CRI Cancer Research Institute™

WINTER 2025 CRI IRVINGTON AND IMMUNO-INFORMATICS

Postdoctoral Fellows

CRI Postdoctoral Fellowships

The Cancer Research Institute (CRI) is building a world immune to cancer by investing in the brightest minds at the forefront of immunotherapy. With postdoctoral fellowships that provide $243,000 over three years, CRI enables exceptional young scientists to pursue groundbreaking research and advanced training in immunology, immuno-oncology, and data science at leading universities and research centers around the world.

CRI's commitment to early-career scientists traces back to 1971, when CRI's founding scientific and medical director Dr. Lloyd J. Old established a program to fund postdoctoral researchers studying the immune system and cancer. His idea was to train a new generation of immunologists, building support for immunotherapy from the ground up. Now known as the CRI Irvington Postdoctoral Fellowship, the program has supported over 1600 scientists from 176 institutions in 30 U.S. states and 15 additional countries, many of whom have gone on to make transformative contributions in cancer immunotherapy. In 2022, CRI expanded this vision by launching the CRI Immuno-Informatics Postdoctoral Fellowship to equip the next generation of immunologists with the knowledge and practical tools of bioinformatics and computational biology. This new program has supported 21 scientists from 15 institutions in four U.S. states, Canada, Israel, and Sweden.

Together, these fellowships support researchers investigating some of the most promising frontiers in cancer immunotherapy. With more than $186 million invested to date, CRI selects new fellows twice a year – and in response to recent disruptions in federal science funding, has committed an additional $2.5 million above CRI's annual commitment to fund 10 new fellowships over the next year.

The profiles that follow introduce the Winter 2025 CRI Postdoctoral Fellows – future leaders whose innovative ideas and scientific ingenuity are helping to reshape the future of cancer treatment.

A Letter from Our CEO

A future immune to cancer begins with bold ideas—and the brilliant scientists who pursue them. At CRI, we are proud to support the next generation of leaders in cancer immunotherapy through our steadfast investment in early-career research and training.

This commitment has never been more urgent. In recent years, we have seen a dramatic increase in applications to our fellowship programs, reflecting both the growing momentum in cancer immunotherapy and the exceptional talent entering the field. In response, CRI has deepened its investment. For the Irvington Fellowship alone, we are funding 50 percent more fellows than originally budgeted this cycle, building on the 10 additional fellows we confirmed last year.

We are honored to share the Winter 2025 CRI Irvington Postdoctoral Fellows and Immuno-Informatics Postdoctoral Fellows. These outstanding scientists are advancing pioneering work across tumor immunology, computational biology, and cellular engineering. Their research reflects the urgency, innovation, and collaboration required to transform cancer care.

Each fellow brings a unique perspective, but all share a common goal: to improve the lives of patients and bring lasting hope to families affected by cancer. Their progress is only possible through sustained funding for scientific discovery—support that gives researchers the freedom to pursue bold questions and uncover new answers.

We are deeply honored to support these scientists as they push the boundaries of what's possible. Together, we move closer to a world where cancer is no longer a devastating diagnosis, but a problem science has solved.

Please join us in celebrating the Winter 2025 CRI Postdoctoral Fellows and the hope they carry forward.

With admiration,

Alicia Zhou, PhD
Chief Executive Officer
Cancer Research Institute

CRI Irvington Postdoctoral Fellows

Insights from CRI's Scientific Advisory Council

"Postdoctoral training is one of the most critical periods in a scientist's career — it demands constant reflection, both on the science and of oneself. The CRI fellows selected this year are pursuing some of the most creative, fundamental, and potentially impactful work in immunology and cancer biology. Supporting them now is not just an investment in their futures, but in the future of cancer medicine.

Ellen Puré, PhD
Professor of Biomedical Sciences and Pharmacology and Director of Penn Vet Cancer Center, University of Pennsylvania School of Veterinary Medicine
Associate Director, CRI Scientific Advisory Council

CRI Irvington Postdoctoral Fellow

Emmanuela Adjei-Sowah, PhD

Vanderbilt University

Engineering Lipid Nanoparticles for Targeted Activation of RIG-I to Potentiate Antitumor Immunity in Renal Cell Carcinoma

Mentor:
John Wilson, PhD

Clear cell renal cell carcinoma (ccRCC) is one of the most common and deadly forms of kidney cancer, yet most patients gain little benefit from existing immunotherapies. These tumors are skilled at suppressing local immune activity, while current treatments that broadly activate the immune system often cause damaging inflammation in healthy tissues. Dr. Emmanuela Adjei-Sowah seeks to overcome these barriers by creating a targeted nanomedicine that awakens the body's immune defenses specifically within the tumor—maximizing effectiveness while minimizing harm.

Her research harnesses lipid nanoparticles (LNPs) to deliver a specialized RNA molecule that activates RIG-I, a protein that helps immune cells detect and eliminate cancer. To make this approach safer and more precise, Dr. Adjei-Sowah is engineering a "masked" version of the RNA that stays dormant in circulation and becomes active only once inside the tumor microenvironment. She will optimize this delivery system, then investigate how tumor-selective RIG-I activation alters the immune landscape—reviving exhausted T cells, strengthening long-term immune memory, and reducing the chance of cancer recurrence. Through advanced sequencing tools, she will identify which cells drive these beneficial effects, guiding the design of future immunotherapies.

Dr. Adjei-Sowah's cross-disciplinary expertise in biomaterial engineering, molecular biology, and translational medicine uniquely equips her to design innovative drug delivery systems and develop next-generation immunotherapies that overcome resistance and immune evasion in cancer. By finding new ways to "heat up" cold tumors, this research could greatly expand the number of patients who benefit from immunotherapy and pave the way for safer, more effective cancer treatments.

CRI Irvington Postdoctoral Fellow

Maria Cardenas Conti, PhD

University of Pennsylvania

Intrinsic and Extrinsic Regulation of Stem-like CD4 T Cell Differentiation in Cancer

Sponsor:
E. John Wherry, PhD

Immunotherapy has revolutionized cancer treatment, yet many patients still do not achieve lasting benefit. Among the key players in anti-tumor immunity, CD4 T cells act as essential coordinators, helping other immune cells mount effective and sustained responses. A unique subset known as "stem-like" CD4 T cells has the potential to drive long-term immunity by continuously generating new effector cells. However, in cancer, these same cells often take a wrong turn—producing suppressive cells that weaken immune attack rather than strengthen it. Understanding what determines whether stem-like CD4 T cells become helpers or suppressors could be the key to making immunotherapy more consistently effective.

Dr. Maria Cardenas Conti's research seeks to uncover the molecular and cellular factors that guide stem-like CD4 T cell fate in cancer. Using cutting-edge genomic and immunologic approaches, she will identify the transcriptional regulators, inhibitory receptors, and cellular partners that control whether these cells sustain or suppress anti-tumor immunity. By defining how stem-like CD4 T cells are maintained and how they can be redirected toward beneficial immune functions, her work could uncover new strategies to improve the durability and breadth of immunotherapy responses across cancer types.

Dr. Cardenas Conti is an emerging leader in cancer immunology whose research has illuminated new pathways of T cell regulation and anti-tumor immunity. Her cross-disciplinary expertise—from T cell biology and bioinformatics to translational cancer models—positions her to redefine how the immune system can be guided to achieve lasting cancer control and long-term patient benefit.

CRI Irvington Postdoctoral Fellow

Xueyang Dong, PhD

Harvard University

Systematic Discovery of Microbiome-Derived T Cell Antigens for Colon Cancer Immunotherapy

Mentor:
Kazuki Nagashima, MD, PhD

The immune system's T cells play a central role in recognizing and eliminating cancer, yet how they respond to the trillions of bacteria in the gut—and how these microbes influence cancer progression—remains a major unanswered question. In colorectal cancer, the gut microbiome can shape both anti-tumor immunity and responses to immunotherapy, but the exact bacterial antigens that T cells recognize are largely unknown. Understanding these immune–microbiome interactions could open the door to new ways of improving cancer treatment and reducing side effects.

Dr. Xueyang Dong is developing a groundbreaking high-throughput platform to systematically identify microbial and tumor antigens recognized by T cells. By combining a novel cell-fusion–based method with comprehensive microbiome antigen libraries, his work will map how T cells respond to both bacterial communities and colorectal tumors. This research will clarify how specific microbes and their antigens shape immune cell behavior within tumors and influence patient responses to therapies such as immune checkpoint blockade. The insights gained could inform microbiome-based interventions to enhance anti-tumor immunity while minimizing harmful inflammation.

Dr. Dong is a chemical biologist whose cross-disciplinary expertise bridges microbiology, immunology, and chemical biology. He has developed genetic tools for previously intractable gut bacteria and uncovered new molecular pathways governing microbial metabolism. Building on this foundation, his work now seeks to translate these discoveries into cancer immunology—integrating the microbiome into personalized immunotherapy design and paving the way for safer, more effective treatments for colorectal and other cancers.

CRI Irvington Postdoctoral Fellow

Ron Gejman, MD, PhD

Columbia University in the City of New York

Precision Base-Edited T Cell Therapy in Transplant Recipients with Cutaneous Squamous Cell Carcinoma

Sponsor:
Benjamin Izar, MD, PhD

Organ transplant recipients must take lifelong immunosuppressive drugs to prevent rejection of their transplanted organ. While lifesaving, these drugs weaken the immune system and make patients highly vulnerable to aggressive cancers, such as skin cancers. Unfortunately, the most effective modern cancer treatments—immunotherapies that activate the body's immune cells—are too risky for transplant patients because they can trigger rejection of the donor organ. Dr. Ron Gejman's research aims to solve this dilemma by developing a new type of personalized immune-based therapy that can safely target cancer in transplant recipients without endangering their transplant.

His approach involves collecting a patient's own T cells, reprogramming them in the laboratory so that they resist immunosuppressive drugs, and then returning them to the patient to attack cancer cells. Using advanced base-editing technology, Dr. Gejman will modify key genes to make these T cells both drug-resistant and tumor-specific. He is also developing laboratory models using cells from transplant patients with skin cancer to test how these engineered immune cells function in realistic conditions. Together, these efforts aim to create the first T cell therapies designed specifically for immunosuppressed patients.

Dr. Gejman is a physician-scientist with deep expertise in immunology, oncology, and genetic engineering. His earlier research led to a widely used platform for mapping T cell reactivity and revealed how tumor diversity limits immune attack. Building on this foundation, his current work could transform care for transplant recipients by enabling safe, precision-engineered T cell therapies—offering powerful new options where none currently exist.

CRI Irvington Postdoctoral Fellow

Zhouping Hong, PhD

Boston Children's Hospital

Investigating the NLRP3 and NLRP1 Super-Inflammasome

Sponsor:
Hao Wu, PhD

The innate immune system serves as the body's first line of defense against infection and injury, relying on inflammasomes—molecular signaling platforms that detect danger and trigger inflammation. Each inflammasome is built from a family of proteins known as NOD-like receptors (NLRs), but how these individual inflammasomes might cooperate with one another remains an open question in immunology. Dr. Zhouping Hong's research explores the bold idea that different NLRs can assemble together into "super-inflammasome" complexes—coordinated structures that amplify immune signaling and may play key roles in inflammation, autoimmune disease, and cancer.

Focusing on two major inflammasomes, NLRP3 and NLRP1, Dr. Hong is combining advanced biochemical, biophysical, and structural approaches to uncover how these complexes form and function. Her studies aim to determine whether the two inflammasomes physically interact, how this cooperation enhances immune activation, and what molecular mechanisms govern their assembly. By establishing the first mechanistic framework for super-inflammasome formation, this work could transform our understanding of how innate immunity is regulated and identify new therapeutic targets for inflammation-driven diseases.

Dr. Hong is a biochemist and structural biologist whose research bridges molecular mechanisms and immune regulation. Her expertise spans biochemistry, membrane biology, and structural immunology, and she has made key discoveries on lipid transport between organelles and protein–lipid interactions. Now applying this rigorous training to innate immunity, she is uncovering how inflammasome networks coordinate immune defense—insights that could ultimately guide new strategies to control inflammation and treat immune-related disorders.

CRI Irvington Postdoctoral Fellow

Fubo Ji, PhD

The University of Texas Southwestern Medical Center

Dissecting the Role of Lipid Uptake in Cancer Immune Evasion

Mentor:
Javier Garcia Bermudez, PhD

Immunotherapy has transformed cancer treatment by harnessing the body's own immune system to destroy tumors. Yet most patients still fail to benefit, often because their cancer-fighting T cells cannot effectively reach or attack tumors. Dr. Fubo Ji's research explores a surprising new explanation for this problem—competition for fats, or lipids, between tumor cells and immune cells. Cancer cells consume large amounts of "bad cholesterol" (LDL) to support their growth, and emerging evidence suggests this deprives T cells of the lipids they need to function properly. Strikingly, patients with higher LDL levels tend to respond better to immunotherapy, raising the question of whether improving lipid availability could boost anti-tumor immunity.

Dr. Ji's project investigates how tumor LDL uptake interferes with T cell activity and how manipulating lipid levels might restore immune function. He will study how changes in lipid metabolism affect the performance of $CD8^+$ T cells—the immune cells responsible for killing cancer—and test whether adjusting LDL availability enhances responses to widely used immunotherapies such as anti–PD-1 and anti–PD-L1 treatments. By pinpointing which specific lipids support T cell activity, this work could reveal new ways to strengthen the immune response and improve cancer therapy outcomes.

Dr. Ji is a cancer biologist with broad expertise in tumor metabolism, immunology, and therapeutic discovery. His previous research uncovered key genetic and metabolic vulnerabilities in liver cancers and revealed new mechanisms of tumor progression. Building on this foundation, he now seeks to translate insights from cancer metabolism into strategies that enhance immune-based treatments and ultimately improve patient survival.

CRI Irvington Postdoctoral Fellow

Joseph Kern, PhD

Dana-Farber Cancer Institute

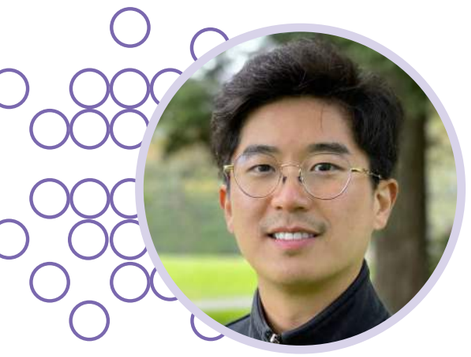
Uncovering Mechanisms of Immune Evasion in Intestinal Tumorigenesis and Regeneration

Sponsor:
Judith Agudo, PhD

One of the major challenges in cancer biology is understanding how tumors escape detection by the immune system. Under normal conditions, the body can identify and destroy cells that begin to grow abnormally. However, early-stage cancer cells often adopt survival strategies that allow them to avoid immune attack. Dr. Joseph Kern's research focuses on uncovering how intestinal tumors—particularly colorectal cancers—evade immune surveillance, and how these mechanisms overlap with the body's natural processes for healing damaged tissue. Understanding this connection could reveal new strategies to prevent cancer development or halt its progression at its earliest stages.

Dr. Kern's project investigates how epithelial cells in the intestine, which regenerate after injury, may exploit similar pathways to protect emerging tumor cells from immune destruction. By studying both intestinal repair and the initiation of colorectal cancer, his research aims to identify the molecular and cellular mechanisms that allow early tumor cells to hide from the immune system. These insights could uncover new therapeutic targets to make precancerous and early cancer cells more visible and susceptible to immune attack, ultimately improving the effectiveness of cancer prevention and immunotherapy.

Dr. Kern is a cancer biologist with expertise in epithelial regeneration and tumor immunology. His earlier research uncovered how tumor-suppressive signaling networks regulate early tumor formation and shape the tumor microenvironment. Building on this foundation, he now applies advanced cellular and molecular tools to probe how cancer begins and persists. By bridging the biology of tissue repair and immune evasion, Dr. Kern's work has the potential to transform early detection and lead to new immune-based strategies for colorectal cancer prevention and treatment.



CRI Irvington Postdoctoral Fellow

Hyung Jun Kim, PhD

University of California, San Francisco

Systematic Engineering of Multifunctional Macrophages for Solid Tumor Immunotherapy

Mentor:
Roarke Kamber, PhD

While modern immunotherapies have revolutionized treatment for certain cancers, they remain largely ineffective against solid tumors such as those in the breast, lung, and ovary. These tumors build protective microenvironments that suppress immune activity and block immune cells from penetrating and destroying cancer tissue. Dr. Hyung Jun Kim's research focuses on overcoming this barrier by engineering a new type of immune cell therapy based on macrophages—versatile immune cells that can naturally enter tumors but often get "reprogrammed" by the tumor to help rather than fight it.

Dr. Kim is systematically redesigning macrophages to restore and enhance their anti-tumor potential. Using genetic engineering and synthetic biology, he is equipping macrophages with specialized receptors that recognize and engulf cancer cells with greater precision. He is also programming them with custom gene circuits that release immune-activating molecules directly within tumors, stimulating other immune cells to join the attack. By combining these complementary strategies, his work aims to create a "living medicine" that both kills cancer cells directly and mobilizes a broader immune response against resistant solid tumors.

Dr. Kim brings exceptional cross-disciplinary expertise spanning genetics, cell biology, and immune engineering. His earlier research revealed key mechanisms of chromosome organization during cell division and advanced genome-scale screening and macrophage engineering technologies. Building on this foundation, he is now developing next-generation, macrophage-based immunotherapies designed to overcome the protective barriers of solid tumors and improve outcomes for patients resistant to current treatments.

CRI Irvington Postdoctoral Fellow

Wantae Kim, PhD

The Scripps Research Institute

Molecular Mechanism by Which THEMIS Controls T-Cell Maturation

Sponsor:
Dorothee Kern, PhD

The immune system constantly produces T cells—specialized defenders that patrol the body to detect and destroy abnormal or cancerous cells. Before they can perform this vital task, T cells must undergo a rigorous education process that teaches them to distinguish healthy cells from dangerous ones. When this process fails, the consequences can be severe: the immune system may either overlook cancer or mistakenly attack the body's own tissues. Dr. Wantae Kim's research focuses on understanding how this education process is controlled, with particular attention to a key protein called THEMIS, which is essential for proper T cell development but whose molecular role remains mysterious.

Dr. Kim aims to uncover how THEMIS coordinates the molecular interactions that guide immature T cells to maturity. Using advanced biochemical and structural biology techniques, he will map how THEMIS communicates with other signaling proteins to ensure that only properly trained T cells survive. By identifying the molecular "switches" that control this process, his work will reveal new insights into how the immune system maintains balance—and how this knowledge might be harnessed to design next-generation immunotherapies that strengthen the body's natural defense against cancer.

Dr. Kim brings a distinctive interdisciplinary background that bridges chemistry, molecular biology, and structural biology. His prior research revealed previously unknown molecular mechanisms in enzyme catalysis, RNA processing, and protein phase separation. By applying this mechanistic precision to immunology, he seeks to illuminate how THEMIS safeguards immune function and to lay the groundwork for therapies that enhance T cell–based cancer treatments.

CRI Irvington Postdoctoral Fellow

Konrad Knopper, PhD

University of California, San Francisco

A Nociceptor Neuron–Macrophage–DC Axis Drives Sexual Dimorphic Skin Immunity

Mentor:
Jason Cyster, PhD

The immune and nervous systems are deeply interconnected, constantly exchanging information to keep the body balanced and responsive to threats. Both systems also show differences between males and females, yet how these differences are linked has remained poorly understood. Dr. Konrad Knopper has uncovered a novel pathway in which sensory neurons known as nociceptors communicate with immune cells to shape immune activity in the skin—and intriguingly, this interaction occurs only in females. His discovery reveals a previously unrecognized neuro-immune circuit that may help explain sex-based differences in immune responses and disease susceptibility.

Dr. Knopper's project aims to uncover the molecular and cellular mechanisms that enable this neuron–immune cell communication. Using advanced genetic, imaging, and CRISPR-based tools in mouse models, he will study how nociceptor neurons signal to macrophages, which then activate dendritic cells—key immune sentinels that alert and coordinate other immune cells—to migrate and initiate protective responses. By mapping this cascade, his work will clarify how nervous system signals influence immunity and identify potential targets to modulate this circuit in diseases such as cancer, chronic inflammation, or autoimmune disorders.

Dr. Knopper is an immunologist with broad expertise in cellular immunology, microscopy, and computational biology. His research bridges fundamental immunology and neuroscience, revealing how communication between the nervous and immune systems shapes immune defense. Building on his discovery of a sex-specific neuro-immune pathway, he seeks to uncover principles that could guide the development of more precise and personalized immune-based therapies.

CRI Irvington Postdoctoral Fellow

Anna Kolarzyk, PhD

Cornell University

Illuminating Immune Cell Trafficking: Spatiotemporal Mapping of Lymphocyte Dynamics in Pancreatic Cancer

Sponsor:
Deborah Fowell, DPhil

Pancreatic ductal adenocarcinoma (PDAC) is among the deadliest forms of cancer, largely because it spreads quickly and resists current immunotherapies. Although considered an "immunologically cold" tumor, PDAC contains many immune cells—some that fight the tumor and others that help it grow. Understanding how these cells move between the tumor and surrounding lymph nodes, where immune responses are coordinated, could reveal new ways to strengthen the body's natural defenses. Dr. Anna Kolarzyk's research focuses on uncovering how the movement and behavior of these immune cells change over time as pancreatic cancer progresses.

Using advanced imaging and cell-tracking technologies, Dr. Kolarzyk has mapped how immune cells circulate between tumors and draining lymph nodes in preclinical models. Her findings show that beneficial immune cells can exit the tumor and travel to lymph nodes, where they stimulate other immune cells, including T cells, to attack cancer more effectively. Interestingly, the types of cells that make this journey vary depending on the tumor's stage, suggesting that the immune system's activity evolves as the disease advances. Building on these insights, her work aims to define how this shifting pattern can be leveraged to design stage-specific immunotherapies for pancreatic cancer.

Dr. Kolarzyk brings broad expertise spanning molecular biology, vascular biology, and cancer immunology. Her work has illuminated how blood and lymphatic vessels influence tumor growth and immune regulation. By mapping how immune cells traffic between tumors and lymphoid tissues, she seeks to uncover new therapeutic strategies that reawaken immune responses and improve outcomes for patients with pancreatic cancer.

CRI Irvington Postdoctoral Fellow

Georgia Lattanzi, PhD

Salk Institute

Deciphering the Mechanisms of B Cell–Mediated Epitope Spreading in Cancer

Mentor:
Daniel Hollern, PhD

Even with major advances in cancer immunotherapy, many tumors continue to evade immune detection and resist treatment. Most current therapies focus on activating T cells—the immune system's direct cancer killers—but another key player, the B cell, may hold untapped potential. B cells can enhance anti-tumor immunity by producing antibodies and by presenting tumor fragments, or antigens, to T cells to broaden their attack. Dr. Georgia Lattanzi's research seeks to understand how B cells expand and diversify immune responses against cancer, a process that could help overcome resistance to current therapies.

Dr. Lattanzi's project investigates how tiny particles released by tumor cells, known as tumor extracellular vesicles (tEVs), activate B cells to coordinate stronger and more comprehensive immune attacks. Her studies suggest that tEVs carry tumor-associated proteins to nearby lymph nodes, where they stimulate B cells to "teach" T cells to recognize additional tumor targets—effectively broadening the immune system's ability to detect and destroy cancer cells. By uncovering how this B cell–driven communication amplifies T cell responses, Dr. Lattanzi aims to identify new strategies that engage both arms of the immune system to create more durable and effective cancer treatments.

Dr. Lattanzi is a cancer immunologist with expertise spanning molecular oncology, microbiome–immune interactions, and translational immunotherapy. Her research integrates fundamental and applied approaches to understand how immune cells collaborate to eliminate cancer. Building on these insights, she aims to translate discoveries about B and T cell cooperation into new combination immunotherapies that produce stronger, longer-lasting responses for patients who do not benefit from current treatments.

CRI Irvington Postdoctoral Fellow

Yi-Tsang Lee, PhD

La Jolla Institute for Immunology

A Novel Approach to Study T-Cell Activation and Exhaustion

Sponsor:
Patrick Hogan, PhD

T cells play a central role in the immune system's ability to detect and destroy cancer, yet their effectiveness often fades over time, especially in solid tumors. This decline, known as T cell exhaustion, limits the success of promising therapies like CAR T cell treatment. The process is controlled by molecular switches that determine whether T cells remain active or enter a dysfunctional state. One such switch involves the transcription factor NFAT, which can drive either immune activation or exhaustion depending on its binding partners. Dr. Yi-Tsang Lee's research aims to uncover how these molecular partnerships shape T cell behavior and how they might be redirected to sustain anti-tumor activity.

Dr. Lee is developing an innovative "event-triggered" molecular labeling system that marks proteins only when they form cooperative complexes on DNA. Using this approach, he will map how NFAT pairs with different partners—such as AP1 or NR4A3—to control gene programs involved in T cell activation and exhaustion. This cutting-edge platform will allow researchers to visualize these dynamic interactions with unprecedented precision, revealing how shifts in NFAT's binding patterns can either sustain immune responses or drive exhaustion.

Dr. Lee is a molecular biologist and protein engineer whose work bridges synthetic biology, gene regulation, and cancer research. His earlier studies created molecular tools that control cellular activity with light or small molecules. Building on this expertise, his current research uses these engineering approaches to better understand and improve T cell–based cancer therapies. At the same time, the new technology he is developing offers a powerful platform to study how genes are switched on and off across many biological systems, broadening its potential impact well beyond cancer.

CRI Irvington Postdoctoral Fellow

Shi Li, PhD

Fred Hutchinson Cancer Center

Purinergic Checkpoints Governing Microglial Clearance of Disseminated Tumor Cells

Sponsor:
Cyrus Ghajar, PhD

Co-Sponsor:
Evan Newell, PhD

When cancer spreads to the brain, it often establishes hidden reservoirs of dormant tumor cells long before forming detectable metastases. These disseminated tumor cells (DTCs) evade immune clearance in part because the brain's immune environment is unique—lacking many conventional immune cells and relying instead on specialized brain-resident cells called microglia. Although microglia can detect invading tumor cells, they rarely eliminate them under normal conditions. Dr. Shi Li's research seeks to understand why these cells remain inactive and how to reawaken their tumor-fighting potential.

Dr. Li has discovered that neighboring support cells in the brain, called astrocytes, send signals that restrain microglial activity against tumor cells. His project will identify these suppressive "checkpoints" and determine how to overcome them by activating a purinergic signaling pathway—an energy-sensing communication system involving molecules such as ATP. Using advanced tools like intravital imaging, spatial transcriptomics, and genetic manipulation, Dr. Li will define how astrocyte and microglial interactions dictate whether tumor cells are ignored or destroyed. He will also test a novel therapeutic approach using nanoparticles that deliver activation signals directly to the tumor niche, potentially preventing brain metastasis before it takes hold.

Dr. Li is a neuroimmunologist and imaging scientist whose background spans nanomedicine, molecular imaging, and brain tumor biology. His work bridges cutting-edge imaging with immune mechanism discovery to reveal how the brain's own cells can be harnessed to fight cancer.

CRI Irvington Postdoctoral Fellow

Zhiyuan Mao, PhD

University of California, Los Angeles

Deciphering Heterogeneous Tumor Responses to Immunotherapy by Profiling Cancer–T Cell Doublet Interactions

Sponsor:
John Lee, MD, PhD

Solid tumors such as prostate, ovarian, and pancreatic cancers remain among the most difficult to treat, often resisting even the most advanced forms of immunotherapy. These epithelial cancers are highly diverse, with tumor cells using multiple strategies to hide from immune attack. A key unanswered question is why some tumor cells are effectively eliminated by engineered T cells while others survive. Understanding these differences could reveal how to make immunotherapies more effective for the majority of cancer patients.

To address this, Dr. Zhiyuan Mao has developed an innovative technology called Cell-Cell-Seq, which captures and analyzes a single cancer cell paired with a single T cell inside a microscopic droplet. This allows researchers to measure, in real time, how individual immune cells interact with different tumor cells and how those encounters shape therapeutic success or failure. By mapping both the molecular "conversations" and the outcomes of these interactions across diverse epithelial cancers, Dr. Mao aims to uncover the genetic and cellular features that determine whether tumors resist or respond to immunotherapy.

Dr. Mao is a cancer immunologist and bioengineer whose work bridges molecular biology, computation, and biophysics. His pioneering platform combines single-cell sequencing with precision microengineering to study immune–tumor interactions at unprecedented resolution. By revealing the hidden rules that govern how T cells recognize—or fail to recognize—cancer cells, his research could guide the design of more effective, personalized immunotherapies for patients with currently treatment-resistant cancers.

CRI Irvington Postdoctoral Fellow

Andrea Muñoz Zamora, PhD

Icahn School of Medicine at Mount Sinai

In Search of Immune Engrams: Mapping the Brain's Memory of Systemic Inflammation

Sponsor:
Michel Enamorado, PhD

Sepsis—a life-threatening condition that occurs when the body's response to infection causes widespread inflammation—affects more than 1.7 million people in the U.S. each year. Cancer patients are particularly vulnerable, facing higher risks of developing sepsis and dying from its complications. Yet the long-term effects of severe infections like sepsis on tumor growth and immune function remain largely unknown. At the same time, new research suggests that the brain can influence the immune system—and that activating certain brain regions can suppress tumor progression. Dr. Andrea Muñoz Zamora's research seeks to uncover how the brain stores and reactivates "immune memories" from diseases such as sepsis and cancer, and whether these memories can be harnessed to strengthen the body's defenses against tumors.

Building on her pioneering work in memory and brain–body communication, Dr. Muñoz Zamora is mapping the neurons that encode immune-related information—what she calls immune engrams. She will determine whether the same brain circuits record immune responses to both infection and cancer, and test whether reactivating these neurons can alter immune cell activity or slow tumor growth. Using state-of-the-art tools such as neuronal labeling and optogenetics, her project aims to uncover how the brain's "memory" of inflammation might be used to boost immune function and fight disease.

Dr. Muñoz Zamora brings a rare combination of training in psychology, neuroscience, and physiology, along with extensive experience in mapping how memories are formed and influence the body. Her background in both behavioral and cellular neuroscience positions her to bridge the gap between brain research and immunology, uncovering new principles that could guide the development of novel strategies to enhance immune health and cancer therapy.

CRI Irvington Postdoctoral Fellow

Qinli Sun, PhD

Stanford University

Therapeutic Development of Engineered TGF-β Agonists for Autoimmunity and Cancer

Sponsor:
K. Christopher Garcia, PhD

The immune system must strike a delicate balance—strong enough to attack infections and cancer, yet controlled enough to avoid damaging healthy tissues. A key regulator of this balance is a molecule called TGF-β, which can either suppress or activate immune responses depending on the context. In cancer, TGF-β often promotes tumor growth and immune evasion, while in autoimmune diseases, it helps maintain tolerance and prevent inflammation. Because of its dual roles, targeting TGF-β has proven challenging, and past attempts to block it broadly have failed in clinical trials. Dr. Qinli Sun's research takes a new approach: rather than blocking TGF-β, he is engineering precise, targeted versions of the molecule that can activate beneficial immune pathways while avoiding harmful side effects.

Building on this idea, Dr. Sun has created an innovative TGF-β agonist platform that allows selective activation of TGF-β signaling in specific immune cells. As a proof of concept, he engineered a combined IL-2–TGF-β agonist that induces regulatory T cells—immune cells that calm inflammation and promote tolerance—without affecting unrelated tissues. This engineered molecule effectively reduced airway inflammation in preclinical studies, demonstrating the potential of such "smart" cytokine therapies. Now, Dr. Sun is expanding this platform to design new agonists that could suppress autoimmunity or fine-tune immune responses to improve cancer immunotherapy.

Dr. Sun brings extensive expertise in T cell biology, cytokine signaling, and protein engineering. His work bridges fundamental immunology and translational medicine, aiming to turn complex immune regulators like TGF-β into precise therapeutic tools. By reprogramming how immune cells interpret TGF-β signals, his research could lead to safer, more effective treatments for autoimmune diseases, chronic inflammation, and cancer.

CRI Irvington Postdoctoral Fellow

Lion Uhl, DPhil

Memorial Sloan Kettering Cancer Center

Molecular Mechanisms of Fine-Tuning of the Regulatory T cell Transcriptional and Functional Program by Interleukin-2

Mentor:
Alexander Rudensky, PhD

The immune system must constantly balance attack and restraint—fighting infections and cancer while avoiding the harmful inflammation that leads to autoimmune disease. Regulatory T cells (Tregs) are central to maintaining this balance, acting as the body's immune "brakes." One of the key molecules guiding their function is interleukin-2 (IL-2), a signaling protein that also fuels the activity of other immune cells. Yet how Tregs interpret IL-2 signals to adapt their behavior in different inflammatory environments remains poorly understood.

Dr. Lion Uhl's project explores how immune cells decide when to activate or hold back, focusing on the partnership between two proteins inside Tregs—Foxp3, which defines their identity, and STAT5, which responds to IL-2 signals. He is investigating how Foxp3 fine-tunes STAT5's activity, helping Tregs adjust their response to maintain immune balance under stress. By uncovering how this molecular dialogue shapes Treg function, Dr. Uhl's research could reveal new ways to strengthen immune control in cancer or restore it when it fails in autoimmune disease.

Dr. Uhl brings an interdisciplinary background spanning immunology, cytokine signaling, and infection biology. His prior work uncovered mechanisms that preserve diversity within CD8 T cell responses and revealed new ways that cytokine communication shapes immune memory. Building on this foundation, he now aims to decode how IL-2 and Foxp3 cooperate to direct immune tolerance—knowledge that could ultimately guide the development of more precise cellular immunotherapies and targeted treatments for immune-related diseases.

CRI Irvington Postdoctoral Fellow

Hao Wang, PhD

Dana-Farber Cancer Institute

Modulation of T cell-B Cell Crosstalk in Tertiary Lymphoid Structures Via the CD161-CLEC2D Pathway

Sponsor:
Kai Wucherpfennig, MD, PhD

Immune checkpoint therapies have revolutionized cancer treatment by reawakening the body's immune system to attack tumors. Yet, for most patients, these treatments still fail to produce lasting benefits. Dr. Hao Wang's research explores an alternative strategy—strengthening special immune hubs that form inside some tumors, known as tertiary lymphoid structures (TLSs). These organized clusters of T cells and B cells act like miniature lymph nodes, coordinating immune attacks against cancer. Tumors that contain TLSs are often more responsive to therapy and linked to improved patient outcomes.

Dr. Wang's project focuses on a newly identified inhibitory pathway called CD161–CLEC2D, which may suppress immune activity within TLSs. Using an innovative humanized mouse model, he will test whether blocking this pathway can enhance communication between T cells, B cells, and natural killer (NK) cells, boosting the tumor-fighting capacity of TLSs. His studies will also examine how CD161 blockade influences tumor control in models of lung and ovarian cancer, where TLSs naturally arise. This work could uncover a powerful new way to engage the immune system, complementing existing checkpoint therapies and improving outcomes for patients with solid tumors.

Dr. Wang brings a unique combination of expertise in molecular genetics, immunology, and translational cancer research. His previous discoveries revealed new immune checkpoint pathways and led to antibody-based strategies that enhance immune attack against cancer. Building on this foundation, he now aims to develop therapies that not only "release the brakes" on T cells but also reprogram the broader immune ecosystem within tumors—paving the way for more effective and durable cancer immunotherapies.

CRI Irvington Postdoctoral Fellow

Hejia Wang, MD, PhD

Johns Hopkins University

T-cell Determinants of Clinical Response to a Mutant KRAS Vaccine in Colorectal Cancer

Mentor:
Elizabeth Jaffee, MD

Colorectal cancer is the second leading cause of cancer deaths in the United States, and for patients whose disease has spread, survival rates remain dismally low. While immune checkpoint inhibitors have revolutionized cancer care, they benefit only a small fraction of colorectal cancer patients because most tumors evade immune recognition. A common mutation in the KRAS gene—found in roughly 40% of colorectal cancers—offers a promising new target for therapy. Dr. Hejia Wang is investigating how the body's immune system can be trained to recognize this mutation through a novel KRAS-targeted vaccine. In an early clinical trial, combining this vaccine with checkpoint inhibitors led to tumor shrinkage in several patients with advanced colorectal cancer, offering hope that targeting mutant KRAS could transform treatment for this difficult-to-treat cancer.

Dr. Wang's project seeks to uncover why only some patients respond to this combination therapy. Using blood and tumor samples from trial participants, he will isolate and study the T cells activated by the vaccine to determine how effectively they recognize and attack tumor cells. By mapping the unique molecular and functional features of these immune cells, his work aims to reveal biomarkers that predict response and to refine future vaccine designs that generate more potent and durable anti-tumor immunity.

Dr. Wang brings a blend of expertise in biochemistry, cancer immunology, and clinical oncology. His past research has led to advances in protein engineering and intracellular drug delivery, and his current work bridges laboratory discovery with patient care. By uncovering how KRAS-targeted vaccines engage the immune system, Dr. Wang aims to pave the way for next-generation immunotherapies that extend the life-saving benefits of immunotherapy to more patients with colorectal and other KRAS-driven cancers.

CRI Irvington Postdoctoral Fellow

Yebin Wang, PhD

Harvard Medical School

Tolerization of Goblet Cell Self-Antigens by Thymic Mimetic Cells

Sponsor:
Diane Mathis, PhD

The immune system must strike a delicate balance: protecting the body from infection and cancer while avoiding attacks on healthy tissues. This balance is learned in the thymus, where developing T cells are trained to distinguish "self" from "non-self." Within the thymus, a unique group of teacher cells—known as mimetic thymic epithelial cells—adopt the molecular identity of other organs to help prevent autoimmunity. One rare type, the goblet medullary thymic epithelial cell (goblet mTEC), imitates the mucus-producing goblet cells found in the gut and airways. These mimetic cells may play an essential role in preventing immune attacks that lead to inflammatory diseases of barrier tissues such as the intestine and lung.

Dr. Yebin Wang's project investigates how goblet mTECs educate T cells to tolerate self-antigens from mucosal tissues and what happens when this process fails. Using advanced single-cell and spatial genomics approaches, his research will map how these thymic cells model goblet cell identity and interact with developing T cells. By comparing findings from animal models to patient data from inflammatory bowel disease, Dr. Wang aims to uncover how defects in this "immune education" contribute to chronic inflammation. This knowledge could reveal new strategies to prevent autoimmunity and guide the safer design of cancer immunotherapies that modulate immune tolerance.

Dr. Wang brings an interdisciplinary background spanning bioinformatics, developmental biology, and immunology. His earlier research uncovered how cells regulate growth, tissue organization, and migration—fundamental processes that maintain healthy tissues and, when disrupted, contribute to cancer and immune imbalance. Building on this foundation, he now applies advanced molecular and computational approaches to uncover how the thymus maintains immune tolerance, with the goal of translating these insights into strategies to prevent autoimmunity and improve immune-based cancer therapies.

CRI Immuno-Informatics Postdoctoral Fellows

Insights from CRI's Scientific Advisory Council

"Postdocs are the invisible engine of biomedical discovery. At a time when federal support is faltering, CRI is once again stepping up – just as it did decades ago when cancer immunology had few champions. Supporting these young scientists is not just an act of resilience; it's a commitment to the future of science and the patients who depend on it.

Miriam Merad, MD, PhD
Dean of Translational Research and Therapeutic Innovation, Chair of the Department of Immunology and Immunotherapy, and Director of the Precision Immunology Institute, Mount Sinai School of Medicine in New York and Director of the Mount Sinai Human Immune Monitoring Center
Member, CRI Scientific Advisory Council

CRI Immuno-Informatics Postdoctoral Fellow

José Almeida Santos, PhD

Dana-Farber Cancer Institute

Determining the Role of Latent Neonatal T cells in Immunity to Cancer Across the Lifespan

Mentor:
Jared Rowe, MD, PhD

Co-Mentor:
David Liu, MD

Most successful cancer treatments rely on the ability of T cells to recognize and destroy tumor cells, yet many T cells inside tumors become exhausted and lose their ability to function. Neonatal $CD8^+$ T cells—produced early in life—stand out as a naturally resilient population that resists exhaustion, proliferates robustly, and produces a diverse set of inflammatory signals. These unique properties, observed in certain infant cancers that spontaneously regress, suggest untapped potential for improving adult cancer immunotherapy. Dr. José Almeida-Santos is investigating how neonatal-like $CD8^+$ T cells behave in tumors and whether their biology can be harnessed to prevent exhaustion and strengthen anti-cancer immunity.

Dr. Almeida-Santos will combine human tumor datasets with new mouse models to define the molecular and functional characteristics of neonatal $CD8^+$ T cells within the tumor microenvironment. Using cross-species computational analysis, he will determine whether neonatal-like $CD8^+$ T cells exist in human cancers, how they differentiate, and which signals shape their activity. By mapping their cellular interactions and identifying pathways that sustain their resilience, Dr. Almeida-Santos aims to pinpoint molecular targets that could be modulated to preserve T cell vigor, enhance immune infiltration, and improve responsiveness to treatments such as immune checkpoint blockade. These discoveries may guide new therapeutic strategies rooted in the natural strengths of early-life immunity.

Dr. Almeida-Santos brings extensive experience in developmental immunology, T cell biology, and immunotherapy research, spanning gene editing in zebrafish, studies of T cell regulation in cancer, and immuno-informatics applied to human disease. His work integrates experimental models with computational approaches to uncover mechanisms that determine T cell effectiveness. By illuminating how neonatal $CD8^+$ T cells resist exhaustion, his research seeks to provide a blueprint for next-generation immunotherapies that maintain T cell potency and deliver more durable cancer control.

CRI Immuno-Informatics Postdoctoral Fellow

Sui Yuk (Candace) Chan, PhD

University of Texas at Austin

Liquid Biopsy Neomer Profiling for Glioblastoma Cancer Detection and Precise Immunotherapy

Sponsor:
Ilias Georgakopoulos-Soares, PhD

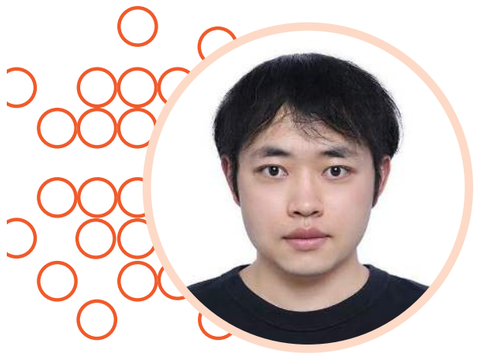
Co-Sponsor:
B.J. Kim, PhD

Glioblastoma is the most aggressive and deadly form of brain cancer, yet it is usually discovered only after symptoms appear—when treatment options are limited and far less effective. Current diagnostic tools rely on invasive brain biopsies, making early detection difficult and leaving many patients without access to personalized immunotherapies that could better target their tumors. Dr. Sui Yuk (Candace) Chan aims to answer a critical question: Can we detect glioblastoma earlier and design individualized treatments using only a blood sample?

Dr. Chan is pursuing this question using neomers, short DNA and RNA sequences that appear only in tumors and never in healthy tissue. When cancer cells die, they release these fragments into the bloodstream, where they can be detected through a simple blood draw. Her early work shows that neomers can identify glioblastoma with accuracy surpassing many FDA-approved detection tools.

Building on this foundation, she will refine a blood-based test for early detection and use the same neomer signals to pinpoint patient-specific immune targets for personalized cancer vaccines—eliminating the need for risky surgical biopsies.

Drawing on extensive expertise in cancer genomics and computational immunology, including pioneering studies using neomers across multiple cancer types, Dr. Chan is positioned to advance a safer, more accessible approach to both diagnosis and precision treatment for glioblastoma. Her research could help open new pathways for improving outcomes and quality of life for patients facing one of the most challenging cancers.



CRI Immuno-Informatics Postdoctoral Fellow

Baolin Liu, PhD

Broad Institute of MIT and Harvard

Dissecting Causal Regulators of Response and Resistance to Checkpoint Immunotherapy in Human Tumors

Mentor:
Nir Hacohen, PhD

Co-Mentor:
Caroline Uhler, PhD

Immune checkpoint blockade (ICB) has transformed cancer treatment, yet many patients still fail to respond, and the biological reasons behind this resistance remain unclear. Researchers have identified gene expression patterns associated with ICB outcomes, but distinguishing correlation from true causation has proven exceptionally difficult—especially in human tumors, where diverse cell types interact to shape therapeutic success. Dr. Baolin Liu, is developing an integrated, systems-level approach to uncover the genes and pathways that directly determine whether ICB succeeds or fails in melanoma, with the goal of revealing new therapeutic targets to overcome resistance.

Dr. Liu will begin by analyzing single-cell RNA-sequencing data from 400 melanoma tumors collected before and after ICB treatment, identifying cell types and gene programs associated with response or non-response. He will then combine this human dataset with a comprehensive suite of immune-relevant CRISPR screens, perturbation-response profiles, curated signaling pathways, and machine learning–based causal inference to pinpoint genes that causally regulate anti-tumor immune activity. By mapping both cell-intrinsic and cell-to-cell mechanisms—including pathways governing tumor cell killing, interferon signaling, antigen presentation, and immune suppression—he aims to determine how specific genes influence therapy outcomes. Top candidates will be experimentally validated to define their mechanisms and nominate new intervention strategies.

Dr. Liu brings deep expertise in single-cell analysis and immunotherapy research, including influential work identifying tumor-reactive CD8⁺ T cell states associated with ICB responsiveness across multiple cancer types. Building on this foundation, his research seeks to move beyond descriptive associations to uncover actionable mechanisms that explain why some tumors respond while others resist. By establishing a causal roadmap of ICB regulation in human melanoma, this work has the potential to guide more precise immunotherapies and improve outcomes for patients who currently derive limited benefit from existing treatments.

CRI Immuno-Informatics Postdoctoral Fellow

Jingya Qiu, PhD

Gladstone Institutes

Defining, Monitoring, and Intercepting Pre-Malignant Immune Hubs

Mentor:
Matthew Spitzer, PhD

Co-Mentor:
Karin Pelka, PhD

Many cancers develop slowly over years, passing through pre-malignant stages where early immune responses may determine whether disease is contained or progresses to invasive cancer. Oral epithelial dysplasia (OED) is one such pre-malignant condition, yet clinicians currently have no reliable way to predict which lesions will transform into oral squamous cell carcinoma. Dr. Jingya Qiu is working to clarify how immune surveillance operates—and ultimately fails—during this early window of disease. Her recent work has shown that a specialized population of CXCL13^{+} CD8^{+} T cells is strongly enriched in OED lesions that later progress to cancer, suggesting that antigen-specific but functionally constrained T cell responses emerge long before tumors become invasive.

Dr. Qiu's research shows that these CXCL13^{+} CD8^{+} T cells sit within conserved "immune hubs" composed of dysplastic epithelial, myeloid, and stromal cells that exchange immune-modulating signals. As OED evolves, these hubs undergo spatial and molecular remodeling linked to immune escape and malignant transformation. Building on this discovery, she will apply advanced spatial transcriptomics, digital pathology, and computer vision to map how immune cell states change over time in a unique longitudinal collection of OED and oral cancer samples. She will also develop deep learning models to detect prognostic immune features from routine pathology images and investigate whether circulating immune signatures can offer a non-invasive way to monitor risk. Finally, she will use computational perturbation approaches to identify microenvironmental factors capable of restoring productive CD8^{+} T cell responses in high-risk lesions.

Dr. Qiu brings extensive experience in cancer immunotherapy and multimodal genomics, including influential work showing how chronic inflammation imprints lasting, resistance-associated epigenetic states—a process known as inflammatory memory. By defining how immune surveillance unfolds in pre-malignancy and pinpointing when and why it breaks down, her work aims to deliver clinically actionable biomarkers and reveal new opportunities for immune-based early intervention—providing a path to detect and intercept cancer before it begins.

CRI Immuno-Informatics Postdoctoral Fellow

Jiayu Ye, PhD

Stanford University

Leveraging Multimodal Transcriptomics to Uncover the Role of Mesothelial Cells in Ovarian Cancer Progression and Immunosurveillance

Sponsor:
Ansuman Satpathy, MD, PhD

Ovarian cancer remains one of the deadliest cancers for women, in part because many tumors are "immune silent" —they exclude or suppress immune cells that could help control disease or respond to immunotherapy. Increasing evidence shows that stromal cells surrounding the tumor strongly influence whether immune cells can enter, survive, and function. Dr. Jiayu Ye is focused on a key unanswered question: How do mesothelial cells—the barrier cells lining the peritoneal cavity—shape immune responses in ovarian cancer, and can target them restore anti-tumor Immunity?

Dr. Ye's preliminary research points to mesothelial cells as powerful but underappreciated regulators of the ovarian cancer immune microenvironment. Using single-cell RNA sequencing, she found that cancer-associated mesothelial cells communicate extensively with T cells, macrophages, and dendritic cells, and adopt immune-regulatory states that differ by location—ovary, peritoneum, and omentum. These cells also undergo mesothelial-to-mesenchymal transition, potentially giving rise to antigen-presenting cancer-associated fibroblasts known to influence T cell activity and lymphoid structure formation. Her project will define how mesothelial cells evolve during tumor progression and metastasis using longitudinal single-cell and spatial transcriptomics. She will then apply in vivo CRISPR-based screens to uncover genes that control their immune-modulatory behavior and test whether targeting these pathways can improve immune infiltration and slow disease.

Dr. Ye brings deep expertise in stromal biology, single-cell genomics, and tumor immunology, including her discovery of senescent fibroblasts as drivers of breast cancer immunosuppression. By uncovering how mesothelial cells act as immunological gatekeepers in ovarian cancer, her work could reveal new stromal targets to overcome immune exclusion and enhance patient responses to immunotherapy—helping shift historically immune-silent tumors toward more treatable, immunologically engaged states.

About CRI

The Cancer Research Institute (CRI) is a nonprofit organization dedicated to advancing the field of cancer immunotherapy through rigorous scientific research and global collaboration. Since 1953, CRI has been instrumental in uncovering the fundamental biology of the immune system and its application to cancer treatment, laying the groundwork for breakthroughs such as checkpoint blockade, cancer vaccines, and engineered cell therapies.

CRI's mission is to create a world immune to cancer by driving scientific discovery, accelerating collaboration, and turning breakthroughs into life-saving treatments. Our work bridges the gap between discovery and patient impact, ensuring that scientific innovation translates into real-world treatments.

To date, CRI has committed over $560 million to research impacting more than 30 cancer types. Our funding strategy is built on the framework of People × Biology × Data: supporting world-class scientists, deepening understanding of tumor–immune system interactions, and harnessing data to guide discovery and translation. By uniting these elements, CRI catalyzes innovation through our global research ecosystem to drive the next generation of discoveries forward.

CRI Cancer Research Institute™

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