed postdoctoral training at The Rockefeller University in New York City with Professor Hidesaburo Hanafusa from 1989 to1994. He was Assistant Professor at Rockefeller University from 1994 to 1998, and acting head of the Laboratory of Molecular Oncology at Rockefeller University from 1998-2000. Dr. Birge joined Rutgers in 2000 (formerly UMDNJ) and currently holds leadership positions including Professor and Vice Chair for Research in the Department of Microbiology, Biochemistry and Molecular Biology and Director of the Center for Cell Signaling at Rutgers Biomedical and Health Sciences. He is Editor-in-Chief of Cell Communication and Signaling (BMC Springer) and Chief Scientific Officer at Targeron Therapeutics. Dr. Birge was one of the founding members of the International Cell Death Society (CDS), and currently serves as the Vice President and Executive Board member of ICDS. His work focuses on Cell Death and Cancer and has authored over 100 papers in signaling and cancer biology.

Tessa Bergsbaken, PhD

Associate Professor, Rutgers New Jersey Medical School



Tessa Bergsbaken, PhD, is an Associate Professor at Rutgers New Jersey Medical School. Research in her laboratory is focused on development of adaptive immune responses in the intestine and the unique requirements for differentiation and persistence of T lymphocytes in this tissue during infection and tumorigenesis. Current research addresses the plasticity of tissue-resident memory subsets, the unique contributions of tissue-resident T cell subsets during secondary infection, and the regulatory mechanisms restraining T cell responses in tissues.

Meet the Commission Members

Kenneth Adler, MD

Chair

Kenneth Adler, MD, 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, MD, FACP

Vice-Chair

Generosa Grana, MD, is the Director of MD Anderson Cancer Center at Cooper. She is also a Professor of Medicine at Cooper Medical School of Rowan University and 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's Silver Chalice Award and the Susan G. Komen for the Cure "Light of Life" Award. Dr. Grana serves on the Board of Trustees of Cooper University Health Care, the Cooper Foundation, the Coriell Institute for Medical Research, and the College of Physicians of Philadelphia.

Wendy Budin, PhD, RN-BC, FAAN

Wendy Budin, PhD, 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, MD, FACP, CPE, MBA

Michele Donato, MD, 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.

She 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. Dr. Donato 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.

Jane Flint, PhD

Jane Flint, PhD, 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 6th edition.

Li Li, PhD, DABT

Li Li, PhD, is currently the Executive Director at the Novartis Institute for Biomedical Research, where he has worked for over 20 years. He received his PhD, 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

Kathleen Scotto, PhD

Kathleen Scotto, PhD, 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 PhD 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.

Christine Schell, MPA
NJDEP Commissioner's Designee

Christine Schell, MPA, is a 30+ year veteran of the New Jersey Department of Environmental Protection (NJDEP) and currently manages the Office of Environmental and Public Health Analysis. In partnership with the New Jersey Department of Health, Christine jointly coordinates the state Environmental Public Health Tracking Program through a CDC grant. She also 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 meaningful actions on a broad array of issues. These issues include environmental justice, climate resilience, and sustainability. In addition to representing NJDEP Commissioner on the NJCCR, she serves as the Commissioner's designee as ex-officio on the NJ One Health Taskforce. Christine 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.

Uta Steinhauser, MPH
NJDOH Commissioner's Designee

Uta Steinhauser, MPH, is a career public health professional with over 20 years of experience in the field. Since 2010, Uta has served as Community Health and Wellness Unit (CHWU) evaluation and surveillance lead. 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 also 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. Uta 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



NJCCR Pediatric Cancer Research Advisory Group

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Cora Connor
Caregiver



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