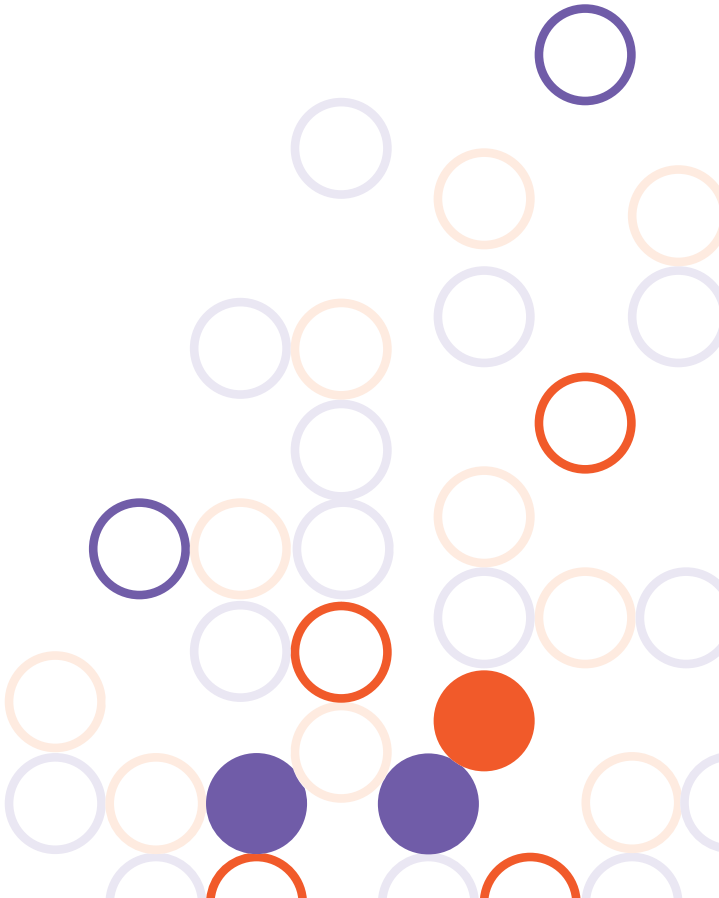
CRI Cancer Research Institute™

FALL 2025 & SPRING 2026 CRI
IRVINGTON AND IMMUNO-INFORMATICS

Postdoctoral Fellows



Uniting for a World Immune to Cancer

About 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 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 nearly $200 million invested to date, CRI selects new fellows twice a year – and in response to recent disruptions in federal science funding, committed an additional $2.5 million above CRI's annual commitment to fund 10 new fellowships over this year. The profiles that follow introduce the Winter 2025 and Spring 2026 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.

We are honored to share the Winter 2025 and Spring 2026 CRI Irvington and Immuno-Informatics Postdoctoral Fellows. These outstanding scientists are advancing pioneering work across tumor immunology, computational biology, and cellular engineering. Their research meets 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 and Spring 2026 CRI Postdoctoral Fellows and the hope they carry forward.

With gratitude,

Alicia Zhou, PhD
Chief Executive Officer
Cancer Research Institute

CRI Irvington Postdoctoral Fellows

Insights from CRI's Scientific Advisory Council

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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-Fall 2025

Emmanuela Adjei-Sowah, PhD

Vanderbilt University

Mentor(s): John Wilson, PhD

Engineering lipid nanoparticles for targeted activation of RIG-I to potentiate antitumor immunity in renal cell carcinoma

Relevance: Renal Cell Carcinoma

Project Summary:

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-Spring 2026

Mariano Aufiero, PhD

Massachusetts General Hospital

Mentor(s): Eric Dang, PhD

Defining and harnessing Dectin-1-mediated signaling in tumor immunity

Relevance: Melanoma

Project Summary:

Many cancers avoid destruction by manipulating the cells that surround them. One important group of these immune cells, called macrophages, can either help destroy tumors or, in some cases, unintentionally support tumor growth. Scientists still do not fully understand how to reprogram macrophages, so they consistently fight cancer effectively, especially in tumors that resist current immunotherapies.

Dr. Aufiero's research focuses on a molecule called Dectin-1, a receptor found on macrophages that helps these immune cells recognize threats and activate anti-tumor responses. His earlier work uncovered important mechanisms of immune defense against dangerous fungal infections in cancer patients and challenged long-standing assumptions about how immune cells kill pathogens. He is now applying this expertise in innate immunity to cancer research. This project will investigate two distinct forms of Dectin-1 and determine how they influence macrophage behavior inside tumors. Dr. Aufiero will study how these receptors control communication between macrophages and other immune cells and how they affect the immune system's ability to recognize and attack cancer.

The goal is to use this knowledge to improve engineered macrophage therapies. Early "CAR-macrophage" treatments have shown promise but have not yet produced strong tumor shrinkage in patients. By designing more effective receptors inspired by Dectin-1 biology, this research could lead to macrophage-based immunotherapies that better recognize, engulf, and stimulate immune attacks against cancers that currently evade treatment.

CRI Irvington Postdoctoral Fellow-Spring 2026

Po-Ta Chen, PhD

Boston Children's Hospital

Mentor(s): Taekjip Ha, PhD, Jared Rowe, PhD

Dissecting epigenetic maintenance in exhausted T cells by multimodal single-cell imaging and locus-proximal proteomics to identify targets for exhaustion reversal

Relevance: Hodgkin Lymphoma, Lung Cancer, Melanoma

Project Summary:

Cancer immunotherapy can produce remarkable results, but many patients eventually stop responding because their immune cells become "exhausted." These exhausted T cells lose their ability to attack tumors effectively, even after treatment with powerful therapies such as PD-1 checkpoint inhibitors. Scientists still do not fully understand why this exhausted state becomes so difficult to reverse.

Dr. Chen's research combines cutting-edge imaging technology with cancer immunology to investigate the physical organization of DNA inside exhausted T cells. During his doctoral training at Princeton University, he built advanced microscopy systems capable of visualizing gene activity in living cells with extraordinary precision. More recently, he developed a powerful new imaging platform called GOLDFISH-X, which allows scientists to simultaneously study DNA structure, gene activity, and protein interactions within individual cells.

Using this technology, Dr. Chen will examine whether exhaustion-related genes become locked into stable three-dimensional DNA structures that prevent T cells from regaining normal function. He will identify the proteins that maintain these structures and test whether disrupting them can restore long-term T cell activity in tumors. This work could reveal entirely new drug targets for improving immunotherapy. By identifying the molecular "anchors" that stabilize T cell exhaustion, Dr. Chen's research may help scientists design combination therapies that allow more patients to achieve durable responses to cancer treatment.

CRI Irvington Postdoctoral Fellow-Spring 2026

Agnieszka Chryplewicz, PhD

Memorial Sloan Kettering Cancer Center

Mentor(s): Charles Sawyers, PhD

Identifying oncogenic signals that drive aberrant myelopoiesis and systemic immunosuppression in prostate cancer

Relevance: Prostate Cancer

Project Summary:

Advanced prostate cancer remains one of the deadliest cancers in men, and current immunotherapies have shown only limited success. One reason is that tumors do not act alone: they reshape the body's immune system both locally and throughout the body, creating conditions that suppress immune responses and help cancer spread.

Dr. Chryplewicz has spent her career studying how tumors manipulate immune cells. During her PhD work in Switzerland, she helped discover innovative strategies that turned treatment-resistant tumors into ones responsive to immunotherapy, leading to a clinical trial in patients. Her research has also uncovered entirely new molecules involved in immune suppression and helped launch a biotechnology company focused on cancer therapies. In this project, Dr. Chryplewicz will investigate how prostate tumors communicate with distant organs such as bone marrow to influence the production and behavior of immune cells. Using advanced mouse models, organoid systems, and single-cell technologies, she will trace how immune-suppressive cells develop and migrate into tumors.

By uncovering the signals that allow tumors to weaken the immune system, her research could identify new strategies to block immune suppression and make immunotherapies more effective for prostate cancer patients. Her findings may also reveal broader principles of how cancers reshape the immune system throughout the body.

CRI Irvington Postdoctoral Fellow-Fall 2025

Maria Cardenas Conti, PhD

University of Pennsylvania

Mentor(s): E. John Wherry, PhD

Intrinsic and extrinsic regulation of stem-like CD4 T cell differentiation in cancer

Relevance: Broad Cancer Relevance

Project Summary:

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-Fall 2025

Xueyang Dong, PhD

Harvard University

Mentor(s): Kazuki Nagashima, MD, PhD

Systematic discovery of microbiome-derived T cell antigens for colon cancer immunotherapy

Relevance: Colon Cancer

Project Summary:

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-Fall 2025

Ron Gejman, MD, PhD

Columbia University in the City of New York

Mentor(s): Benjamin Izar, MD, PhD

Precision base-edited T cell therapy in transplant recipients with cutaneous squamous cell carcinoma

Relevance: Squamous Cell Carcinoma

Project Summary:

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-Spring 2026

Vassilis Glaros, PhD

The Rockefeller University

Mentor(s): Gabriel Victora, PhD

Towards identification of cancer-specific fingerprints in B cell repertoires

Relevance: Breast Cancer, Colorectal Cancer, Lung Cancer, Pancreatic Cancer

Project Summary:

B cells and the antibodies they produce are increasingly recognized as important players in cancer immunity. In several cancers, patients whose tumors contain high numbers of B cells respond better to immunotherapy and have improved survival. However, scientists still do not know exactly what these B cell responses are recognizing or whether consistent immune "fingerprints" exist across patients.

Dr. Glaros has spent his career studying how B cells develop, diversify, and form long-lasting immune memory. His earlier research challenged long-standing assumptions about how memory B cells arise and established him as a leading young investigator in fundamental B cell biology.

One of the biggest challenges in studying B cells is their enormous diversity: every person's B cells are slightly different, making it difficult to identify meaningful patterns. To overcome this obstacle, Dr. Glaros will use a novel experimental system in which B cell diversity is intentionally simplified and more reproducible. Using this approach, he will compare immune responses across different tumor settings to determine whether cancers produce recognizable B cell signatures. The goal is to identify immune fingerprints that could improve cancer monitoring, help classify disease more accurately, and deepen understanding of how the immune system responds to tumors.

Dr. Glaros' research could ultimately lead to new ways of predicting disease progression and tailoring immunotherapy based on a patient's unique immune response.

CRI Irvington Postdoctoral Fellow-Spring 2026

L. Benjamin Hills, MD, PhD

University of California, San Diego;
La Jolla Institute for Immunology;
Rady's Children's Hospital

Mentor(s): Shane Crotty, MD, PhD, Hojun Li, MD, PhD

Beyond blood: How cancer therapy reshapes adaptive immunity across tissues

Relevance: Hodgkin Lymphoma, Leukemia in Children, Non-Hodgkin Lymphoma in Children, Soft Tissue Sarcoma

Project Summary:

Cancer treatments such as chemotherapy and immunotherapy save lives but can also severely weaken the immune system, leaving patients vulnerable to dangerous infections. Doctors typically monitor immune health using blood tests, but most long-term protective immune cells live in tissues and lymphoid organs throughout the body, where their response to cancer therapy remains poorly understood.

Dr. Hills is a pediatric hematology-oncology physician-scientist with expertise in human immunology and advanced immune profiling. His earlier research helped pioneer methods for sequencing individual human neurons and later uncovered important mechanisms controlling immune memory formation.

His project will provide one of the first comprehensive studies of how cancer therapies affect protective immune cells throughout the body, not just in the bloodstream. Using minimally invasive nasal swabs alongside blood and bone marrow samples collected during routine care, Dr. Hills will track how T and B cells change during and after treatment.

His research aims to determine which immune cell populations are lost, which survive, and how effectively the immune system recovers over time. These findings could directly improve patient care by helping doctors better predict infection risk, optimize vaccination timing, and personalize supportive treatments. As more cancer patients survive long term, understanding how therapies affect immune health will become increasingly important for ensuring survivors can live healthier lives after treatment.

CRI Irvington Postdoctoral Fellow-Fall 2025

Zhouping Hong, PhD

Boston Children's Hospital

Mentor(s): Hao Wu, PhD

Investigating the NLRP3 and NLRP1 super-inflammasome

Relevance: Broad Cancer Relevance

Project Summary:

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-Fall 2025

Fubo Ji, PhD

The University of Texas Southwestern Medical Center

Mentor(s): Javier Garcia Bermudez, PhD

Dissecting the role of lipid uptake in cancer immune evasion

Relevance: Broad Cancer Relevance

Project Summary:

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 affects 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-Spring 2026

Minwoo Kang, PhD

Stanford University

Mentor(s): Hawa Racine Thiam, PhD

Hijacking an innate immune enzyme: tumor-intrinsic PAD4 remodels nuclear mechanics to promote breast cancer invasion

Relevance: Breast Cancer

Project Summary:

Most cancer deaths occur not because of the original tumor, but because cancer spreads throughout the body. To spread, cancer cells must squeeze through extremely tight spaces in tissues and blood vessels. One major obstacle is the nucleus — the large, stiff structure that houses DNA — which can rupture when cells attempt to force their way through confined spaces.

Dr. Kang studies how physical forces and cellular mechanics influence cancer progression. His earlier work showed how tissue tension and the physical structure of the tumor environment regulate fibroblast behavior and tumor growth. He also developed advanced imaging and computational tools to analyze cell behavior in real time.

His research focuses on an enzyme called PAD4, which normally helps immune cells reorganize their DNA during inflammatory responses. Dr. Kang believes cancer cells may hijack this same mechanism to loosen and reshape their nuclei, making it easier for them to migrate and spread.

Using advanced imaging technologies and microengineered systems that mimic the tight spaces cancer cells encounter in the body, he will investigate how PAD4 changes nuclear structure and mechanics during metastasis. If successful, this work could identify PAD4 as a promising new target for therapies aimed at preventing cancer spread. More broadly, it may reveal entirely new ways that cancer cells exploit immune-related processes to become more invasive.

CRI Irvington Postdoctoral Fellow-Fall 2025

Joseph Kern, PhD

Dana-Farber Cancer Institute

Mentor(s): Judith Agudo, PhD

Uncovering mechanisms of immune evasion in intestinal tumorigenesis and regeneration

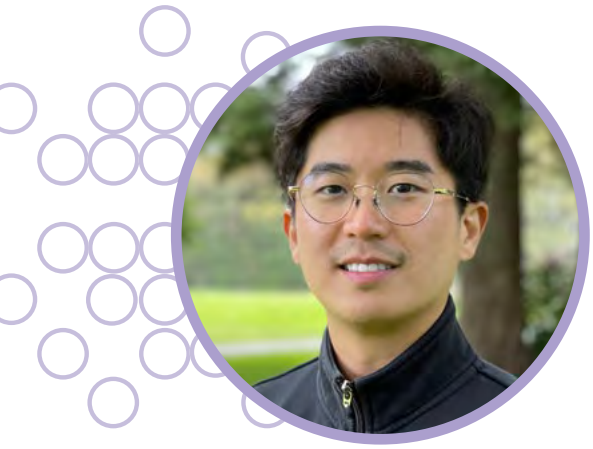
Relevance: Colorectal Cancer

Project Summary:

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-Fall 2025

Hyung Jun Kim, PhD

University of California, San Francisco

Mentor(s): Roarke Kamber, PhD

Systematic engineering of multifunctional macrophages for solid tumor immunotherapy

Relevance: Solid Tumors

Project Summary:

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-Spring 2026

Seoyeong Kim, PhD

NYU Grossman School of Medicine

Mentor(s): Dan Littman, PhD

The role of tolerizing dendritic cells in antigen-specific tolerance in the central nervous system and glioma immune evasion

Relevance: Brain and Spinal Cord Tumors

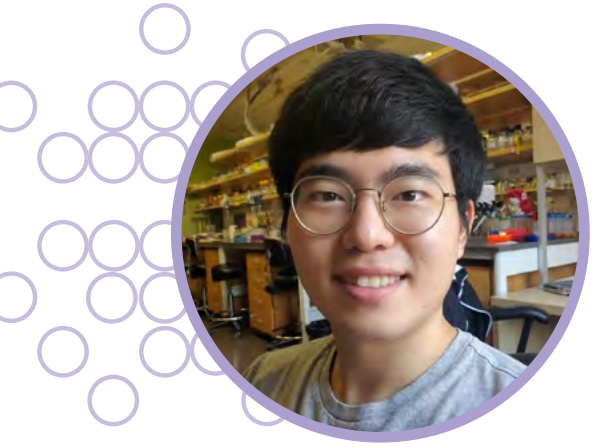
Project Summary:

The brain was once thought to be isolated from the immune system, but scientists now know that immune cells actively monitor and protect the brain. Unfortunately, brain tumors such as gliomas may exploit these protective mechanisms to suppress immune responses and evade attack.

Dr. Kim is uniquely trained at the intersection of neuroscience and immunology. His earlier work uncovered how specific synaptic molecules regulate brain function and behavior and provided new insights into neurodevelopmental disorders. He has now shifted his Relevance: toward understanding how immune cells regulate brain health and disease.

His fellowship centers on specialized immune cells called regulatory T cells, or Tregs, which help prevent harmful inflammation in the brain. Dr. Kim will investigate a recently discovered group of dendritic cells that may generate these Tregs in response to brain-related antigens. He believes that gliomas may hijack this normal immune-balancing process to create an environment that protects tumors from immune attack. Using advanced tumor models, he will determine whether blocking this pathway can improve anti-tumor immunity and enhance responses to immunotherapy.

This work could uncover a fundamental mechanism of immune regulation in the brain and identify new strategies for treating glioma while preserving the delicate immune balance required for healthy brain function.



CRI Irvington Postdoctoral Fellow-Fall 2025

Wantae Kim, PhD

The Scripps Research Institute

Mentor(s): Dorothee Kern, PhD

Molecular mechanisms by which THEMIS controls T-cell maturation

Relevance: Broad Cancer Relevance

Project Summary:

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-Fall 2025

Konrad Knopper, PhD

University of California, San Francisco

Mentor(s): Jason Cyster, PhD

A nociceptor neuron–macrophage–DC axis drives sexual dimorphic skin immunity

Relevance: Broad Cancer Relevance

Project Summary:

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-Fall 2025

Anna Kolarzyk, PhD

Cornell University

Mentor(s): Deborah Fowell, DPhil

Illuminating immune cell trafficking: spatiotemporal mapping of lymphocyte dynamics in pancreatic cancer

Relevance: Pancreatic Cancer

Project Summary:

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-Spring 2026

Jorden Lane, PhD

NYU Grossman School of Medicine

Mentor(s): Amanda Lund, PhD

Uncovering the role of lymphatic stromal IL-33 in the tumor draining lymph node

Relevance: Melanoma, Solid Tumors

Project Summary:

Cancer often spreads first to nearby lymph nodes, which are critical sites where the immune system normally detects threats and coordinates anti-tumor responses. However, tumors can disrupt the normal immune environment within these lymph nodes, allowing cancer to escape immune surveillance and spread further throughout the body.

Dr. Lane studies how the lymphatic system and immune tissues respond to inflammation and cancer. His earlier work uncovered previously unknown differences in intestinal lymphatic vessels and demonstrated how infection reshapes lymphatic structure and immune function. His project focuses on sentinel lymph nodes, the first lymph nodes that drain tumors such as melanoma. His mentor, Dr. Lund, and her team discovered that specialized lymphatic cells within these lymph nodes produce a molecule called IL-33, which acts as an immune alarm signal. In both patients and mouse models, tumors appear to disrupt this protective alarm system.

Dr. Lane will investigate how IL-33 helps maintain lymph node integrity and anti-tumor immunity and determine how tumors suppress this pathway to promote metastasis. Using advanced imaging and immune profiling technologies, he will identify the cells and signals involved in this process.

The findings could reveal entirely new strategies for strengthening immune surveillance in lymph nodes and preventing the spread of melanoma and potentially other solid tumors.

CRI Irvington Postdoctoral Fellow-Fall 2025

Georgia Lattanzi, PhD

Salk Institute

Mentor(s): Daniel Hollern, PhD

Deciphering the mechanisms of B cell-mediated epitope spreading in cancer

Relevance: Broad Cancer Relevance

Project Summary:

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. 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-Fall 2025

Yi-Tsang Lee, PhD

La Jolla Institute for Immunology

Mentor(s): Patrick Hogan, PhD

A novel approach to study T-cell activation and exhaustion

Relevance: Broad Cancer Relevance

Project Summary:

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-Fall 2025

Shi Li, PhD

Fred Hutchinson Cancer Center

Mentor(s): Cyrus Ghajar, PhD, Even Newell, PhD

Purinergic checkpoints governing microglial clearance of disseminated tumor cells

Relevance: Brain Cancer Metastasis

Project Summary:

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-Spring 2026

Yi Liu, PhD

University Health Network

Mentor(s): Tak W. Mak, PhD

Regulation of T cell exhaustion and anti-tumor immunity by Cholinergic receptor alpha 7

Relevance: Broad Cancer Relevance

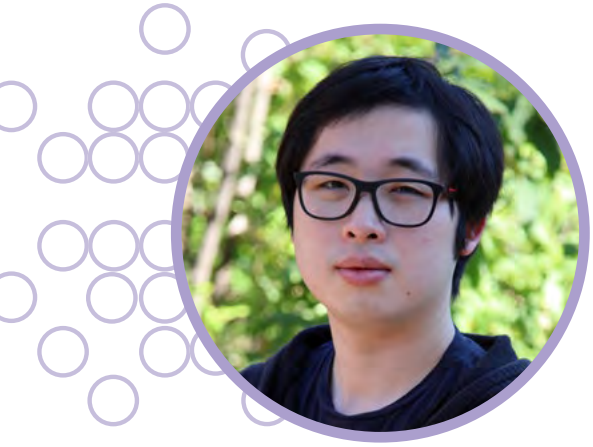
Project Summary:

Immunotherapy works by activating the body's own immune system to fight cancer, but many patients eventually stop responding because their T cells become "exhausted" and lose their ability to attack tumors effectively.

Dr. Liu studies the molecular signals that control T cell function inside tumors. His previous work at leading research Institution:s in China uncovered important mechanisms regulating immune exhaustion and cancer progression, resulting in publications in major scientific journals including Nature, Cancer Cell, and Nature Immunology.

This project investigates a molecule called Chrna7 (Cholinergic receptor alpha 7), a receptor found on T cells that appears to promote T cell exhaustion. Dr. Liu's early studies show that mice lacking this receptor develop stronger anti-tumor immune responses and experience slower tumor growth.

Using advanced genetic and molecular approaches, he will determine how Chrna7 weakens T cell function and whether blocking this pathway can restore the ability of T cells to fight cancer. The long-term goal is to identify new therapeutic strategies that keep T cells active longer and improve the effectiveness of immunotherapy. If successful, this work could help overcome one of the major barriers limiting current cancer immunotherapies.



CRI Irvington Postdoctoral Fellow-Fall 2025

Zhiyuan Mao, PhD

University of California, Los Angeles

Mentor(s): John Lee, MD, PhD

Deciphering heterogeneous tumor responses to immunotherapy by profiling cancer–T cell doublet interactions

Relevance: Prostate, Ovarian and Pancreatic Cancers

Project Summary:

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-Fall 2025

Andrea Muñoz Zamora, PhD

Icahn School of Medicine at Mount Sinai

Mentor(s): Michel Enamorado, PhD

In search of immune engrams: Mapping the brain's memory of systemic inflammation

Relevance: Broad Cancer Relevance

Project Summary:

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-Spring 2026

Hyunjin Noh, PhD

The University of Texas Southwestern Medical Center

Mentor(s): Ayaz Najafov, PhD

Role of SIGLEC12-mediated plasma membrane rupture in anti-tumor immunity

Relevance: Broad Cancer Relevance

Project Summary:

When cancer cells die, they can sometimes trigger powerful immune responses by releasing "danger signals" that alert the immune system to attack the tumor. One form of inflammatory cell death, called necroptosis, is especially important because it can help turn dying cancer cells into a signal that activates anti-tumor immunity.

Dr. Noh studies the molecular machinery that controls necroptosis. In earlier work, Dr. Noh uncovered previously unknown roles for key necroptosis proteins in chronic inflammatory diseases and identified SIGLEC12 as a critical regulator of the final membrane rupture step that allows immune-stimulating signals to escape dying cells.

In this fellowship, Dr. Noh will investigate how mutations in SIGLEC12 found in certain cancers interfere with this process. Dr. Noh believes that when tumors block proper membrane rupture, they may prevent the immune system from fully recognizing and attacking cancer cells. Using tumor models and patient-derived samples, this work will examine whether cancers carrying mutant SIGLEC12 show weaker immune activation and reduced immune cell infiltration.

This research promises to reveal why some tumors fail to respond to immunotherapy and identify new opportunities to strengthen immune responses by restoring immunogenic cancer cell death.

CRI Irvington Postdoctoral Fellow-Spring 2026

Celeste Parra Bravo, PhD

Dartmouth College

Mentor(s): Pamela Rosato, PhD

Defining compartmental control of CNS TRM immunity in leptomeningeal metastasis

Relevance: Central Nervous System Cancers

Project Summary:

Leptomeningeal metastasis is a devastating complication of cancer in which tumor cells spread into the fluid and membranes surrounding the brain and spinal cord. Most patients with this condition have very limited treatment options because the brain's unique immune environment makes it difficult for the immune system to mount effective anti-cancer responses.

Dr. Parra Bravo combines expertise in neuroscience, stem cell biology, and immunology to study how immune cells function within the brain. Her earlier research identified genetic drivers of neurodegenerative disease and helped develop new CRISPR-based approaches for studying disease mechanisms in human neurons.

Her project focuses on tissue-resident memory T cells, or TRM, specialized immune cells that permanently reside within tissues and provide rapid local protection. Dr. Parra Bravo believes that leptomeningeal metastases suppress these resident immune cells and weaken the brain's natural defenses against cancer. Using advanced imaging, mouse models, and high-throughput CRISPR screening, she will map where TRM cells reside in the brain, determine how they control tumor spread, and identify the genetic "switches" that regulate their activity.

The goal is to develop new strategies that harness these naturally occurring immune cells to improve immune surveillance and create more effective treatments for cancers that spread to the central nervous system.

CRI Irvington Postdoctoral Fellow-Spring 2026

Mariana Pereira da Costa, PhD

Institut Pasteur

Mentor(s): Yasmine Belkaid, PhD

Endogenous retroelements in infection-driven cancer immunosurveillance

Relevance: Lung Cancer

Project Summary:

Our immune systems constantly respond to viral infections throughout life, but scientists still do not fully understand how these past infections influence the body's ability to recognize and fight cancer later on.

Dr. Pereira da Costa studies how infections reshape immune responses in tissues such as the lung. Her earlier work demonstrated that viral infections can dramatically reorganize dendritic cell networks and influence the strength and long-term durability of T cell immunity.

Her project focuses on endogenous retroelements (EREs), ancient remnants of viral DNA that make up nearly 40% of the human genome. Although these sequences are usually inactive, they can become reactivated during infections and in many cancers, producing viral-like proteins that may be recognized by immune cells.

Dr. Pereira da Costa proposes that viral infections may "train" T cells to recognize these ERE-derived proteins, creating long-lasting immune memory that later helps the body detect tumors expressing the same targets. Using mouse models of respiratory infection and lung cancer, she will investigate how these ERE-specific immune responses develop, how long they persist, and whether they improve tumor control or responses to immunotherapy. This work could uncover a previously unrecognized connection between infection history and cancer immunity and open new avenues for developing immune-based cancer therapies.

CRI Irvington Postdoctoral Fellow-Spring 2026

Megan Priestley, PhD

Massachusetts Institute of Technology

Mentor(s): Jessica Stark, PhD

Targeting glycans as a new frontier for colorectal cancer immunotherapy

Relevance: Colorectal Cancer

Project Summary:

Colorectal cancer is the second deadliest cancer worldwide, and rates are rising alarmingly among younger adults. Although immunotherapy has transformed treatment for some cancers, more than 90% of colorectal cancer patients do not benefit from currently available immune-based therapies.

Dr. Priestley studies glycans — specialized sugar molecules that coat the surface of cells. Cancer cells often exploit glycans to hide from the immune system and promote tumor growth. During her PhD, she discovered important new mechanisms controlling immune cell migration and received multiple national and international awards for her work.

More recently, Dr. Priestley helped develop a promising new type of immunotherapy called "AbLecs," engineered molecules that simultaneously target cancer cells and the immunosuppressive glycans that protect them. These therapies have already shown strong anti-cancer activity in laboratory and animal studies. In this project, she will investigate how glycans contribute to the earliest stages of colorectal cancer development and use this knowledge to design new AbLec therapies specifically for colorectal tumors.

The goal is to create more effective immunotherapies for the large majority of colorectal cancer patients who currently lack good treatment options and to better understand how cancers exploit glycans to evade immune attack.

CRI Irvington Postdoctoral Fellow-Spring 2026

Nataliya Prokhnevska, PhD

Icahn School of Medicine at Mount Sinai

Mentor(s): Alice Kamphorst, PhD

Re-activation of CD4 T cells in tumors to promote immunotherapy responses

Relevance: Liver Cancer

Project Summary:

Although immunotherapy has revolutionized cancer treatment, many patients still do not respond because their immune systems fail to mount strong enough anti-tumor responses. Most current immunotherapies focus primarily on activating CD8 "killer" T cells, while largely overlooking the critical supporting role of CD4 helper T cells.

Dr. Prokhnevska has spent her career studying how immune cells are activated within tumors and lymph nodes. Her earlier research identified important stem-like T cell populations that help sustain anti-tumor immunity and contributed to major discoveries in kidney, prostate, and liver cancer immunology.

Her research seeks to improve immunotherapy by activating helper CD4 T cells alongside existing treatments. Rather than relying on highly personalized tumor-specific targets, Dr. Prokhnevska proposes using memory CD4 T cells generated from common infections and vaccinations that already exist in most patients.

She will test novel antibody-based therapies designed to bring these helper T cells together with dendritic cells, the immune cells responsible for coordinating anti-cancer responses. The project will also determine how CD4 T cells strengthen CD8 T cell activity during immunotherapy. This work could lead to more broadly effective immunotherapies that harness the full power of the immune system and improve outcomes for patients who currently fail to benefit from existing treatments.

CRI Irvington Postdoctoral Fellow-Spring 2026

Jonah Rosas, PhD

Memorial Sloan Kettering Cancer Center

Mentor(s): Maria Akhmanova, PhD

Epithelial mechanics guide macrophage infiltration

Relevance: Solid Tumors

Project Summary:

One of the greatest obstacles facing immune cell therapies for solid tumors is that immune cells often cannot physically penetrate deep into dense tumor tissue. While most immunotherapy research focuses on improving how immune cells recognize cancer, far less is understood about the physical barriers that block immune infiltration.

Dr. Rosas studies the mechanics of how cells move and interact within tissues. During his doctoral work, he developed innovative three-dimensional tumor models and uncovered how physical forces alone can disrupt normal tissue structure and behavior. This project focuses on macrophages, immune cells capable of entering tumors and attacking cancer cells. Dr. Rosas aims to determine how the physical properties of tumor cells — including surface tension and cell-cell adhesion strength — influence the ability of macrophages to infiltrate tumors.

Using advanced live-cell imaging and next-generation RNA sequencing, he will measure how macrophages respond to different epithelial surface properties and identify the adhesion molecules that control these interactions. The findings could reveal entirely new ways to improve immune cell infiltration into solid tumors and enhance the effectiveness of future cell-based immunotherapies.

CRI Irvington Postdoctoral Fellow-Spring 2026

Ayse Sahan, PhD

The University of Chicago

Mentor(s): Chuan He, PhD

Mapping the chromatin-associated snoRNA–RNA interactome to define emerging transcriptional control in leukemia

Relevance: Acute Lymphocytic Leukemia

Project Summary:

Many aggressive blood cancers become resistant to immunotherapy by altering the activity of genes involved in immune recognition. However, scientists still have only a limited understanding of the hidden molecular mechanisms that regulate these immune-related genes.

Dr. Sahan's research combines cancer biology, engineering, and molecular signaling. Her previous work uncovered how physical properties of tissues influence cancer behavior and led to the development of advanced biosensors capable of tracking cellular metabolism and signaling with remarkable precision.

Her project investigates how a poorly understood class of RNA molecules controls immune-related gene expression in aggressive blood cancers. Dr. Sahan believes that interactions between RNAs and other molecules inside cancer cells may contribute to immune evasion and resistance to treatment.

Using a new technology developed in the laboratory, she will generate a detailed map of RNA interactions across more than 20 types of blood cancer and identify molecular pathways that could be targeted therapeutically. This work could reveal an entirely new layer of cancer biology and help establish a future generation of RNA-based immunotherapies designed to overcome treatment resistance and restore anti-cancer immune responses.

CRI Irvington Postdoctoral Fellow-Spring 2026

Kaushik Sen, PhD

Weill Cornell Medicine

Mentor(s): Juan Cubillos-Ruiz, PhD

Harnessing nutrient stress adaptation in dendritic cells to enhance immunity against ovarian cancer

Relevance: Ovarian Cancer

Project Summary:

High-grade serous ovarian cancer is one of the deadliest forms of cancer because it often spreads rapidly, returns after treatment, and responds poorly to current immunotherapies. Scientists are increasingly interested in whether changing tumor metabolism — the way cancer cells use nutrients — could improve immune responses against these tumors.

Dr. Sen studies how metabolism shapes immune cell behavior. He previously uncovered important metabolic pathways that control the function of dendritic cells, key immune cells responsible for activating anti-tumor T cell responses.

His fellowship focuses on methionine, an essential dietary nutrient that ovarian cancer cells heavily depend on for growth. Dr. Sen's preliminary studies show that reducing methionine levels slows tumor growth while simultaneously improving dendritic cell function and anti-tumor immunity. He proposes that methionine restriction acts as a controlled stress signal that helps immune cells remain functional within the harsh tumor environment. Using dietary interventions, drug-based strategies, and advanced immune profiling, he will determine how methionine restriction reshapes immune responses and whether it improves the effectiveness of checkpoint immunotherapy.

This work could establish a strong scientific foundation for combining metabolic therapies with immunotherapy and ultimately lead to more effective treatments for ovarian cancer patients.

CRI Irvington Postdoctoral Fellow-Fall 2025

Qinli Sun, PhD

Stanford University

Mentor(s): K. Christopher Garcia, PhD

Therapeutic development of engineered TGF-β agonists for autoimmunity and cancer

Relevance: Broad Cancer Relevance

Project Summary:

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-□, which can either suppress or activate immune responses depending on the context. In cancer, TGF-□ 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-□ 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-□, 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-□ agonist platform that allows selective activation of TGF-□ signaling in specific immune cells. As a proof of concept, he engineered a combined IL-2-TGF-□ 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-□ into precise therapeutic tools. By reprogramming how immune cells interpret TGF-□ signals, his research could lead to safer, more effective treatments for autoimmune diseases, chronic inflammation, and cancer.

CRI Irvington Postdoctoral Fellow-Fall 2025

Lion Uhl, DPhil

Memorial Sloan Kettering Cancer Center

Mentor(s): Alexander Rudensky, PhD

Molecular mechanisms of fine-tuning of the regulatory T cell transcriptional and functional program by interleukin-2

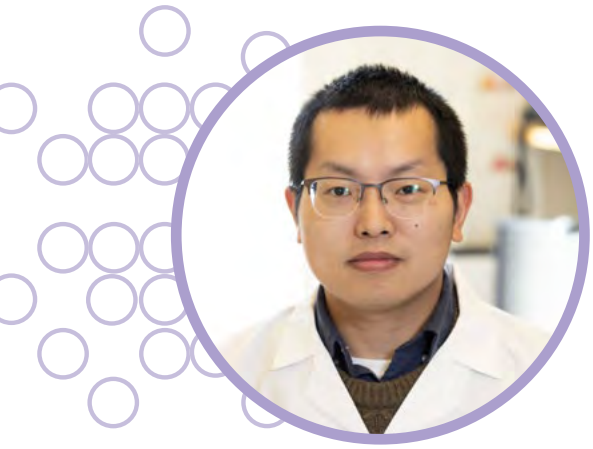
Relevance: Broad Cancer Relevance

Project Summary:

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-Fall 2025

Hao Wang, PhD

Dana-Farber Cancer Institute

Mentor(s): Kai Wucherpfennig, MD, PhD

Modulation of T cell–B cell crosstalk in tertiary lymphoid structures via the CD161–CLEC2D pathway

Relevance: Broad Cancer Relevance

Project Summary:

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-Fall 2025

Hejia Wang, MD, PhD

Johns Hopkins University

Mentor(s): Elizabeth Jaffee, MD

T-cell determinants of clinical response to a mutant KRAS vaccine in colorectal cancer

Relevance: Colorectal Cancer

Project Summary:

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-Fall 2025

Yebin Wang, PhD

Harvard Medical School

Mentor(s): Diane Mathis, PhD

Tolerization of goblet cell self-antigens by thymic mimetic cells

Relevance: Broad Cancer Relevance

Project Summary:

Immune checkpoint therapies have 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 Irvington Postdoctoral Fellow-Spring 2026

Qian Xue, PhD

Stanford University

Mentor(s): Rogelio Hernandez-Lopez, PhD

Mapping and engineering cell-intrinsic T cell infiltration programs for enhanced solid tumor immunotherapies

Relevance: Solid Tumors

Project Summary:

CAR T-cell therapy has produced remarkable success against blood cancers but has largely failed against solid tumors such as breast, lung, and colon cancer. One major reason is that engineered T cells cannot efficiently move through the dense tissue surrounding solid tumors.

Dr. Xue studies how cells migrate through complex physical environments. Her earlier research overturned long-standing assumptions about how cells move within living tissues and contributed to the development of CAR-macrophage approaches for solid tumors.

In this project, she will use CRISPR-based gene activation technology to systematically identify genes that improve the ability of T cells to infiltrate dense tumor tissue. Using advanced three-dimensional tumor models that closely mimic real cancers, she will screen thousands of genes to determine which ones enhance T cell movement.

Dr. Xue has already identified several promising genes that improve migration. She will now incorporate the best candidates into next-generation CAR T cells while also engineering built-in safety systems that limit activity to tumor environments. If successful, this work could overcome one of the biggest barriers facing CAR T-cell therapy and open a path toward making these treatments effective for solid tumors.

CRI Irvington Postdoctoral Fellow-Spring 2026

Chengzhi Yu, PhD

University of Massachusetts Chan Medical School

Mentor(s): Tianmin Fu, PhD

Decoding the RHIM signalosome in the ZBP1-RIPK3 pathway for anti-tumor immunity

Relevance: Solid Tumors

Project Summary:

Cancer cells often find ways to hide from the immune system, limiting the effectiveness of today's most promising immunotherapies. One powerful but poorly understood immune pathway centers on a protein called ZBP1, which acts as an internal alarm sensor. When activated, ZBP1 can trigger inflammation and a specialized form of cell death known as necroptosis. These responses may help the immune system recognize and attack tumors by releasing signals that expose cancer cells to immune surveillance. Yet scientists still do not understand how this pathway is switched on, how it is organized inside cells, or how viruses and other factors can shut it down.

Dr. Yu's research aims to uncover the molecular "assembly instructions" that govern ZBP1 signaling. Using cutting-edge structural biology, high-resolution imaging, and biochemical approaches, he will determine how ZBP1 and its partner protein, RIPK3, come together to form larger signaling complexes that control inflammatory cell death. The project will also investigate how a herpesvirus protein blocks this process, taking advantage of viral strategies that have evolved to evade immune defenses.

Dr. Yu is uniquely positioned to tackle this challenge. Throughout his training, he has built an impressive track record of revealing how immune systems are activated, regulated, and subverted across diverse biological systems. His discoveries—including a new anti-CRISPR immune evasion protein, a key regulatory mechanism in plant immunity, and the structural basis of a novel antiviral defense system—have been published in leading journals including Nature and Molecular Cell. By revealing how ZBP1 signaling is controlled at the molecular level, his work could identify new strategies to enhance anti-tumor immunity, overcome immune suppression, and improve patient responses to cancer immunotherapies such as checkpoint blockade.

CRI Immuno-Informatics Postdoctoral Fellows

Insights from CRI's Scientific Advisory Council

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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 Associate Director

Immuno-Informatics Postdoctoral Fellow-Fall 2025

Jose Almeida Santos, PhD

Dana-Farber Cancer Institute

Mentor(s): Jared Rowe, MD, PhD, David Liu, MD

Determining the role of latent neonatal T cells in immunity to cancer across the lifespan

Relevance: Broad Cancer Relevance

Project Summary:

One of the greatest barriers to successful cancer immunotherapy is T-cell exhaustion. Over time, immune cells inside tumors lose their ability to multiply, attack cancer cells, and sustain effective anti-tumor responses. This exhaustion limits the effectiveness of many of today's most promising cancer treatments. Dr. Jose Almeida Santos has spent his scientific career studying how the immune system develops and responds to disease. His work spans developmental biology, immunology, and computational analysis, and he has contributed discoveries ranging from novel gene-editing approaches to mechanisms that influence cancer immunotherapy outcomes. Now, as a postdoctoral fellow at Dana-Farber Cancer Institute, he is investigating a particularly intriguing immune population: neonatal CD8 T cells. Unlike most adult T cells, neonatal CD8 T cells appear naturally resistant to exhaustion. They proliferate more effectively, produce stronger immune responses, and retain their ability to function under challenging conditions. Yet their role in human cancers remains largely unexplored. Dr. Santos will combine analyses of human tumor datasets with innovative mouse studies to determine whether neonatal-like T cells are present in tumors and to understand how they interact with other cells in the tumor microenvironment. His goal is to identify the molecular pathways that allow these cells to resist exhaustion and maintain potent anti-cancer activity. By uncovering the biological secrets of these uniquely resilient immune cells, this research could reveal new ways to strengthen existing immunotherapies, improve patient responses, and inspire the next generation of cancer treatments.

Immuno-Informatics Postdoctoral Fellow-Spring 2026

Joshua Bromley, PhD

Yale University

Mentor(s): Noah Palm, PhD, Akiko Iwasaki, PhD

Shaping commensal evolution in colorectal cancer through mucosal immunity

Relevance: Colorectal Cancer

Project Summary:

Colorectal cancer is one of the leading causes of cancer death worldwide, and growing evidence suggests that bacteria living in the gut may play an important role in driving the disease. Some potentially harmful bacteria are found even in healthy individuals, but scientists still do not understand why these microbes sometimes become dangerous or how the immune system shapes their behavior.

Dr. Bromley studies how the immune system interacts with microbes at mucosal surfaces such as the gut. His earlier work used advanced single-cell technologies to reveal how immune responses change during infections such as COVID-19 and tuberculosis, uncovering important mechanisms that determine protection versus disease. This project focuses on two bacterial species linked to colorectal cancer: Fusobacterium nucleatum and Akkermansia muciniphila. Dr. Bromley will investigate how antibodies and inflammation within the gut influence the evolution and behavior of these microbes. One particularly innovative aspect of the project involves developing a vaccine designed to target Fusobacterium nucleatum. The goal is to generate antibodies that trap the bacteria in a weakened state, preventing them from promoting tumor growth.

This work could reveal fundamental new insights into how the immune system shapes the microbiome and how harmful bacteria contribute to colorectal cancer. Ultimately, the findings may open the door to entirely new prevention and treatment strategies that harness the immune system to control cancer-promoting microbes.

Immuno-Informatics Postdoctoral Fellow-Fall 2025

Sui Yuk (Candace) Chan, PhD

University of Texas at Austin

Mentor(s): B.J. Kim, PhD, Ilias Georgakopoulos-Soares, PhD

Liquid biopsy neomer profiling for glioblastoma cancer detection and precise immunotherapy

Relevance: Glioblastoma

Project Summary:

Glioblastoma is the deadliest form of brain cancer, and most patients survive only about 15 months after diagnosis. Because tumors are typically discovered only after symptoms appear, opportunities for effective treatment are limited. Current approaches to diagnosing and characterizing these tumors often require invasive brain biopsies, creating additional risks for patients. Dr. Candace Chan is uniquely positioned to tackle this challenge. A computational biologist with expertise spanning cancer genomics, immunology, and precision medicine, she has already helped develop innovative liquid biopsy technologies that detect cancer-specific DNA sequences in blood. Her previous work identified novel genomic signatures called "neomers," which are absent from healthy tissue but present in tumors, and demonstrated their potential for early cancer detection across multiple cancer types. Building on these advances, Dr. Chan proposes a blood-based approach to detect glioblastoma and guide treatment using the same tumor-specific signatures. By analyzing fragments of DNA and RNA shed by tumors into the bloodstream, her team aims to identify glioblastoma earlier and without invasive procedures. Importantly, these signatures could also reveal targets for personalized cancer vaccines tailored to each patient's tumor, potentially eliminating the need for surgical biopsies. If successful, this work could transform brain cancer care by enabling earlier diagnosis, reducing reliance on invasive procedures, and accelerating access to personalized immunotherapies. Ultimately, it could improve survival and quality of life for patients facing one of the most devastating forms of cancer.

Immuno-Informatics Postdoctoral Fellow-Spring 2026

Helen Huang, PhD

University of Washington

Mentor(s): Venu Pillarisetty, MD

Interpretable virtual tumor to simulate TIME remodeling in PDAC during chemo-immunotherapy

Relevance: Pancreatic Cancer

Project Summary:

Pancreatic cancer remains one of the deadliest cancers, and most patients do not benefit from current immunotherapies. One major reason is that pancreatic tumors create a dense, scar-like environment that physically blocks immune cells from entering the tumor and suppresses their activity.

Dr. Huang develops computational models that connect molecular biology with tissue-level immune behavior. Her earlier work combined genetics, systems immunology, and mathematical modeling to understand how immune cells make decisions and respond to disease. This project will study how the tumor environment changes in pancreatic cancer patients receiving a combination of chemotherapy, immunotherapy, and a drug designed to help immune cells enter tumors. Using advanced imaging and molecular profiling, Dr. Huang will analyze tumor samples collected before and during treatment to map where immune cells are located and how they interact with cancer cells.

She will then use artificial intelligence to identify tissue patterns that predict whether a patient is likely to respond to therapy. Finally, Dr. Huang will create personalized "virtual tumor" simulations that can safely test different treatment combinations and dosing strategies. If successful, this work could help doctors better predict which patients will benefit from immunotherapy, reduce exposure to ineffective treatments, and accelerate development of more personalized therapies for pancreatic cancer.

Immuno-Informatics Postdoctoral Fellow-Fall 2025

Baolin Liu, PhD

Broad Institute of MIT and Harvard

Mentor(s): Nir Hacohen, PhD, Caroline Uhler, PhD

Dissecting causal regulators of response and resistance to checkpoint immunotherapy in human tumors

Relevance: Broad Cancer Relevance

Project Summary:

Immune checkpoint inhibitors have transformed cancer treatment, producing remarkable and sometimes long-lasting responses. Yet many patients do not benefit from these therapies, and researchers still do not fully understand why. A major challenge is distinguishing genes that merely correlate with treatment outcomes from those that cause response or resistance. Dr. Baolin Liu has built an impressive track record at the forefront of cancer immunology and computational biology. During his doctoral training, he developed innovative single-cell analysis tools and helped identify the tumor-reactive immune cells that drive responses to checkpoint blockade across multiple cancer types. His work demonstrated that specific immune signatures could predict immunotherapy responsiveness with remarkable accuracy and was published in leading journals including Nature Cancer. As a postdoctoral researcher, Dr. Liu is taking the next step: moving beyond observation to uncover causation. His project will integrate single-cell sequencing data from melanoma patients with large-scale functional screening, curated biological pathways, and advanced machine-learning approaches. This powerful framework will identify which genes actively control anti-tumor immune responses and determine how they influence treatment outcomes. The resulting discoveries will be tested directly in human tumor-derived cells, providing critical validation and deeper mechanistic insight. By identifying the true drivers of immunotherapy resistance, Dr. Liu hopes to reveal new therapeutic targets and strategies that could help more patients benefit from these life-saving treatments. Ultimately, this work could accelerate the development of more precise and effective immunotherapies, bringing the promise of cancer immunotherapy to a much larger patient population.

Immuno-Informatics Postdoctoral Fellow-Spring 2026

Joseph Palmeri, PhD

Memorial Sloan Kettering Cancer Center

Mentor(s): Caleb Lareau, PhD

Interrogating the immunogenicity of de novo AI-designed protein minibinder therapeutics

Relevance: Broad Cancer Relevance

Project Summary:

Artificial intelligence is rapidly transforming cancer drug development by enabling scientists to design entirely new protein-based therapies. Among the most promising are tiny engineered proteins called "minibinders," which can direct the immune system to recognize and destroy tumors. However, a major unanswered question remains: will the human immune system mistakenly recognize these AI-designed proteins as foreign and reject them?

Dr. Palmeri is an immunoengineer focused on developing next-generation cancer immunotherapies. During his doctoral work at MIT, he engineered innovative antibody therapies that remained anchored within tumors, improving anti-cancer activity while reducing dangerous side effects. His research also uncovered important mechanisms controlling T cell activation inside tumors.

In his research, he will investigate the safety of AI-designed protein therapies by studying what makes them immunogenic — capable of triggering unwanted immune reactions. Using computational protein design, cell culture systems, and mouse models, Dr. Palmeri will engineer variants of AI-designed proteins containing different immune-triggering features and determine how their structural stability influences immune recognition.

The goal is to uncover the rules that determine whether the body views these proteins as safe or foreign. This knowledge could help scientists design future AI-generated cancer therapies that are both highly effective and "immunologically silent," reducing side effects while improving long-term treatment success. As AI-designed medicines move closer to clinical use, this work could play a critical role in ensuring their safe introduction into patients.

Immuno-Informatics Postdoctoral Fellow-Spring 2026

Sean Paulsen, PhD

Memorial Sloan Kettering Cancer Center

Mentor(s): Roni Shouval, PhD

A scalable image-based platform for real-time monitoring of cellular immunotherapy

Relevance: Blood Cancer, Leukemia, Lymphoma, Multiple Myeloma

Project Summary:

CAR T-cell therapy has transformed treatment for certain blood cancers, producing long-lasting remissions for some patients. However, not all patients respond successfully, and others develop serious immune-related side effects. Doctors currently lack simple and widely accessible tools to monitor how a patient's immune system is behaving in the critical days and weeks after treatment.

Dr. Paulsen combines expertise in artificial intelligence, digital imaging, and immunology to tackle this problem. During his doctoral training in computer science, he developed advanced machine-learning systems capable of extracting meaningful biological patterns from complex data such as brain imaging. He has since applied these computational tools to cancer immunotherapy at Memorial Sloan Kettering Cancer Center.

His project focuses on routine blood smears, a simple laboratory test performed worldwide. Although these smears contain valuable visual information about immune cells, they are traditionally interpreted only qualitatively by specialists. Dr. Paulsen has already developed AI systems capable of analyzing hundreds of thousands of immune cells from blood smears of CAR T-cell patients and linking specific cell patterns to treatment response and survival.

He will now expand this work to create a practical monitoring tool that can track immune activity during CAR T-cell therapy in real time. By identifying visual signatures associated with treatment success or dangerous side effects, this research could help doctors improve risk assessment, personalize supportive care, and enhance patient outcomes. The approach may also be broadly useful for monitoring many other forms of cancer immunotherapy.

Immuno-Informatics Postdoctoral Fellow-Fall 2025

Jingya Qiu, PhD

Gladstone Institutes

Mentor(s): Matthew Spitzer, PhD, Karin Pelka, PhD

Defining, monitoring, and intercepting pre-malignant immune hubs

Relevance: Oral Cancer

Project Summary:

Early detection saves lives, yet clinicians still struggle to identify which precancerous oral lesions will progress to cancer and which will remain harmless. As a result, patients often face a difficult choice between surgery that can cause significant complications or watchful waiting that risks missing the window for early intervention. Dr. Jingya Qiu has devoted her career to understanding how the immune system influences cancer development and treatment outcomes. During her PhD at the University of Pennsylvania, she made important discoveries about how chronic inflammation helps tumors resist immunotherapy, leading to high-impact publications and multiple research awards. More recently, she helped lead a landmark study of patients with oral precancerous lesions, uncovering immune signatures that predict progression to cancer years before tumors appear. Her proposed research builds on this discovery.

Dr. Qiu has identified a population of CD8 T cells—immune cells normally responsible for eliminating abnormal cells—that appear dysfunctional in lesions destined to become cancerous. Using advanced spatial imaging, single-cell technologies, and artificial intelligence-driven image analysis, she will investigate how these immune cells interact with their surrounding environment and develop biomarkers that can accurately identify high-risk patients. Beyond improving risk prediction, the project seeks to uncover targets for therapies that could restore protective immune responses before cancer develops. By focusing on the earliest stages of disease, Dr. Qiu aims to shift cancer care from treatment to prevention. The long-term impact could be profound: more accurate diagnosis, fewer unnecessary surgeries, and entirely new strategies to intercept cancer before it becomes life-threatening.

Immuno-Informatics Postdoctoral Fellow-Spring 2026

Moritz Schaefer, PhD

Stanford University

Mentor(s): Zinaida Good, PhD, Jure Leskovec, PhD

Decoding the lymphoma microenvironment to understand CAR T cell therapy resistance

Relevance: Non-Hodgkin Lymphoma

Project Summary:

CAR T-cell therapy has revolutionized treatment for some patients with aggressive lymphoma, but only about half of patients experience durable long-term benefit. Doctors still do not fully understand why these therapies succeed in some tumors but fail in others.

Dr. Schaefer combines expertise in artificial intelligence, computer science, and systems biology to study cancer. His previous work led to the development of innovative AI systems capable of linking complex biological data with natural language and advanced tissue analysis. He has also contributed to major efforts aimed at improving CAR T-cell function through genome-wide screening technologies.

This project will develop an AI-based system capable of analyzing standard tumor biopsy slides in extraordinary detail. By training the system on tissue samples that include both microscope images and molecular information from individual cells, the AI will learn to identify cancer cells, immune cells, and the ways they are organized within tumors.

Dr. Schaefer will apply this approach to biopsy samples from hundreds of lymphoma patients treated with CAR T-cell therapy. By comparing tumors from patients who responded well with those who did not, he hopes to uncover the specific features of the tumor environment that interfere with treatment success. The findings could improve understanding of why CAR T-cell therapy fails in certain patients and guide the design of more effective immunotherapies. Beyond lymphoma, this AI system may become a powerful new tool for helping pathologists uncover hidden information within routine cancer biopsies.

Immuno-Informatics Postdoctoral Fellow-Spring 2026

Adam Weiner, PhD

Gladstone Institutes

Mentor(s): Barbara Engelhardt, PhD, Matthew Spitzer, PhD

Spatiotemporal modeling of cellular interactions to guide next-generation immunotherapies

Relevance: Broad Cancer Relevance

Project Summary:

Cancer immunotherapy depends on successful communication between different immune cells that work together to recognize and destroy tumors. However, many cancers develop ways to disrupt these interactions, allowing tumors to evade immune attack and resist treatment.

Dr. Weiner develops advanced computational tools to study how immune cells behave and communicate within tumors. His earlier research combined bioengineering, artificial intelligence, and cancer genomics to analyze how tumors evolve and how immune cells respond to therapy. This project seeks to bridge a major gap in cancer research: while modern genomic technologies can reveal which genes are active inside immune cells, they cannot easily capture the dynamic physical behaviors that determine whether immune cells successfully attack cancer.

To address this challenge, Dr. Weiner will combine live-cell imaging, genomic profiling, and artificial intelligence to observe thousands of immune cells interacting with one another and with cancer cells in real time. Using these data, he will create a "digital library" of successful and failed immune responses. The goal is to identify the cellular behaviors and interactions that lead to effective anti-cancer immunity and use this knowledge to improve the design of immunotherapies. This work could help accelerate the development of safer, more effective immune-based treatments and provide entirely new ways to study how tumors resist immune attack.

Immuno-Informatics Postdoctoral Fellow-Fall 2025

Jiayu Ye, PhD

Stanford University

Mentor(s): Ansuman Satpathy, MD, PhD

Leveraging multimodal transcriptomics to uncover the role of mesothelial cells in ovarian cancer progression and immunosurveillance

Relevance: Ovarian Cancer

Project Summary:

Ovarian cancer remains the deadliest gynecologic cancer, and nearly 80% of patients eventually develop metastatic disease that is difficult to treat. While immunotherapy has revolutionized care for several cancers, it has delivered limited benefits in ovarian cancer, in part because immune cells often fail to penetrate and function effectively within tumors. Dr. Jiayu Ye has built her career studying how the tumor microenvironment suppresses immune responses. During her doctoral training, supported by a prestigious NCI fellowship, she discovered a previously unrecognized population of senescent stromal cells that actively weaken anti-tumor immunity in breast cancer. Her findings, published in Cancer Discovery, demonstrated that eliminating these cells could restore immune activity and slow disease progression. Now at Stanford University, Dr. Ye is applying cutting-edge genomic technologies to ovarian cancer.

Her project focuses on mesothelial cells, specialized cells that line the abdominal cavity and become a major component of ovarian tumors as the disease spreads. Although these cells occupy a critical position between tumors and the immune system, their role in helping cancers evade immune attack remains poorly understood. Using advanced spatial and time-resolved genomic approaches, Dr. Ye will map how mesothelial cells interact with immune cells throughout disease progression. She will then identify specific factors produced by these cells that could be targeted to enhance immune function and slow tumor growth. This work could uncover entirely new therapeutic targets and help make immunotherapy effective for more ovarian cancer patients, offering new hope for a disease where treatment options remain urgently needed.

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 $600 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.

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