New Jersey Commission on Cancer Research

Annual Report 2024–2025

Dedicated to conquering cancer through scientific research

NJCCR NEW JERSEY COMMISSION ON CANCER RESEARCH

NJHealth New Jersey Department of Health

Annual Report 2024–2025

NJCCR
NEW JERSEY COMMISSION ON CANCER RESEARCH

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

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A Message From the Chair

The Honorable Philip D. Murphy,
Governor of New Jersey
Office of the Governor
125 West State Street
Trenton, New Jersey 08608

Dear Governor Murphy:

On behalf of the New Jersey Commission on Cancer Research (NJCCR), I am pleased to present the Annual Report for Fiscal Year 2024.

Cancer remains the second leading cause of death in New Jersey, followed by heart disease. The Commission remains committed to advancing cancer research in New Jersey. This year the Commission announced new research opportunities, ranging from translational research to racial and ethnic cancer disparities. We are confident that these new opportunities will afford insight into the scientific reasons for these glaring disparities in New Jersey.

The Commission remains optimistic on finding a cure for cancer through continued research by scientists in qualified research institutions in New Jersey. I wish to express my gratitude and thank you for your support of our cancer research work.

Sincerely,

Kenneth Adler

Kenneth Adler, MD, FACP
Chairperson, New Jersey Commission on Cancer Research

Executive Summary

The New Jersey Commission on Cancer Research (NJCCR), established by P.L. 52:9U-1 in 1983 under Governor Thomas Kean, is governed by the "Cancer Research Act." The legislative mandate of the Commission is to review and authorize approved cancer research projects in New Jersey.

Cancer is the second leading cause of death in New Jersey, claiming approximately 15,000 lives annually. It also accounts for the most years of potential life lost (premature mortality) among the state's population, with around 57,000 residents diagnosed each year. In 2021, the National Cancer Institute ranked New Jersey eighth in the nation for cancer incidence rate (per 100,000).

Cancer research plays a crucial role in increasing survival rates and enhancing the quality of life for those affected by improving treatment options, developing targeted therapies, and advancing early detection methods. Through ongoing research funded by the NJCCR, scientific knowledge regarding cancer prevention, detection, and treatment continues to advance, improving cancer outcomes for the population in New Jersey.

The 2024 annual report highlights the significant strides made possible through medical research. These investments i.e., cancer research grants have catalyzed a wave of greater understanding of the biological complexities of cancer and accelerated the development of more effective treatment. In 2024, the NJCCR funded 21 grants proposal ranging from $100,000 to $200,000 for two years. These proposals provided support for predoctoral fellowship, postdoctoral fellowship, bridge grants, and pilot grants. This report provides an overview of each type of grant award and highlights the impact on various cancer types. It also emphasizes the need for consistent and stable funding to advance cancer research.

Additionally, the report addresses NJCCR activities, additional funding sources, the grant application and review process, and financial statements. For questions about this report, please e-mail njccr@doh.nj.gov.

Introduction

Established in 1983, the New Jersey Commission on Cancer Research (NJCCR) was created to promote and fund cancer research conducted by scientists at qualified research institutions in New Jersey. NJCCR membership consists of 11 volunteer members appointed by the Governor with the advice and consent of the Senate. These include commissioners of the Department of Health and the Department of Environmental Protection or their designees, along with nine New Jersey citizens or individuals associated with the State who are recognized for their expertise, experience, or interest in medical research.

This NJCCR Annual Report 2024 is prepared in compliance with the enabling statute §52:9U-1, also known as the Cancer Research Act, which mandates that the NJCCR provide a report to the Governor and Legislature detailing the Commission's activities and the outcomes of its funded research initiatives. A copy of the statute is included as Appendix 1. The legislation dictates that NJCCR receive no less than $1 million annually for research into the causes, prevention, and treatment of cancer. In more recent years, NJCCR received a $4 million State appropriation.

Over its 41-year history, NJCCR has awarded more than $56 million in funding, supporting 936 peer-reviewed cancer research grants and student fellowships. It remains the only statewide commission in New Jersey dedicated to providing peer-reviewed cancer research funding to researchers through a merit-based process. This rigorous system has a proven track record of supporting groundbreaking research through fellowships and grants to cancer researchers. The value of NJCCR's contributions extends beyond state borders. An independent analysis of NJCCR grant recipients revealed that researchers leveraged $10 in federal funding for every $1 of NJCCR funding, highlighting the critical role of state investment in scientific advancements.[1]

Looking to the future, NJCCR recognizes the vital need for sustained and future funding to advance cancer research in New Jersey. NJCCR remains optimistic about securing predictable and stable funding to drive future research. Recent advances in genomics and immunology have ushered in innovative treatments that are transforming cancer care. Continued investment in these fields will enable New Jersey scientists to remain at the forefront of medical innovation. NJCCR is steadfast in its mission to support research that addresses the challenges of reducing the negative impact of cancer on New Jersey residents.

NJCCR was established in 1983 to promote and fund cancer research projects to scientists at qualified research institutions in New Jersey. Throughout its 41-year history, the NJCCR has awarded over $56 million for 936 peer-reviewed cancer research grants and student fellowships.

1. *Hanson, C. S., Schneider, D., & Hill, A. M. (2008). Seed grants as a means of stimulating cancer research funding. Health policy, 88(2-3), 243-249.*

Mission and Goals

The mission of the New Jersey Commission on Cancer Research is to: ***Ensure that the citizens of New Jersey receive the fullest benefit of our nation's fight against cancer through the promotion and funding of research into the causes, prevention, survival, and treatment of cancer.***

Simply stated, the Commission's goals are:

- To support meritorious research projects that advance the understanding of prevention, diagnosis, treatment, and survivorship of cancer.
- To support the progression of research from bench to bedside.
- To enhance the reputation of New Jersey as a leader in funding cancer research.
- To facilitate the initiatives of New Jersey scientists to obtain grants from sources such as the National Institutes of Health.
- To support promising investigators experiencing a short-term interruption in funding for research focused on cancer prevention, diagnosis, treatment, and survivorship.

More specifically, the NJCCR works to:

- Advance the field of scientific cancer research in New Jersey by encouraging established scientists to apply their expertise to cancer research.
- Foster integration of emerging technologies such as liquid biopsies and artificial intelligence.
- Nurture future generations of cancer researchers by supporting scientists and pre-doctoral and post-doctoral fellows.
- Disseminate the research findings at the Commission's annual cancer research symposium.
- Compile and update the NJCCR directory of all cancer research projects in the State.

Membership and Organization

Created as a semi-independent public body, the New Jersey Commission on Cancer Research (NJCCR) is "...allocated within the Department of Health but not withstanding that allocation, the commission shall be independent of any supervision or control by the department or by any board or officer thereof." It is subject to all the administrative rules and procedures of the Department, but is not part of the Department's budget.

The Commission establishes and oversees the administrative operations of the grant-making process as well as other programmatic activities that are implemented by its administrative staff. The Governor appoints 11 uncompensated members to serve on this committee. These members include the Commissioners of the New Jersey Department of Health, the Department of Environmental Protection (or their appointed designees), and nine citizens of New Jersey. Their term of service is three years, and their appointments are made with the advice and consent of the Senate.

New Jersey residents wishing to be considered for an appointment may submit their name to the Governor's Office of Appointments.[2]

Administrative Functions

The Commission's leadership and staff provide vital linkages to implement its programs and ensure day-to-day operations. The office staff manages the operations including program administration, interaction with applicants and grantees, contract administration, budgeting, and financial matters, record-keeping, reporting, and managing outside contracts such as advisory consultants. The administrative staff is also responsible for coordinating and overseeing all activities, including the scientific merit review process, engaging in negotiations with external vendors, and managing the essential relationships within the state government.

Looking to the future, NJCCR recognizes the vital need for sustained and future funding to advance cancer research in New Jersey. NJCCR remains optimistic about securing predictable and stable funding to drive future research.

2. Information on how to apply can be found at: https://www.nj.gov/governor/admin/bca.

Cancer as a Public Health Challenge in New Jersey

Cancer remains a public health challenge in New Jersey and the nation. The statistics below highlight this public health challenge and its impact in New Jersey.

- Cancer remained a significant cause of mortality, accounting for 18.4% of deaths in 2021. It ranked **second** in terms of causes of death, trailing behind the heart diseases.[3]
- National Cancer Institute has ranked New Jersey as **eighth in the nation** for the age adjusted incidence rate for cancer (all cancer sites)[4] for year 2021 and is ranked **47th** among all the states for age adjusted cancer deaths (all cancer sites)[5] for year 2022.
- According to the American Cancer Society, in New Jersey, approximately 57,740 individuals are projected to receive a cancer diagnosis and 15,110 people are expected to die in year 2024[6], which is slightly higher than 2023 projections.[7]
- According to the American Cancer Society, new cancer diagnosis for New Jersey is estimated for selected cancers for year 2024 as follows:[6]
  - Prostate: **9,860**
  - Female breast: **8,880**
  - Lung and bronchus: **5,600**
  - Colon and rectum: **4,240**
  - Melanoma of the skin: **2,330**
  - Non-Hodgkin lymphoma: **2,490**
  - Uterine corpus: **2,230**
  - Urinary bladder: **2,540**
  - Leukemia: **1,940**
  - Uterine cervix: **370**
- Similarly, according to American Cancer Society, new cancer death for New Jersey is estimated for selected cancers for year 2024 is as follows:[6]
  - Lung and bronchus: **2,700**
  - Pancreas: **1,440**
  - Colon and rectum: **1,330**
  - Female breast: **1,170**
  - Prostate: **740**

3 https://www-doh.state.nj.us/doh-shad/indicator/view/LCODall.Count10.html
4 https://statecancerprofiles.cancer.gov/incidencerates/index.php
5 https://statecancerprofiles.cancer.gov/deathrates/index.php
6 https://acsjournals.onlinelibrary.wiley.com/doi/full/10.3322/caac.21820
7 https://acsjournals.onlinelibrary.wiley.com/doi/10.3322/caac.21763

- Leukemia: **630**
- Liver and intrahepatic bile duct: **620**
- Non-Hodgkin lymphoma: **520**
- Brain and other nervous system: **500**
- Ovary: **340**

These figures underscore the critical need for ongoing research in cancer prevention, early detection, and treatment. NJCCR promotes significant and original research into the causes, prevention and treatment of cancer and serves as a resource to providers and consumers of cancer services.

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Current Grant Programs

The NJCCR grant programs are designed to provide scientific opportunities that attract research scientists. These awards aim to promote collaboration among cancer researchers in New Jersey and spark innovative research. The purpose of the NJCCR grants is to advance research rather than provide long-term support. NJCCR anticipates that this support will help investigators advance cancer research and create opportunities for securing larger federal grants, such as those from the National Institutes of Health and Centers for Disease Control and Prevention. The types of grants funded by NJCCR include the following:

1) **NJCCR Predoctoral Fellowship Grant:** The NJCCR Predoctoral Fellowship Grant provides two-year fellowship awards to attract and retain talented scientists who wish to pursue a career in cancer research in New Jersey.

2) **NJCCR Postdoctoral Fellowship Grant:** The NJCCR Postdoctoral Fellowship Grant provides two-year fellowship awards to retain postdoctoral scientists who have devoted their careers to cancer research in New Jersey.

3) **NJCCR Research Bridge Grant:** The NJCCR Research Bridge Grant is designed to enhance cancer-related research at New Jersey institutions by providing funding to motivated and productive investigators who anticipate a short-term interruption in funding for research projects focused on cancer prevention, diagnosis, treatment, and survivorship. This two-year funding helps position New Jersey investigators to compete for National Institutes of Health (NIH) and/or other federal grant awards.

4) **NJCCR Pilot Program Research Grant:** The NJCCR Pilot Program Research Grant is designed to support cancer research projects that address priority areas such as: health disparities; the impact of COVID-19 on access, disease progression, treatment, and outcomes of persons with cancer; palliative care, pain management and the psychosocial effects of cancer.

5) **NJCCR Pediatric Research Grant:** The NJCCR Pediatric Research grants are intended to encourage and support projects that address causes, prevention, education, treatment, or cure of pediatric cancer as well as address the symptoms or effects that pediatric populations encounter after completing cancer treatment.

Grants Application and Review Process

The NJCCR grant application and review process is designed to follow similar standards used by the National Institutes of Health (NIH).[8] This ensures a fair and thorough evaluation of research proposals. Over time, this approach has gained respect from both grant recipients and applicants.

Application Process

NJCCR Grant applications are submitted electronically by applicants using the System for Administering Grants Electronically (SAGE).[9] This online system ensures easy access, convenience, and flexibility, while reducing administrative workloads for applicants, the NJCCR office, and the scientific merit review panel.

Grant Review Process

The grant evaluation process consists of a two-step process:

1) **Administrative Review:** Upon receipt, all grant applications are reviewed by NJCCR staff for compliance with all applicable New Jersey State statutes and regulations to ensure completeness and accuracy.

2) **Scientific Merit Review:** An independent expert panel of scientific merit reviewers who use the NIH scoring guidelines to evaluate grant applications, provide critiques and assign scores. While their feedback and scores are considered for funding decisions, the final authority to authorize grants rests with NJCCR, as mandated by New Jersey Statute N.J.S.A. 52:9U-1.[10]

NJCCR anticipates that this support will lead investigators to advance cancer research and provide opportunities for securing larger federal grants.

8 https://www.niaid.nih.gov/grants-contracts/review-process

9 https://dohsage.intelligrants.com/

10 https://www.nj.gov/health/ces/documents/njccr/cancer-commission-on-research-legislation-NJSA-529U.pdf

Drive Hope Forward Campaign

Cancer remains the second leading cause of death in New Jersey. To raise awareness about cancer and highlight the need for cancer research as well as stimulate donations, NJCCR embarked on a campaign, entitled, *Drive Hope Forward*. In 2022, NJCCR solicited the help of a multicultural marketing firm to plan and implement the campaign.

Since its inception, the campaign has focused on the planning and implementation of digital marketing ads for use on the various NJDOH platforms. In addition, short video clips have been produced to inform communities and key stakeholders about cancer research. On the next page, you can find QR codes to view the video clips for the *Drive Hope Forward* campaign.

Research is the backbone of scientific progress against cancer. It spurs the development of innovation, medical breakthroughs, and better approaches to preventing, detecting, diagnosing, treating, and curing some of the many diseases known as cancer.

NJCCR *Drive Hope Forward* Campaign

For example, funding by NJCCR has resulted in the discovery of life-saving scientific breakthroughs, including the recent discovery of a unique gene by scientists at Princeton University and the Cancer Institute of New Jersey. Specifically, this discovery resulted in the finding of the gene (Metadherin) critical to breast cancer metastasis. Most recently, another discovery by Dr. Sharon Pine, formerly of Rutgers Robert Wood Johnson Medical School and currently at the University of Colorado, has helped pave the way for the discovery of liquid biopsies that have the potential to impact the early detection, diagnosis, and treatment of breast cancer.

Research continues to be our best defense against cancer because it is the driving force behind all clinical and policy advances that can improve cancer prevention, detection, diagnosis, treatment and, increasingly cures for cancer.

Despite more than 41 years of funding lifesaving research and progress, its impact is limited by a lack of stable and predictable funding. Although the NJCCR receives no less than $1 million annually for cancer research, the Commission has not had a significant increase in funding since 1983. To advance cancer research and facilitate robust innovation and new therapies aimed at treating cancer, a dedicated non-lapsing fund would provide the stability and predictability in funding needed.

Since its inception, the Drive Hope Forward Campaign has focused on the planning and implementation of digital marking ads for use on the various NJDOH platforms.

Drive Hope Forward Video Clip #1

Drive Hope Forward Video Clip #2

NJCCR Funding

Aside from the annual $1 million to $4 million State budget appropriation, NJCCR receives funding from other sources, such as State income-tax checkoffs and the purchase of a Conquer Cancer License Plate by New Jersey residents. This specialty license plate is making good on its promise to "Drive Hope Forward" and advance cancer research. Since its inception in 1998, over 100,000 license plates have been sold, and more than $5.7 million have been raised for cancer research in New Jersey.

Other sources of funding for cancer research include the following:

New Jersey Breast, Prostate, Lung and Pediatric Cancer Research Funds

- NJCCR administers targeted funds for cancer research. The New Jersey Breast Cancer Research Fund was created in 1995 as a result of legislation §54A:9-25.7. This Fund provides each taxpayer with the opportunity to indicate on a New Jersey gross income tax return a portion of the taxpayer's tax refund. Alternatively, individuals can make a contribution to be deposited in this special fund. For example, New Jersey Breast Cancer Research Fund: Individuals can contribute any dollar amount to this Fund.
- Created by statute §54A:9-25.21, this law established the New Jersey Prostate Cancer Research Fund. This fund allows taxpayers the opportunity to dedicate a portion of their tax refund or make a contribution to be deposited in this special fund.
- Similarly, §52:9U-6.3 ushered in the New Jersey Lung Cancer Research Fund to allow NJCCR to solicit, receive, evaluate, and approve applications from qualified research institutions for grants from the New Jersey Lung Cancer Research Fund. Under this statute, a "qualified research institution" shall mean an academic medical institution, State or local government agencies, public or private organizations within New Jersey, and any other institution approved by the commission, which is conducting a lung cancer research project.
- NJCCR administers a special fund known as the New Jersey Pediatric Cancer Research Fund §54A:9-25.47 (C.52:9U-4). This fund allows taxpayers the opportunity to indicate on a New Jersey gross income tax return a portion of their refund or an enclosed contribution to the fund.

New Jersey Pediatric Cancer Research Fund & New Jersey Pediatric Cancer Research Advisory Group

According to the State Cancer Profiles, the incidence rate for childhood (<15 y/o) cancer in New Jersey was 17.5 (per 100,000) for the period between 2014-2018 with the rate stable. Although mortality rates for cancer within this group have declined (due to major advances in treatment modalities), it is still the second leading cause of death in children under the age of 15 years, behind accidents.

In 2021, Senate bill 1431 ushered in the Pediatric Cancer Research Fund, which resulted in a one-time State appropriation of $5 million for pediatric cancer research. Subsequently, the New Jersey Commission on Cancer Research established a Pediatric Cancer Research Advisory Group (Advisory Group) to consult with the NJCCR on how the money from the fund should be utilized to support pediatric cancer research projects to qualified research institutions in New Jersey.

The Advisory Group held its first meeting in April 2022. This group consists of seven members who either treat patients with pediatric cancer, conduct research into pediatric cancer, advocate to advance pediatric cancer research or treatment, or have been affected by their own or a family member's diagnosis of pediatric cancer. Since this time, NJCCR continues to stay engaged with the Advisory Group to assist with the identification and funding of scientific research projects that focus on translational research (from laboratory to the bedside) and pediatric cancer research projects surrounding the causes, prevention, education, treatment, or cure of pediatric cancer, or the symptoms or effects experienced by patients following completion of a course of treatment for pediatric cancer. Currently, the Advisory Group Members include the following:

- Aubrey Reichard-Eline, Chair
- Dr. Peter Cole, Co-Chair
- Dr. Daniel Notterman
- Paulette Forbes
- Dr. Rafat Ahmed and Dr. Alfred Gillio

Research continues to be our best defense against cancer because it is the driving force behind all clinical and policy advances that can improve cancer prevention, detection, diagnosis, treatment and, increasingly cures for cancer.

2024 Grant Recipients

Predoctoral Research Fellowship Recipients:

Category: Other (Anal cancer)

Project Title:

Preparing for implementation: a mixed methods study for anal cancer screening among people living with HIV in New Jersey. COCR24PPR004

Racquel Kelly Kohler, PhD, MSPH, kelly.kohler@rutgers.edu

Affiliation: *Rutgers Biomedical and Health Sciences*

The number of new cases of anal cancer and deaths from the disease have been increasing in the United States, especially among people living with HIV. All people living with HIV have a substantially increased risk, but men who have sex with men and live with HIV have the highest risk of anal cancer. Black and Hispanic men and women living with HIV are also more likely to be diagnosed with anal cancer. Infection with anal human papillomavirus (HPV) is common among high-risk groups and can progress to precancerous lesions, which cause anal cancer. A historic randomized trial, which included people recruited from Rutgers New Jersey Medical School in Newark, recently found that diagnosing and treating precancerous lesions can reduce the risk of anal cancer by half. Public health agencies and experts are reviewing the new evidence to make much needed anal cancer screening recommendations. We want to determine how the screening and treatment approach, which is similar to cervical cancer screening, should be integrated in routine care for people living with HIV. However, some practical and logistical issues must be explored and addressed before implementing a new anal cancer prevention program. For example, diagnosing precancerous lesions is a key step in the screening process, but there are very few health care providers with this expertise. We will assess the local health system capacity to provide anal cancer screening, diagnosis, and treatment services and create a plan to make them more widely available. The goal of this study is to develop a highly acceptable anal cancer screening and treatment program with a toolkit specifically designed to support implementation in the Greater Newark Area. We will obtain input from patients and advocates to ensure the program is designed to meet the community's needs and preferences. We will work with experts, health care providers, and hospital leaders to prioritize feasible strategies that will support the program's successful implementation. The research will take place in Essex and Hudson counties, which have some of the highest HIV rates across the state. This study is an important next step to reduce cancer disparities and improve anal cancer prevention for those with the greatest risk of disease.

Category: Pancreatic

Project Title:

Evaluation of the Tumor-Microbiome-Immune Interactions in Malignant Progression of Pancreatic Cystic Neoplasms. COCR24PPR006

Brett Ecker, MD, Brett.Ecker@rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

Pancreatic ductal adenocarcinoma (PDAC) is the third leading cause of cancer-related deaths in the United States, where a lack of effective primary and secondary prevention strategies contributes to frequent clinical presentation in the incurable, metastatic state. Early detection and effective characterization of premalignant lesions can reduce mortality. Intraductal papillary mucinous neoplasms (IPMNs) are the most common type of pancreatic cyst and one of the only known precursors to PDAC. To date, the key determinants of IPMN progression from low-grade cyst to high-grade cyst to PDAC are not well understood. Furthermore, nonwhite patients with IPMN are significantly more likely to have invasive IPMN at surgical resection, mirroring overall patterns of increased incidence and later detection of PDAC amongst nonwhite patients. To date, genetic driver mutations have offered limited clues to observed racial disparities in IPMN progression. We hypothesize that aspects of the tumor microenvironment may hold important clues. While normal pancreas is typically sterile, emerging evidence from others and us indicates that microbes colonize the pancreatic tumor microenvironment, influencing key cancer-associated pathways and also tumor-immune interactions. We hypothesize that racial differences in the pancreatic microbiome exist and that such differences may underlie some of the disparities in IPMN-related outcomes – which we propose to examine in this project. This project aims to evaluate tumor-microbiome-immune crosstalk during the stages of malignant progression from IPMN to PDAC in a racially diverse clinical cohort. We aim to: (1) characterize the burden and diversity of IPMN-associated microbes at different stages of dysplastic progression and (2) evaluate microbial-associated transcriptional changes in pancreatic cystic epithelium and IPMN-associated immune infiltrates. We have already received Scientific Review Board approval and have coordinated with the biorepository to obtain necessary archival samples. Utilizing emerging spatial transcriptomics and sophisticated computational methods developed in our lab, we will systematically recover and denoise microbial signals in human IPMN specimens to assess host-microbiome-immune interactions at near-single-cell resolution. The findings developed from this

NJCCR grant programs are designed to provide scientific opportunities to attract research scientists. Awards are intended to promote collaboration among cancer researchers in New Jersey as well as spark innovative research.

2024 Grant Recipients

proposal may (1) identify clinically applicable bacterial biomarkers to guide patient surveillance strategies, and (2) provide a novel data-driven, genomic approach for further hypothesis-generating and corroborative studies on causal pathways of microbiome and immune interactions during IPMN dysplastic progression.

Postdoctoral Research Fellowship Recipients:

Category: Liver

Project Title:

Modeling chronic hepatitis B virus infection and virally-induced hepatocarcinogenesis in a small non-human primate model. COCR24PDF002

Yongzhen Liu, PhD, yongzhen@princeton.edu
Affiliation: *The Trustees of Princeton University*

Liver cancer is one of the most common causes of cancer-related deaths. 80% of all liver cancers are a result of infections with hepatitis B virus (HBV). Worldwide about 2 billion people have become exposed to HBV, resulting in over 257 million infections that persist in the body of the infected individual. This persistent, or chronic hepatitis B frequently causes severe liver diseases to which close to 900,000 people succumb every year. Unfortunately, this number is estimated to increase significantly over the next two decades. HBV infection can be prevented with a prophylactic vaccine; antiviral therapy can suppress viremia but rarely leads to a cure, i.e. a complete elimination or permanent inactivation of the virus in the body. Thus, current antiviral therapy only decreases, but does not eliminate, the risk of liver cancer development. A major roadblock for developing more effective therapies and for gaining mechanistic insights into how HBV causes liver cancer is the scarcity of suitable animal models. Naturally, HBV can only infect humans and chimpanzees. The reason why HBV cannot infect other monkey species or rodents - both of which are frequently used in biomedical research - is not understood. HBV hijacks a bile acid transporter called NTCP to enter liver cells. The building blocks and consequently the overall structure of NTCP differ between humans and species that cannot be infected with HBV. We demonstrate that these differences create a barrier for the virus that precludes infection. Notably, when we engineered monkey liver cells to express human NTCP HBV can complete its entire life cycle. This finding demonstrates that viral uptake is the only major barrier that would have to be overcome to establish infection in a small non-human primate model for HBV. We reasoned that it would be easier to adapt the

virus to a new host rather than creating a genetically engineered monkey. To adapt HBV, we analyzed whether a virus related to HBV that has been previously identified in wooly monkeys (thus called WMHBV), an endangered species, could possibly infect liver cells from other primates. Interestingly, WMHBV can indeed infect liver cells isolated from marmosets, a small non-human primate species that is commonly used in biomedical research. We went one step further and constructed a chimeric virus that combines a very small piece of WMHBV responsible for mediating viral uptake while keeping 98.5% of the HBV genome intact. This minimally monkey-adapted HBV variant can also infect marmoset cells and has putatively much greater utility for evaluating treatments intended for humans. We now propose to test whether this approach can be more generally applied to other HBV strains. We will further attempt to increase the robustness of this new "chimeric" virus using state-of-the-art cell culture and animal models pioneered in our lab. Collectively, our work will not only answer why HBV does not infect a more diverse set of animals but also paves the path toward an urgently needed animal model for antiviral therapeutic development, studying in vivo HBV infection, immunopathogenesis and HBV-related liver cancer.

Category: Colon and Rectum

Project Title:

Targeting intestinal epithelium-immune cell crosstalk in colorectal cancer. COCR24PDF006

Xue Yang, PhD, xy263@cinj.rutgers.edu

Affiliation: *Rutgers Biomedical and Health Sciences*

Colon cancer is a deadly disease and one of the leading cause of cancer death worldwide. In 2022, approximately 151,000 cases of colon cancer were diagnosed, and 52,000 patients died of the disease in the United States. With the early detection, surgical resection, chemotherapy, there is an increase of the survival rate of colon cancer patients. However, there are still many colon cancer patients, especially those who are diagnosed at advanced stages, develop recurrent disease or metastasis with a poor survival rate. Therefore, there is an urgent need to gain a better understanding of the underlying mechanisms contributing to the progression of colon cancer to help develop novel and effective therapies for colon cancer. Currently, the potential molecular mechanisms involved in colon cancer development and progression are still far from clear. Previous work from our lab has suggested that LIF (leukemia inhibitory factor), a multi-functional cytokine, is often overexpressed in colon cancers, and its overexpression is associated with poor prognosis of colon cancer patients.

Research is critical to reducing the burden of cancer in New Jersey. This 2022-2023 annual report highlights the remarkable progress that has been made against cancer by scientists in New Jersey.

Further, LIF promotes the growth of colon cancer cells and renders them resistance to chemotherapeutic drugs. These data suggested that LIF may play an important role in colon cancer development and progression. However, the precise role of LIF in colon cancer and its underlying mechanism are not well-understood.

The anti-tumor immune response plays crucial roles in limiting tumor development and progression. Tumors often evade immune recognition and thus escape immune surveillance. However, its underlying mechanisms are not well-understood. Results from my preliminary study suggest that LIF inhibits anti-tumor immune response which could be an important mechanism by which LIF promotes colon cancer development and progression. In this proposed study, I will test the effect of LIF on anti-tumor immune response and identify its underlying mechanisms using genetically engineered colon cancer mouse models with different LIF expression levels in intestinal tissues. I will further test if targeting LIF can activate anti-tumor immune response and inhibit the development and progression of colon cancer. The goal of this study is to determine the role of LIF in colon cancer and immune function to provide effective therapeutic targets/strategies for colon cancer. This work is highly cancer relevant and is expected to have a great potential to develop new and effective treatment strategies for colon cancer.

Category: Colon and Rectum

Project Title:

The mechanism of SUMO-Specific Protease 6 in Cancer. COCR24PDF007

Leilei Gou, PhD, lg942@rutgers.edu

Affiliation: *Rutgers Biomedical and Health Sciences*

Colorectal cancer is the third most commonly diagnosed cancer and the third leading cancer death in the United States. Better understanding the molecular mechanism of CRC will provide novel targets and strategies for colorectal cancer therapies. Metabolic reprogramming is a hallmark of cancers. Cancer cells frequently reprogram their metabolism to meet their needs for rapid growth and proliferation. SUMO-Specific Protease 6 (SENP6) is a protein involved in a special type of protein modifications in cells named SUMOylation, which plays an important role in regulating many important cellular processes. Emerging evidence has suggested a potential role of SENP6 in cancer. However, the role and mechanism of SENP6 in cancer, including colorectal cancer, are poorly defined.

Our preliminary studies suggest that SENP6 plays a critical role in colorectal cancer and regulating metabolic reprogramming is a critical mechanism by which SENP6 promotes colorectal cancer progression. Based on our preliminary studies, we hypothesize that SENP6 plays a critical role in promoting colorectal tumorigenesis through metabolic reprogramming. In this proposed study, we will investigate the underlying mechanism by which SENP6 promotes colorectal cancer (Aim 1), and will test novel strategies to treat colorectal cancer with SENP6 overexpression (Aim 2).

The goal of this study is to determine the mechanism of SENP6 in colorectal cancer, and test novel therapeutic strategies for colorectal cancer, especially colorectal cancer with SENP6 overexpression. We anticipate that this proposed study will deepen our understanding of the mechanism of colorectal cancer, and have potential to provide novel and effective therapeutic strategies for colorectal cancer, which is urgently needed in clinical cancer therapies.

Category: Breast

Project Title:

Integrated analysis of imbalanced allelic expression to infer gene regulatory patterns in cancer. COCR24PDF008

Mona Arabzadeh, PhD, ma1748@cinj.rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

When two alleles of a gene in a cell are expressed at different levels, allelic imbalance (AI) phenomenon has happened. This phenomenon may occur as a consequence of a genetic variation/rearrangement in regulatory regions, copy-number variation, or epigenetic inactivation of one of the two alleles. Allelic imbalance can be the result of normal physiology (i.e., imprinting, X-chromosome inactivation) and/or cancer signature. Gene regulatory patterns are employed as a causality network-interface to reveal the reason of tumor growth. Using AI as an expression signal, we need to justify a methodology to integrate data to find the underlying mechanism of regulatory patterns to find cancer drivers as of genomic alternations (i.e., mutations in promoter regions or rearrangements). To test this hypothesis, we will develop a quantitative framework for accurately measuring AI, and then integrating DNA, RNA, and methylation data to investigate AIs' underlying mechanisms. We will apply this pipeline to tumor bulk data to assess the consequences of AI on regulatory networks specifically as cancer drivers and to single-cell data to investigate the spatial transcriptomic profiles of co-regulated genes. We aim to discover regulatory pathways in cancer that reveal allelic imbalance as part of their function.

In 2024 and 2025, the NJCCR awarded predoctoral, postdoctoral, bridge, and pilot grants ranging from $100,000 to $200,000.

2024 Grant Recipients

Category: Hematologic

Project Title:

Development of a novel single-cell transcriptomics analysis toolkit. COCR24PDF015

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Affiliation: *Rutgers Biomedical and Health Sciences*

Single cell gene expression studies are rapidly transforming our understanding of cellular processes at the level of single cells, especially in the case of complex diseases such as cancer. In all these studies, the computational analysis of the data plays a vital role. Here, we propose to develop a completely novel single cell gene expression analysis toolkit that will enable more meaningful and impactful biological inferences to be drawn from single cell gene expression studies. We will use this toolkit to examine data sets obtained from a study that will explore how blood cancer cells adapt to different environments within the body and how they may be reshaping these environments to avoid detection and response from the immune cells.

Category: Other (Epithelial carcinomas; solid tumors)

Project Title:

Dissecting the role of Disheveled in the spatial organization of Frizzled during epithelial planar cell polarity establishment. COCR24PDF018

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Affiliation: *Princeton University*

The incredible diversity of life-forms in nature and the complexity of shapes and structure of their body plan, arise due to tight coordination in the behavior of thousands of cells that make up the underlying tissue and organs, as the organism develops from a single cell to embryos and finally to adults. As one would appreciate from the fast-paced videos of developing embryos, there are visibly large tissue-scale movements, which are mediated by none other than the collective motion of cells in the embryonic tissue. Fruitful and directional

movements can only arise by co-operative action between each constituent cell of the tissue. Thus, to contribute productively to the global movement, each cell must have a local sense of direction. This is very similar, for example, to how every individual must be aligned to a line of thought for any philosophical or political movement to succeed. Such a local sense of direction comes from cellular compasses, set by unique groups of proteins that exclusively populate opposing sides of the cell. In other words, the sense of direction is achieved by positioning unique sets of proteins on anterior (towards the developing head) versus the posterior (towards the developing tail). When such directional signposts are set up by thousands of cells across the plane of a tissue, it gives rise to planar cell polarity and the proteins that are involved can be called planar cell polarity proteins. Losing sense of direction can result in aberrant tissue movements, leading to a wide range of birth defects. This project is focused on studying how such a cellular compass gets established so that the cells can decode positional information and the overall orientation of the developing organism.

To move effectively, groups of cancer cells use the same set of planar cell polarity proteins as needed for proper embryonic development. These proteins can specify which direction to move, and the cells can more effectively disseminate to healthy tissues without spending unnecessary time in deciding the course of their movement. Moreover, the polarity proteins called Disheveled can be used by cancer cells to multiply in an uncontrolled manner. It is therefore critical to check the activity of these proteins so that they cannot be misused by malignant cancer cells. Like humans, proteins are highly interactive entities and depending on where and when it interacts with whom, they can determine the behavior of cells. The polarity proteins in the embryonic skin behave as dormant directional signposts without triggering excessive proliferation or aggressive migratory behavior in these cells. Learning what keeps them dormant in this tissue can help check their role in aggressive behavior of malignant cancer cells. Therefore, in this project I plan to study how polarity proteins come together and accumulate at the cell-cell boundaries in the healthy skin tissue and how they create protein assemblies of unique composition at the anterior versus the posterior borders, to set up their cellular compasses.

Research is the backbone of scientific progress against cancer. It spurs the development of innovation, medical breakthroughs, and better approaches to preventing, detecting, diagnosing, treating, and curing some of the many diseases known as cancer.

2024 Grant Recipients

Category: Other (Drug screening and assay development)

Project Title:

Synthetic discovery of isoform specific TET inhibitors by a novel high-throughput screening assay. COCR24PDF019

Mikel Ghelfi, mghelfi@coriell.org
Affiliation: *Coriell Institute for Medical Research*

Every living cell contains DNA, which harbor the information to create and change every protein structure in our body. To regulate which part of the DNA is used to make proteins, it is tagged by a convertible marker. In cancer these markers are changed in a way to ensure cancer cell rapid growth and longevity. Removal of DNA markers hinders cell growth in some cancers. There are three similar looking proteins performing the task of marker removal called TET. Depending on the cancer type, inhibition of all three TETs has beneficial or detrimental effects on cancers progression. Precise inhibition of each individual TET is crucial to stop growth in particular cancers, but so far, no specific inhibitor drug has been developed. Our goal is to develop a drug for each TET and use them for cancer therapy.

Predoctoral Research Fellowship Recipients:

Category: Breast

Project Title:

Gut microbiome impact on tumor progression and immunotherapy response. COCR24PRF003

Salma Youssef, sy4767@princeton.edu
Affiliation: *Princeton University*

The treatment of melanoma and other cancers has been revolutionized by the discovery of immune checkpoint blockade, for which the 2018 Nobel Prize in Physiology was awarded. The use of monoclonal antibodies against the programmed cell death protein 1 (PD-1) has been a breakthrough therapy that activates the patient's own immune cells to fight off the cancer. However, tumor response to immune checkpoint blockade is varied which limits its widespread application. We can classify patients as responders or non-responders to therapy based on their clinical outcomes following anti-PD-1 treatment. In melanoma patients, the gut microbiome composition plays a significant role in patient response to therapy. Furthermore, recent exciting

studies showed success improving patient response to immune checkpoint blockade therapy by administering fecal microbiota transplants (FMT) as an adjuvant therapy. Despite the success of these studies in establishing the role of the microbiome in modulating the immune response, we have yet to discover the molecular mechanisms of how bacteria in the gut can modulate tumor response to anti-PD1 therapy. What is the gut microbiome producing that alters the metabolic and immune environment of the host?

Metagenomic sequencing has enabled us to identify key microbiome constituents and active pathways by analyzing the biosynthetic gene clusters upregulated in fecal samples from melanoma patients undergoing immune checkpoint blockade treatment. Implanting germ-free mice with donor microbiomes that express these signatures will allow us to closely inspect how it affects tumor progression along with immune metabolic profiling of the host and tumor. Given our expertise in studying tumor-immune crosstalk using our co-culturing system along with the ex-vivo cultures of donor microbiomes methodology we developed, we can further pinpoint the mechanism of action behind improved response to anti-PD-1 therapy.

Taken altogether, this project will determine the mechanisms by which the gut microbiome modulates tumor progression and response to cancer immunotherapy and identify specific bacterial species and small molecules that can be used to improve the clinical outcomes of immune checkpoint blockade therapy.

NJCCR membership consists of 11 volunteer members appointed by the Governor with the advice and consent of the Senate.

Category: Liver

Project Title:

Evaluation of combination mTOR agonist and CD147-IL15-CAR-NK cell therapy in transgenic human CD147 Hepatocellular Carcinoma models. COCR24PRF004

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Affiliation: *Rutgers Biomedical and Health Sciences*

Liver cancer is the second most common cause of cancer-related death worldwide. Liver cancer is caused by hepatitis B virus (HBV) and/or HCV infections; though, high fat diets and excessive alcohol consumption increases the risk of liver cancer developing. The worldwide burden of liver cancer is projected to be over 1 million cases by 2030. Liver cancer ranks fifth in terms of global cases and second in terms of death for men. Approximately 80% of liver cancer patients die within 12 months post-diagnosis due to limited effective treatments. It is becoming more evident that one's immune system is capable of naturally eradicating tumor cells. However, cancer cells develop different methods to evade the immune system, resulting in uncontrolled tumor growth. Chimeric

antigen receptors (CARs) are a new promising approach used in cancer therapy where either the patient's or the donor's blood is used for genetic modification of the immune cells. The engineered cells are grown in the lab and infused into a patient in order to target and kill cancer cells, but not healthy cells. The biomarker which our lab focuses on is called CD147, also known as Basigin (BSG) or extracellular matrix metalloproteinase inducer (EMMPRIN). It is shown to be abundantly present in liver cancer tissues but not healthy tissues. In leveraging this genetic modification technology, our lab has successfully engineered CD147-CAR-natural killer (NK) cells derived from healthy donors' blood to specifically target liver cancer cells. We have shown that CD147-CAR-NK cells significantly control liver cancer progression in immunodeficient mice when the tumor cells are engrafted under the skin of these animals (or subcutaneous engraftment). Though the results are promising, this model does not truthfully represent the development of liver cancer. The cancer cells in a complex organ such as the liver are known to acquire various mechanisms to escape the immune-surveillance of CD147-CAR-NK cells. Since our last discovery, we have been: 1) equipping CD147-CAR-NK cells to functionally excel in the suppressive tumor microenvironment (TME) and 2) establishing a more physiologically and clinically relevant liver cancer model in immunodeficient mice carrying a human CD147 gene. The proposed study will thoroughly evaluate the therapeutic efficacy of the improved CD147-CAR-NK cells in a more recapitulative hostile liver cancer TME. Completion of the proposed project will accelerate the bench-to-bedside of CD147-CAR-NK cells in treating liver cancer patients.

Category: Hematologic

Project Title:

Investigating genetic susceptibility for chemotherapy-induced cognitive impairment in a juvenile ApoE4 rat model. COCR24PRF009

Chadni Patel, ckp39@gsbs.rutgers.edu

Affiliation: *Rutgers Biomedical and Health Sciences*

The number of pediatric cancer cases is expected to significantly increase in the coming years. While survival rates have improved significantly with advances in treatment, many survivors experience a treatment-induced neurocognitive deterioration, described as "chemobrain", leading to inferior quality of life. Despite extensive research into the multifactorial causes of chemobrain, there are no FDA approved drugs to prevent or reduce its severity, and the mechanism is unclear. We propose to investigate in a juvenile model to mimic the pediatric population since we predict developing

brain is more susceptible to damage when compared to an adult brain. ApoE4 has recently been linked as a genetic predisposition to chemobrain. Not all patients experience chemobrain, therefore, discovery of potential biomarkers for identifying patients who are more prone to developing it prior to their treatment is crucial. These studies will lead to a long-term impact to improve the quality of care/life of not only pediatric patients, but also adult patients, as we are using a broadly used chemotherapeutic agent, doxorubicin. These studies will not only begin to point to key biomarkers to predict patients who will be more susceptible and how to intervene, but it will also allow us to begin to unravel the mechanism for chemobrain. In this study, we will assess if memantine, a drug for dementia that binds to the NMDA receptor and prevents synaptic plasticity changes because of the inhibition of calcium ions, will rescue the effects of chemobrain. Memantine poses as a promising candidate for a preventative measure as it is FDA-approved and previous research indicates that memantine can prevent chemotherapy-related cognitive deficits in a juvenile rat model.

Category: Hematologic

Project Title:

Dissecting OTUD5 as a novel therapeutic target in leukemia. COCR24PRF011

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Affiliation: *Rutgers Biomedical and Health Sciences*

T-ALL is a hematological malignancy that predominantly affects children but can also occur in adults. Cure rates have increased due to recent advances; however, 20-50% of patients still relapse, and therapeutic options are scarce at that point, leading to high mortality rates. Thus, we need to discover new targets for the treatment of T-ALL. Related to this, innovative genetic experiments in vitro have suggested that eliminating a specific gene/protein (OTUD5) might broadly synergize with currently used antileukemic drugs in the clinic. Of note, our preliminary results support this very idea, since using genetic tricks that allow us to eliminate OTUD5 from leukemia cells resulted in significant antileukemic effects on its own, which drastically synergized in combination with Daunorubicin, one of the mainstay chemotherapeutic drugs in T-ALL treatment. However, nothing is known regarding the role of OTUD5 in leukemia. Thus, here we will use cutting-edge techniques in combination with unique novel mouse models to study the specific role and mechanisms of OTUD5 in T-ALL. Overall, our results might translate into better treatments for leukemia patients in the short term.

This year, NJCCR announced a new research grant opportunity on the impact of cancer and health disparities. We are confident that this new opportunity will provide insight into the scientific reasons for these glaring disparities in New Jersey.

2024 Grant Recipients

Category: **Other** (Cancers that are amenable to photodynamic therapy)

Project Title:

Leveraging production of reactive oxygen species in photodynamic therapy for the development of a novel drug targeting strategy. COCR24PRF012

Weronika Wasniowska, wkw11@gsbs.rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

The broad long-term goal of this research is to improve cancer therapies by targeting drug delivery to the tumor site and thereby limit the dose of drug and off-target effects. Photodynamic therapy is a current form of cancer treatment that involves the delivery of molecules called photosensitizers that preferentially accumulate in tumors. When the photosensitizers are exposed to specific wavelengths of red light, it causes a reaction that leads to the production of an excess of free radicals called reactive oxygen species (ROS). These free radicals lead to the death of cancer cells, though PDT is often followed by rounds of chemotherapy or other treatments. Our goal is to combine PDT with a targeted drug delivery system that we are developing that leverages the elevated presence of free radicals. Our system comprises polymers that react with the free radicals to create a polymer net at the site of free radicals production. This net is functionalized to specifically catch a drug or other payload, which can be released from the net by enzymes produced by tumor cells. We had planned to rely on tumor cells to over-produce free radicals. However, with PDT, we can specifically introduce radicals and drive net formation and subsequent drug targeting. In this proposal, we establish compatibility of PDT with our catch-and-release system. Our first aim is to characterize the production of free radicals from different photosensitizers in agar gel models made to mimic tissue. Our second aim is to test these photosensitizers in 3D cell models called spheroids made up of cancer cells followed by analyzing the delivery of our catch-and-release polymer and payload. We will be testing the effects of these on lung cancer, breast cancer, and basal cell carcinoma cells as these are three types of cancer where photodynamic therapy is used and drug resistance occurs. Once complete, we will be prepared to move to pre-clinical animal studies.

Category: Colon & Rectum

Project Title:

CD74 Receptor Activated Paneth Cells Modulate Intestinal Inflammation and Cancer Progression. COCR24PRF017

Jared Bianchi-Smak, jfb114@newark.rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

Colorectal cancer is one of the most commonly diagnosed cancers in the United States contributing to 150,000 new cases and 52,000 deaths annually. Inflammation-associated colon cancer is a subtype of colorectal cancer with a very poor prognosis and represents one of the most severe complications resulting from chronic intestinal inflammation. Patients having chronic intestinal inflammation conditions are at a significantly higher risk of developing colorectal cancer if left untreated or the treatment plans fail. Current literature suggests that the pathogenesis of colorectal cancer is associated with genetic, dietary, and environmental risk factors. However, the pathways linking inflammation to colon cancer progression remain incompletely understood at cellular and molecular levels. This project tests a newly discovered immune signaling pathway that could shed light on the missing link from the damaged intestinal epithelial cell lining to the activation and aggregation of inflammatory immune cells at the site of pathology. Our preliminary data suggest that a specialized intestinal epithelial cell type, named the Paneth cell, is activated through a surface receptor by a specific cytokine produced by stimulated white blood cells. These activated Paneth cells may propagate the local inflammation through attracting more inflammatory immune cells to the site of damage to drive the disease progression. This process, if constitutively and continuously stimulated, may ultimately lead to intestinal tumor formation. I will test above pathway in two specific aims. I will first use microscopic imaging and flow cytometry analysis to map out these receptor-expressing Paneth cells and determine if their activity can be dampened when I delete this surface receptor. I will then use mouse inflammation-associated colon cancer models to determine if by blocking this molecular communication between Paneth cells and inflammatory immune cells, I can reduce the formation or aggressiveness of the colon cancer. This project is significant because it studies an important type of colon cancer with limited treatment strategy. This project is innovative because it studies a new pathway that may mediate the missing link between two types of cells in the inflamed intestine.

Our grantees have leveraged a return of over $10 in federal research funding for every NJCCR dollar awarded for a total of $455 million. These grants have been awarded to the Cancer Institute of New Jersey, Rutgers University both in Newark and Piscataway, Princeton University, Rowan University, and many others.

2024 Grant Recipients

Category: Other (Non-specific)

Project Title:

Elucidating the genetic drivers of immune activation by dendritic cells as novel targets for cancer immunotherapy. COCR24PRF021

Ryan McNulty, ryan.mcnulty@princeton.edu

Affiliation: *The Trustees of Princeton University*

The human immune system is a complex network of proteins, cells, and tissues that work in a coordinated manner to protect the body against harmful pathogens such as cancerous cells. Over the past few decades, researchers have made significant progress in understanding the mechanism that govern immune system function, leading to the development of a new class of cancer treatments known as immunotherapies which harness the body's own immune system to fight cancer. One promising class of immunotherapies is immune checkpoint inhibitors (ICIs) – drugs which block proteins on the surface of immune cells that normally inhibit immune response. While these checkpoint proteins exist to prevent the body's immune system from overreacting to a particular event and damaging healthy tissue, cancer cells often hijack this mechanism to downregulate the body's immune response and evade detection. ICIs thus act to release the "brake" applied on the immune system and promote a strong immune response by allowing immune cells to better recognize and target cancer cells.

The emergence and clinical success of immune checkpoint inhibitors in the past decade represents a breakthrough in our ability to fight cancer by tuning the biological signals that control the immune system. In this study, we aim to identify additional proteins that regulate the immune response, either by the relay of inhibitory or stimulatory signals, as potential targets for immunotherapies. To achieve this, we focused our work on dendritic cells, a type of immune cell which plays a specialized role in activation of the immune system. Specifically, DCs recognize foreign agents, such as those produced by cancer cells, in the tissue environment and respond by becoming potent activators of downstream effector cells (such as T cells) which then target and kill the cancer cells. Communication between DCs and T cells is achieved through proteins on the surface of the two cell types that propagate inhibitory or stimulatory signals upon interacting. Therefore, we considered DCs a ripe source of novel immunotherapeutic targets.

In our study, we obtain DC precursors from human donors and block the expression of individual proteins expressed by these cells through modern genetic engineering techniques. We then screen the edited cells for their ability to perform critical immune functions such as activate T cells or migrate upon recognition of antigen. If a protein expressed by DCs is important in mediating immune response, we observe a differential response by the perturbed cells in our screens. With this approach, we can systematically identify proteins that regulate the human immune system and thus are strong candidates for novel cancer immunotherapies. Drugs such as monoclonal antibodies can then be designed to target these regulatory proteins, thus modifying cell behavior and adding to an expanding toolbox of treatments that tune the immune system's response to cancer.

Pediatric Research Grant Recipients:

Category: Pediatric (Hematologic)

Project Title:

Identification of biomarkers for chemobrain in pediatric ALL patients. COCR24PRG001

Mi Hyeon Jang, PhD, mihyeon.jang@rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

While current pediatric cancer treatment approaches are effective, chemotherapy-induced cognitive impairment (CICI or chemobrain) has emerged as a medical problem with a significant negative impact on quality of life in cancer survivors. Hence, we aim to identify biomarkers that can predict and/or mediate susceptibility to cognitive decline, in conjunction to novel therapeutic interventions to treat CICI. In our prior study, we demonstrated that CICI is causally associated with depletion of nicotinamide adenine dinucleotide (NAD+), a key metabolite in energy metabolism that is linked to aging and age-related neurodegeneration. Hence, this study aims to determine if NAD+ loss can be novel prognostic/diagnostic indicators that can predict/mediate CICI using cerebrospinal fluid (CSF) collected longitudinally from over 3000 children treated with acute lymphoblastic leukemia (ALL) being treated on one of two multi-institutional cooperative group trials. In addition, our preliminary studies also demonstrate that methotrexate, a chemotherapeutic drug commonly used for treating ALL, increases PARP1 (the main NAD+ consuming enzyme) levels, resulting in NAD+ loss in the adult mouse hippocampus in vivo. This

The Commission has an amazing group of dedicated volunteers who serve on NJCCR. The commission members represent academia, non-profits, and health care institutions.

led us to hypothesize that hyperactivation of PARP1 by methotrexate causes NAD+ loss to exacerbate neuronal and cognitive dysfunction. Therefore, we propose to test the hypothesis that PARP1 inhibition (through administration of brain permeable ABT-888; veliparib) could sustain NAD+ levels against methotrexate-induced cellular damage and consequently prevent CICI in non-tumor and primary T-ALL mouse models.

Category: Pediatric (Hematologic)

Project Title:

Targeting Sprouty Signaling for Overcoming FLT3i Resistance in Pediatric AML. COCR24PRG005

Jian Huang, MD, PhD, jhuang@coriell.org
Affiliation: *Coriell Institute for Medical Research*

Pediatric Acute myeloid leukemia (AML) is a malignant hematopoietic disease. It is the second most common childhood leukemia and is associated with high rates of chemotherapy resistance and relapse. One major obstacle to greater success with target therapy of pediatric AML is drug resistance. The mechanisms underlying drug resistance in pediatric AML are poorly understood. FLT3 is a cytokine receptor that belongs to the receptor tyrosine kinase (RTK) class III. Activating mutations in FMS-like tyrosine kinase 3 (FLT3) are now recognized as the most common molecular abnormality in AML and FLT3ITD mutations are found in nearly 30% of AML patients. Both Quizartinib (AC220) and gilteritinib are potent and selective second-generation FLT3 inhibitors (FLT3is). AC220 is in clinical trials for the treatment of relapsed or refractory FLT3ITD positive and negative AML patients and as maintenance therapy. Gilteritinib was already approved by the FDA in 2018 to treat patients with relapsed or refractory AML with an FLT3 mutation. However, drug resistance to AC220 and gilteritinib has also been reported through early clinical studies and clinical treatments, respectively. To understand the underlying mechanisms of drug resistance to FLT3i, we undertook an unbiased approach with a novel CRISPR pooled library to screen new genes whose loss of function confers resistance to AC220. In our screen, we identified SPRY3, an intracellular inhibitor of RTK signaling, and demonstrated the re-activation of downstream RTK/Ras/ERK signaling as a major mechanism of resistance to the FLT3i. Furthermore, we also confirmed

our findings in primary AML patient samples. We demonstrated that the expression level of SPRY3 is dramatically reduced in AC220 resistant AML samples and SPRY3 deleted primary AML cells are resistant to AC220. Additionally, we treated SPRY3 knockout AML cells with a potent MAP kinase inhibitor demonstrated that it re-sensitized AML cells to AC220. Importantly, we found that at least two SPRY mutants (SPRY1 and SPRY3 KOs) pediatric AML were resistant to gilteritinib in culture. In this proposal, we hypothesize that Sprouty (SPRY) play critical roles in the response to FLT3i gilteritinib in pediatric AML. The Ras/MEK/ERK pathway regulated by SPRY3 is important for the acquired FLT3i resistance in pediatric AML. Next, we will perform a series of comprehensive studies to explore novel downstream effectors/ interacting partners of SPRY3 in pediatric AML and the molecular mechanisms of their action. Furthermore, we will examine the possibility of translating our findings into new clinical therapies.

Category: **Pediatric** (Breast, Prostate, Brain and Other Nervous System, Melanoma of the skin)

Project Title:

The role of Nodal signaling in promoting partial EMT migration. COCR24PRG007

Rebecca Burdine, rburdine@princeton.edu
Affiliation: *The Trustees of Princeton University*

Metastasis is the end stage of cancer progression and the primary cause of death. Thus, therapeutic interventions to prevent progression of cancer cells to a metastatic state are critical for improving the survivability of cancer patients. To become metastatic, cancer cells acquire the ability to migrate. While we once thought migratory cells lost all contact with other cells and moved as single cells, we now appreciate cancer cells can migrate without losing contact with other cancer cells. Instead, they stay connected to one another and migrate as a collective group, a state that is referred to as a partial epithelial to mesenchymal transition or pEMT. As part of becoming migratory, these cells create small structures called podosomes, or invadosomes, that degrade material they encounter to clear the way for cells to migrate. We are studying a pEMT cell migration that occurs during heart development in the developing zebrafish embryo. We

Aside from the annual $1 million to $4 million State budget appropriation, NJCCR receives funding from other sources, such as State income-tax checkoffs and the purchase of a Conquer Cancer License Plate by New Jersey residents.

have shown that this migration is driven by two cell signaling pathways, Nodal and RAS/MAPK/ERK, both of which are misregulated in metastatic and pediatric cancers. Furthermore, we found that the migrating heart cells make podosomes, similar to those observed in cancer cells. We will determine how Nodal and RAS/MAPK/ERK signals promote pEMT migration and podosome formation during heart cell migration in zebrafish. By better understanding the nature and process of pEMT in a normal, developmental migration, we can in turn better understand how pEMT is co-opted by cancer cells to become metastatic. Furthermore, this study will provide information needed to identify components to target to prevent this type of cell migration in cancer patients.

Category: **Pediatric** (Hematologic)

Project Title:

Poverty, Neuroinflammation, and Symptoms in Childhood ALL. COCR24PRG008

Beth Savage, PhD, CPNP, CPON, savagebe@sn.rutgers.edu
Affiliation: *Rutgers Biomedical and Health Sciences*

Great strides have been made in the treatment of childhood leukemia, but most children can expect to develop side effects of chemotherapy. These side effects can be quite distressing and for some children, may last long after treatment has ended. A cluster of side effects that occur together in both children and adults receiving chemotherapy consists of fatigue, disturbed sleep, pain, mood changes, and distracted thoughts. Exposure to stress can cause inflammation which is likely a means by which these symptoms are linked. Research conducted in adults with cancer has demonstrated that stress does cause unexpected inflammation and that those experiencing stress early in life develop worse symptoms following exposure to chemotherapy. Based on this body of research we seek to determine if these same links between stress, in the household and the neighborhood, increased inflammation, and subsequently, worse symptoms exist in a group of children undergoing leukemia treatment. If successful, this study will lay the groundwork for future research to develop targets in which both stress and the inflammation it leads to can be minimized, thus decreasing the burden of treatment for children with leukemia.

Members of the New Jersey Commission on Cancer Research

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The Commission's leadership and staff provide vital linkages to implement its programs and ensure day-to-day operations.

Highlights from the 2024 Cancer Commission Symposium

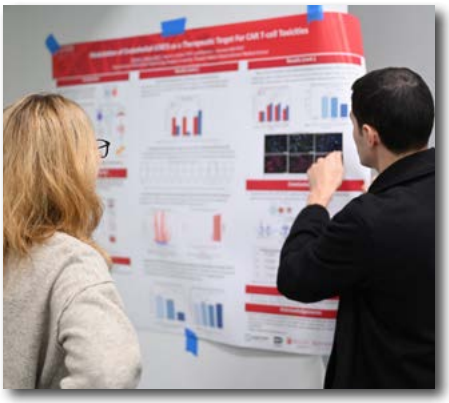
Award Recipients

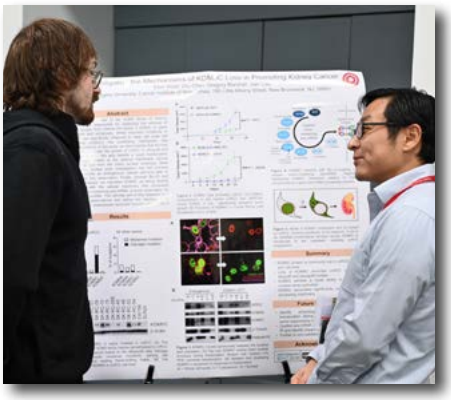
James S. McGarry, recipient of the Legislative Champion Award, pictured with Commission member Loletha Johnson.

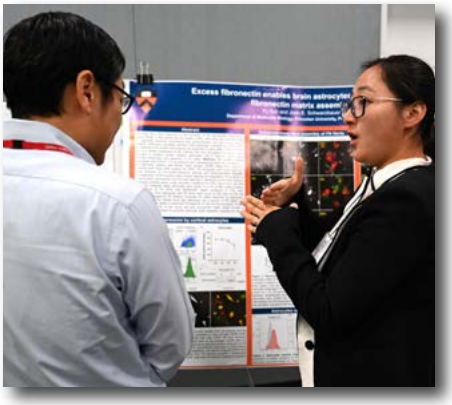
Deborah Q. Belfatto, recipient of the Patient Advocate Award, pictured with Commission chair Kenneth Adler.

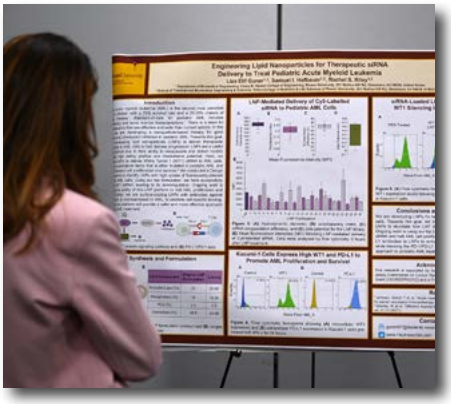
Danelle Davenport, MSc, PhD, recipient of the Dr. Jonathan Yavelow Mentor Award, pictured here with one of her mentees, Parijat Sil, and with Commission Chair Kenneth Adler.

Poster Presentations









Keynote Speaker

Alexander Ploss, PhD

Panel Discussion Members

Soumitra Bhuyan, PhD, MPH

Tanya Borsuk, PhD

Peter Cole, MD

Mary E. O'Dowd, MPH

Ramy Sedhom, MD

Antoinette M. Stroup, PhD

The New Jersey Commission on Cancer Research promotes significant and original research into the causes, prevention and treatment of cancer and serves as a resource to providers and consumers of cancer services.

Appendix I: Cancer Commission on Research Legislation (N.J. Stat § 52:9U-1 (2011))

THIS SECTION IS CURRENT THROUGH NEW JERSEY 214TH LEGISLATURE

2ND ANNUAL SESSION (P.L. 2011 CHAPTER 51 AND JR 3)

STATE CONSTITUTION CURRENT THROUGH THE NOVEMBER, 2010 ELECTION; ANNOTATIONS CURRENT THROUGH MAY 5, 2011.

TITLE 52. STATE GOVERNMENT, DEPARTMENTS AND OFFICERS SUBTITLE I. GENERAL PROVISIONS, CHAPTER 9U. CANCER RESEARCH ACT

GO TO THE NEW JERSEY ANNOTATED STATUTES ARCHIVE DIRECTORY
N.J. Stat.§ 52:9U-1 (2011)

§ 52:9U- 1 . Short title

This act shall be known and may be cited as the "Cancer Research Act."

HISTORY: L. 1983, c. 6, 1, eff. Jan. 17, 1983.

NOTES:
Cross References:
Legislative appropriations; breast cancer research project defined, see 54A: 9-25. 8.
Appropriation of monies deposited, see 54A. 9-25.8.

§ 52:9U-2. Legislative findings and declarations
The Legislature finds and declares that, although this State has the highest cancer death rate in the nation for many of the most frequently fatal types of cancer, it has provided relatively little encouragement for cancer studies at any of its local institutions involved in basic biological research; and that this failure has made New Jersey unattractive for the recruitment of highly skilled cancer investigators, has reduced the State's capacity to compete for its fair share of federal and private research dollars, and has been responsible for delaying the development of services and facilities necessary to conduct productive research. New Jersey's failure to make a concerted and intense effort in the war against cancer has deprived its citizens orthe benefits resulting from the latest advances in basic cancer research.

The Legislature further finds that the State can ill afford to continue its present policy in this regard. Corrective measures should be adopted promptly and funded adequately to make up for lost ground and to make the State competitive in the area of cancer research within the next 5 years.

HISTORY: L. 1983,c.6,2, eff.Jan. 17, 1983.

§ 52:9U-3. Definitions
As used in this act:
a. "Approved research project" means a scientific research project, which is approved by the commission and which focuses on the genetic, biochemical, viral, microbiological and environmental causes of cancer, and may include, but is not limited to, behavioral, socio-economic, demographic and

psychosocial research or research into methods of clinical treatment; or which focuses on pain management and palliative care for persons diagnosed with cancer.

b. "Commission" means the New Jersey State Commission on Cancer Research established pursuant to this act.

c. "Institutional support services" means all services, facilities, equipment, personnel and expenditures associated with the creation and maintenance of approved research projects.

d. "Qualifying research institution" means the Institute for Medical Research in Camden, New Jersey, the University of Medicine and Dentistry of New Jersey, Rutgers--The State University, Princeton University and any other institution approved by the commission, which is conducting an approved research project.

HISTORY: L. 1983, e. 6, § 3: amended 1984, c. 237, §I: 2000, c. 63, § 1, eff. July 13, 2000.

§ 52:9U-4. Commission on Cancer Research

a. There is established in the Executive Branch of the State government, the New Jersey State Commission on Cancer Research. For the purposes of complying with the provisions of Anidt! I·, Section II', paragraph I oftht! New Jersey Constitution, the commission is allocated within the Department of Health, but notwithstanding that allocation, the commission shall be independent of any supervision or control by the department or by any board or officer thereof.

b. The commission shall consist of 11 members, including the Commissioners of the Department of Health and the Depaitment of Environmental Protection or their appointed designees, and nine citizens of New Jersey or persons otherwise associated with the State, who are known for their knowledge, competence, experience or interest in medical research, appointed by the Governor with the advice and consent of the Senate.

c. The term of office of each appointed member shall be three years, but of the members first appointed, three shall be appointed for terms of one year, three for terms of two years, and one for a term of three years. The terms of office of the two additional members appointed pursuant to this amendatory act shall expire upon the expiration of the term of office of the member first appointed for a term of three years. All vacancies shall be filled for the balances of the unexpired tenns in the same manner as the original appointments. The members of the commission shall not receive any compensation for their services, but shall be reimbursed for the actual and necessary expenses incuffed in the performance of their duties as members of the commission.

§ 52:9U-5. Duties of commission

The commission shall:

a. Review and authorize approved research projects;

b. Apportion all available funds to qualifying resean;h institutions to finance approved research projects and necessary institutional support services;

The New Jersey State Commission on Cancer Research shall solicit, receive. evaluate and approve applications of qualified research institutions for grants from the "New Jersey Breast Cancer Research Fund," established pursuant to section I of P.L.1995, c.26 (C 5-IA: 9-25.7), to conduct research relating to the causes, prevention, screening, treatment and cure of breast cancer.

Appendix I: Cancer Commission on Research Legislation (N.J. Stat § 52:9U-1 (2011)

c. Ensure that funds appropriated to approved research projects are not diverted to any other use;
d. Take steps necessary to encourage the development within the State of research projects on:
(1) the causes of cancer; and
(2) pain management and palliative care for persons diagnosed with cancer;
e. Compile a directory of all cancer research projects being conducted in the State; and
f. Provide the Governor and the Legislature with a report by January 30 of each year describing the status of the commission's activities and the results of its funded research efforts.

The commission is authorized to:
a. Adopt rules and regulations concerning the operation of the commission, the functions and responsibilities of its officers and employees and other matters as may be necessary to cany out the purposes of this act;
b. Maintain otrices at such places within the State as it may designate;
c. Employ an executive director and other personnel as may be necessary, whose employment shall be in the unclassified service of the State, except that employees performing stenographic or clerical duties shall be appointed pursuant to Title 11 (Civil Service) of the Revised Statutes;
d. Design a fair and equitable system for the solicitation, evaluation and approval of proposals for cancer research projects;
e. Apply for and accept any grant of money from the federal government, which may be available for programs relating to research on the causes of cancer;
f. Enter into contracts with individuals, organizations and institutions necessary or incidental to the performance of its duties and the execution of its powers under this act: and
g. Accept gilts, grants and bequests offunds from individuals, foundations, corporations, governmental agencies and other organizations and institutions.

§ 52:9U-6. I. Grants; qualified research institution defined
The New Jersey State Commission on Cancer Research shall solicit, receive. evaluate and approve applications of qualified research institutions for grants from the "New Jersey Breast Cancer Research Fund," established pursuant to section I of P.L.1995, c.26 (C 5-IA: 9-25.7), to conduct research relating to the causes, prevention, screening, treatment and cure of breast cancer. As used in this section. "qualified research institution" may include academic medical institutions, State or local government agencies, public or private organizations within New Jersey, and any other institution approved by the commission, which is conducting a breast cancer research project.

§ 52:9U-6.2. Applications for grants
The New Jersey State Commission on Cancer Research shall solicit, receive, evaluate and approve applications of qualified research institutions for grants from the "New Jersey Prostate Cancer Research Fund," established pursuant

to section I of P.L. 200 I, c. 30S (C 54-A: 9-25.21), to conduct research relating to the causes, prevention, screening, treatment and cure of prostate cancer. As used in this section, "qualified research institution" may include academic medical institutions, State or local government agencies, public or private organizations within New Jersey, and any other institution approved by the commission, which is conducting a prostate cancer research project.

§ 52:9U-6.3. Grants from "New Jersey Lung Cancer Research Fund"
The New Jersey State Commission on Cancer Research shall solicit, receive, evaluate and approve applications of qualified research institutions for grants from the "New Jersey Lung Cancer Research Fund," established pursuant to section I of P.L.2009, c.172 (C. 54A: 9-25.27), to conduct research relating to the causes, prevention, education, screening, treatment and cure of lung cancer. As used in this section, "qualified research institution" may include academic medical institutions, State or local government agencies, public or private organizations within New Jersey, and any other institution approved by the commission, which is conducting a lung cancer research project.

§ 52:9U-7. Chairman and vice-chairman; election; duties; duties of executive director
The members of the commission shall annually elect a chairman and a vice-chairman from among their number. The chairman shall be the chief executive officer of the commission, shall preside at all meetings of the commission and shall perform other duties that the commission may prescribe.

The executive director shall serve as secretary to the commission and shall carry out its policies under the direction of the chairman.

§ 52:9U-8. Annual appropriation
$ 1,000,000.00 shall be appropriated annually from the Cancer Research Fund established by P.L.1982. c. 40 (C 54:40A-8 et al.) to effectuate the purposes of this act, except that only $ 500,000.00 shall be appropriated from the fund in fiscal year 1982- 1983.

§ 52:9U-9. Research facilities; direct application for or receipt of funds from public or private agency
Nothing in this act shall preclude a qualifying research institution or any other research facility in the State from directly applying for or receiving funds from any public or private agency to conduct cancer research.

A dedicated non-lapsing is one approach to provide stable and predictable funding for cancer research. Additional funding will facilitate robust innovation and new therapies aimed at treating cancer.

Appendix II: New Jersey Pediatric Cancer Research Fund Statutes

CHAPTER 210 *(CORRECTED COPY)*

AN ACT concerning pediatric cancer research, supplementing chapter 9 of Title 54A of the New Jersey Statutes and supplementing and amending P.L.1983, c.6 (C.52:9U-l et seq.).

BE IT ENACTED by the Senate and General Assembly of the State of New Jersey:

C.54A:9-25.47 "New Jersey Pediatric Cancer Research Fund."

1. a. There is established in the Department of the Treasury a special fund to be known as the "New Jersey Pediatric Cancer Research Fund."

b. Each taxpayer shall have the opportunity to indicate on the taxpayer's New Jersey gross income tax return that a portion of the taxpayer's refund or an enclosed contribution shall be deposited in the special fund.

c. Any costs incurred by the Division of Taxation for collection or administration attributable to this act may be deducted from receipts collected pursuant to this act, as determined by the Director of the Division of Budget and Accounting in the Department of the Treasury. The State Treasurer shall deposit net contributions collected pursuant to this section to the "New Jersey Pediatric Cancer Research Fund."

d. The Legislature shall annually appropriate all funds deposited in the "New Jersey Pediatric Cancer Research Fund" established pursuant to this section to the New Jersey State Commission on Cancer Research, established pursuant to section 4 of P .L.1983, c.6 (C.52:9U-4), for pediatric cancer research projects.

e. As used in this section, "pediatric cancer research project" means a scientific research project approved pursuant to section 2 of P.L.2021, c.210 (C.52:9U-6.4), which scientific research proj ect focuses on the causes, prevention, education, screening, treatment, or cure of pediatric cancer, or the symptoms or effects experienced by patients following completion of a course of treatment for pediatric cancer, and may include, but shall not limited to, basic, clinical, and epidemiologic research.

C.52:9U-6.4 Applications for grants.

2. The New Jersey State Commission on Cancer Research, in con~l. 1tation with theadvisory group established pursuant to section 3 of P.L.2021, c.210 (C.:t2:9U-6.5), shall solicit, receive, evaluate and approve applications of qualifying research institutions for grants from the "New Jersey Pediatric Cancer Research Fund," established pursuant to section 1 ofP.L.2021, c.210 (C.54A:9-25.47), to fund pediatric cancer research projects.

C.52:9U-6.5 Advisory group.

3. a. The New Jersey State Commission on Cancer Research shall establish an advisory group within the commission which shall be responsible for advising the commission on how moneys from the fund established pursuant to section 1 of P.L.2021, c.210 (C.54A:9-25.47) to support pediatric cancer research projects will be distributed by the commission.

4. Section 3 ofP.L.1983, c.6 (C.S2:9U-3) is amended to read as follows:
C.S2:9U-3 Definitions.

3. As used in this act:

"Approved research project" means a scientific research project, which is approved by the commission and which focuses on the genetic, biochemical, viral, microbiological and environmental causes of cancer, and may include, but is not limited to, behavioral, socioeconomic, demographic and psychosocial research or research into methods of clinical treatment; or which focuses on pain management and palliative care for persons diagnosed with cancer.

"Commission" means the New Jersey State Commission on Cancer Research established pursuant to this act.

"Institutional support services" means all services, facilities, equipment, personnel and expenditures associated with the creation and maintenance of approved research projects.

The Pediatric Cancer Research Advisory Group held its first meeting in April 2022. This group consists of seven members who either treat patients with pediatric cancer, conduct research into pediatric cancer, advocate to advance pediatric cancer research or treatment, or have been affected by their own or a family member's diagnosis of pediatric cancer.

Appendix II: New Jersey Pediatric Cancer Research Fund Statutes

"Pediatric cancer research project" means a scientific research project approved pursuant to this act, which scientific research project focuses on the causes, prevention, education, screening, treatment, or cure of pediatric cancer, or the symptoms or effects experienced by patients following completion of a course of treatment for pediatric cancer, and may include, but shall not be limited to, basic, clinical, and epidemiologic research.

"Qualifying research institution" means the Coriell Institute for Medical Research in Camden, New Jersey, Rutgers--The State University, Rowan University, Princeton University and any other institution approved by the commission, which is conducting an approved research project. For the purposes of sections 2 through S of P.L.2021, c.210 (C.S2:9U-6.4 et al.), "qualifying research institution" may include academic medical institutions, State or local government agencies, public or private organizations within New Jersey, and any other institution approved by the commission, which is conducting a pediatric cancer research project.

5. Section S ofP.L.1983, c.6 (C.S2:9U-S) is amended to read as follows:
C.S2:9U-S Duties of commission.

5. The Commission shall:

a. Review ard authorize approved research projects;

b. Apportion all available funds to qualifying research institutions to finance approved research projects and necessary institutional support services;

c. Ensure that funds appropriated to approved research projects are not diverted to any other use;

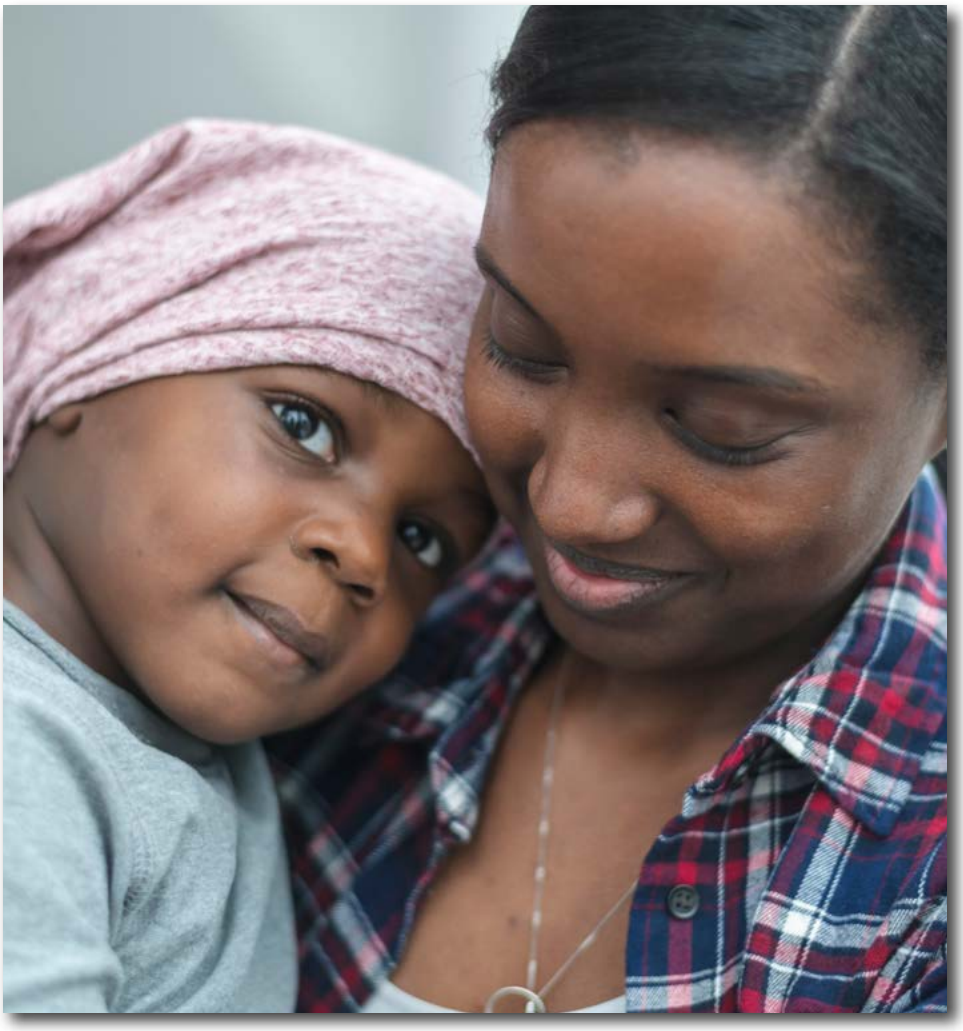
d. Take steps necessary to encourage the development within the State of research projects on:
(1) the causes of cancer; and
(2) pain management and palliative care for persons diagnosed with cancer;

6. Section 8 of P.L.1983, c.6 (C.52:9U-8) is amended to read as follows:
C.52:9U-8 Annual appropriation.

8. $1,000,000.00 shall be appropriated annually from the Cancer Research Fund established by P.L.1982, c40 (C.54:40A-8 et al.) to effectuate the purposes of this act, except that only $500,000.00 shall be appropriated from the fund in fiscal year 1982-1983. The full amount of the annual appropriation from the Cancer Research Fund mandated by this section to effectuate the purposes of this act shall be made notwithstanding any monies received by taxpayer voluntary contribution through gross income tax return to any cancer research fund designated for a specific type or category of cancer by State law.

7. This act shall take effect immediately and apply to taxable years beginning on or after January 1 next following enactment.

Approved September 16, 2021.



NJCCR engaged with the Pediatric Advisory Group to assist with the identification and funding of scientific research projects that focus on translational research (from laboratory to the bedside) and pediatric cancer research projects.

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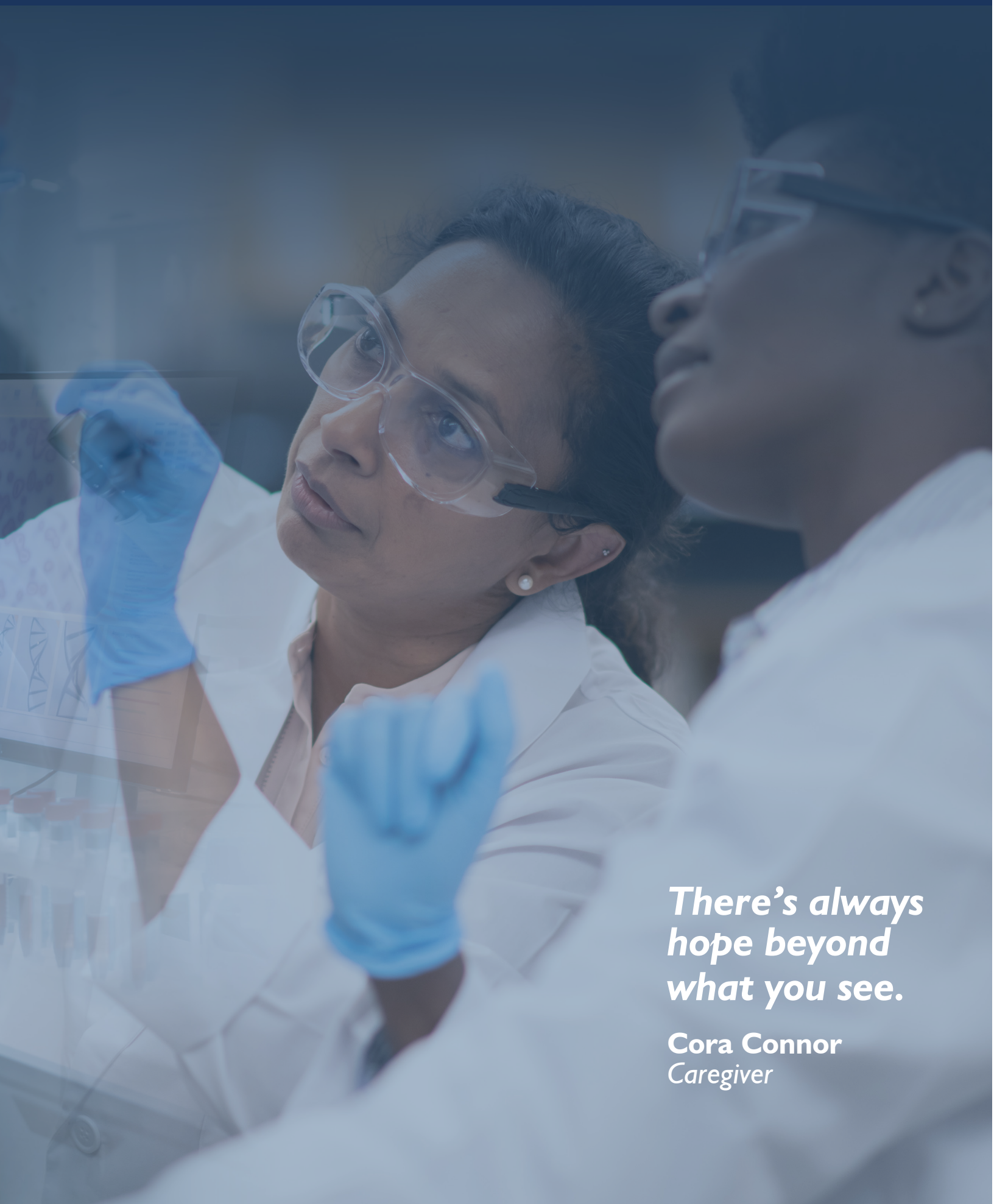

There's always hope beyond what you see.

Cora Connor
Caregiver


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