CRI Cancer Research Institute™

SPRING 2025 CRI IRVINGTON AND IMMUNO-INFORMATICS

Postdoctoral Fellows

CRI Postdoctoral Fellowships

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

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

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

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

A Letter from Our CEO

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

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

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

We are deeply honored to support these researchers 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 Spring 2025 CRI Postdoctoral Fellows and the hope they carry forward.

With admiration,

Alicia Zhou, PhD
Chief Executive Officer
Cancer Research Institute

CRI Irvington Postdoctoral Fellows

Insights from CRI's Scientific Advisory Council

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

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

CRI Irvington Postdoctoral Fellow

Hiroyasu Aoki, PhD

St. Jude Children's Research Hospital

Discovering the rules governing T-cell memory accumulation during the first decade of life for next-generation vaccine development

Research focus:
Cancer vaccines, cancer prevention, T-cell memory

Sponsor:
Thomas Paul, PhD

While most cancers are caused by a combination of genetic and environmental factors, cancers caused by viruses claim over 500,000 deaths worldwide every year. Vaccination against these cancers early in life, even during childhood, is a powerful way to prevent cancer later in life. Most cancer vaccines are designed to establish immune memory by antibodies, but T cells also play a critical role, forming life-long memories against past infections and vaccinations.

Dr. Hiroyasu Aoki's research focuses on how our bodies develop long-lasting T-cell immune memory during childhood. To uncover these mechanisms, he will follow a group of healthy young children from birth and analyze their T-cell memory using advanced techniques. Through this, Dr. Aoki hopes to answer the questions of when T-cell memory accumulates during childhood, how it impacts subsequent T-cell memory formation, and which infections or vaccinations elicit strong and stable T-cell memory in children.

"The knowledge from my research will help improve vaccine design and optimize the timing of childhood immunization, leading to the development of next-generation vaccines for cancer-causing viruses," he states.

Dr. Aoki's aspiration to become an immunologist stemmed from "a desire to combat health threats using the immune system." His doctoral research at the University of Tokyo focused on understanding anti-tumor T-cell responses using T-cell receptor sequencing. He also examined T-cell responses after SARS-CoV-2 mRNA vaccination and identified certain T-cell clones associated with favorable COVID-19 outcomes. Now refocused on cancer, Dr. Aoki ultimately hopes to pave the way for cancer prevention strategies by generating immune memory that can recognize and eliminate diverse cancerous cells.

CRI Irvington Postdoctoral Fellow

Manish Ayushman, PhD

University of California, San Francisco

Programming cell therapies that locally remodel the tumor extracellular matrix to improve efficacy against solid tumors

Research focus:
CAR T-cell therapy, solid tumors, tumor infiltration

Sponsor:
Wendell Lim, PhD

CAR T-cell therapy has shown remarkable success in treating blood cancers, but its effectiveness in solid tumors has been more limited. A key contributing factor is that in solid cancers, like breast and pancreatic cancer, the extracellular matrix (ECM) that surrounds cells and tissues is often dysregulated, forming a barrier that blocks immune cells from reaching and killing cancer cells.

Dr. Manish Ayushman's research aims to transform the ECM from a barrier into a therapeutic ally. His approach is three-fold: 1) engineer cytokines that bind the tumor ECM for sustained immune activation, 2) design ECM-targeting receptors to anchor CAR T cells inside the tumor, and 3) enable CAR T cells to break down the ECM to improve infiltration. These approaches could work individually or in combination with other drugs to help immune cells persist and function more effectively in solid tumors.

"I want to transform the tumor ECM from a barrier into a scaffold for CAR T-cell therapy," Dr. Ayushman explains. If successful, his work could significantly boost the success of cell therapies for some of the toughest cancers.

Trained in chemical- and bio-engineering, Dr. Ayushman has long explored how physical properties of tissues, like stiffness or elasticity, shape cell behavior. His doctoral work uncovered how subtle, local mechanics in engineered hydrogels can influence stem cell fate and disease responses. Now, he's applying that insight to cancer: developing biomaterials that reveal how tumors resist therapy and how we can fight back.

CRI Irvington Postdoctoral Fellow

Pilar Baldominos Flores, PhD

Harvard Medical School

Uncovering the dynamic interplay between immune surveillance and mutational landscape during tumor initiation

Research focus:
Tumor initiation, immunoediting, cancer prevention and treatment

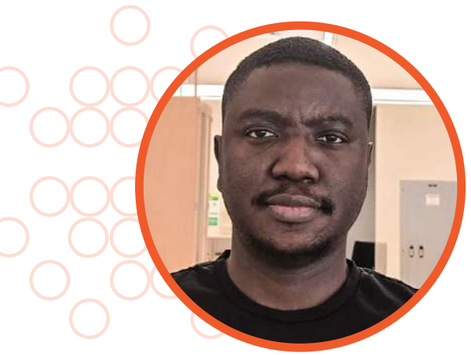
Sponsor:
Joan Brugge, PhD

"Every big problem originates from a small one, and cancers are no exception," says Dr. Pilar Baldominos Flores. While the immune system can often detect and eliminate cells with cancer-causing mutations, some manage to slip past these defenses and seed tumors.

Dr. Baldominos Flores is investigating how that happens – what specific mutations or interactions allow early cancer cells to survive and escape destruction from our immune systems? Using novel mouse models of breast tumor initiation, her work explores three key questions: 1) what immune cells infiltrate early tumors, 2) what mutations help early cancer cells evade immune attack, and 3) how does that immune response affect the mutations that occur in tumors. "Understanding how mutated cells trick the immune system during tumor formation is the first step to prevent it," she explains.

Dr. Baldominos Flores' research could not only illuminate the earliest moments of tumor development, but also identify mechanisms of resistance that undermine later-stage immunotherapies, ultimately guiding new strategies for both cancer prevention and treatment.

Dr. Baldominos Flores' research has been driven by outstanding questions in the field of cancer immunology. During her graduate work, she identified a unique population of dormant cancer cells, called quiescent cancer cells, that blocks immune cell entry and creates pockets of resistance inside tumors. To uncover how this happens, she created a spatial labeling technique that uses live-tissue sectioning and photoconvertible proteins combined with single-cell RNA sequencing. Now, Dr. Baldominos Flores is turning her focus upstream, probing the earliest immune events that allow tumors to take root in the first place.



CRI Irvington Postdoctoral Fellow

Gbolahan Bamgbose, PhD

Huntsman Cancer Institute

The role of novel open reading frames in T-ALL immunogenicity and oncogenesis

Research focus:
T-cell acute lymphoblastic leukemia, T-cell response, low mutation burden

Sponsor:
Birgit Knoechel, MD, PhD

T-cell acute lymphoblastic leukemia (T-ALL) is a rare and aggressive blood cancer. Unlike many other cancers, T-ALL cells carry few genetic mutations, which makes them hard for the immune system to recognize and even harder to target with immunotherapy.

Dr. Gbolahan Bamgbose is exploring a new frontier: overlooked regions of our genome known as "novel open reading frames" (nuORFs). These once-dismissed DNA segments can produce proteins, and recent discoveries suggest they may serve as potent triggers for immune responses. Dr. Bamgbose's project will investigate whether nuORF-derived peptides can activate the immune system against T-ALL and whether these proteins play a critical role in helping leukemia cells survive. If successful, this work could identify entirely new classes of therapeutic targets, not just for T-ALL, but for other hard-to-treat cancers with low mutation burdens.

"We hope to pave the way for therapies that either engage the body's immune system or target these proteins," he says.

Dr. Bamgbose's scientific journey began with exploring the medicinal properties of traditional Nigerian plants and expanded to uncovering treatments for parasitic diseases like ascariasis. For his PhD work, he transitioned to studying how genes are regulated during stress and development using *Drosophila melanogaster* (the common fruit fly). Now focused on cancer immunology, Dr. Bamgbose aims to investigate how T-ALL evades immune detection and seeks to find overlooked vulnerabilities in the genome that could be harnessed for new therapeutic strategies.

CRI Irvington Postdoctoral Fellow

Ondrej Belan, PhD

The Brigham and Women's Hospital

Exploring novel mechanisms of tumor immune evasion using a synthetic virome library

Research focus:
Hepatocellular carcinoma, hepatitis C virus, immune evasion

Sponsor:
Stephen Elledge, PhD

Some viruses do more than make us sick: they can rewire our cells in ways that lead to cancer and, in some cases, shield cancer cells from the immune system. Dr. Ondrej Belan's project seeks to explore exactly how viruses do that in the context of the complex mammalian immune response.

Using a groundbreaking genetic screening technology, Dr. Belan has tested genes from over 400 viruses in immune-competent mouse cancer models to identify which viral proteins promote tumor growth and immune escape. One standout was a gene called *NS5,* which is from the same viral family that includes the liver cancer-causing hepatitis C virus. NS5 drives cancer cells to produce Lect2, a molecule that shields them from immune detection. In this project, Dr. Belan will further explore how NS5 and Lect2 help cancer cells to evade the immune system and whether this viral program can be disrupted to stop liver tumor growth.

"This work has the potential to guide future virus-associated cancer prevention, diagnosis and treatment strategies," he states.

Dr. Belan trained at The Francis Crick Institute, where he pioneered single-molecule imaging systems to monitor DNA repair, the process cells use to fix genetic damage. His work revealed how proteins like BRCA2 and RAD51 regulate repair pathways and how mutations in those genes can lead to cancer, including breast and ovarian cancer. Now, he's turning to viruses – not just as infectious agents, but as evolutionary experts in genetic manipulation – to uncover how cancer cells learn to hide from the immune system.

CRI Irvington Postdoctoral Fellow

Anton Dobrin, PhD

Sunnybrook Research Institute

Establishing a genetic engineering strategy to produce iPSC-derived CD4 T cells for off-the-shelf adoptive cell therapies

Research focus:
Acute lymphocytic leukemia, CAR T-cell therapy, stem cells

Sponsor:
Juan Carlos Zuñiga-Pflücker, PhD

CAR T-cell therapies have transformed cancer treatment, but each treatment is custom-built from a patient's own cells, making it a slow, expensive, and sometimes inaccessible treatment option. A scalable, off-the-shelf version derived from stem cells could remove these barriers, but current protocols mainly produce CD8+ T cells and fail to generate CD4+ helper T cells, which are key players in long-term anti-tumor immunity.

Dr. Anton Dobrin aims to solve that challenge by evaluating two strategies to induce CD4+ T-cell development: first, by turning specific genes on or off at defined times to guide differentiation; and second, by running a broad genetic screen to uncover previously unknown regulators of CD4 lineage commitment. "More broadly, my work will establish a new platform on which extensive cell engineering can be performed and validated for safety, allowing for more carefully controlled and more potent cell immunotherapies," he explains.

Dr. Dobrin's scientific training began in a high school co-op program and led to research in global labs at ETH Zurich and Memorial Sloan Kettering Cancer Center. "I have been excited by the ability to re-engineer biology since high school," he states. In his doctoral work, he devised different strategies for activating T-cell receptor-targeted T-cells that improved anti-tumor efficacy in human cells and preclinical mouse models. Dr. Dobrin is now turning to induced pluripotent stem cells – cells that have been reprogrammed to an embryonic stem cell-like state – to address accessibility and scalability in immunotherapy, designing truly universal, off-the-shelf cancer treatments.

CRI Irvington Postdoctoral Fellow

Zachary Earley, PhD

University of California, San Francisco

Gα13 signaling in intestinal epithelial lymphocyte homeostasis

Research focus:
Inflammatory bowel disease, colorectal cancer, gut-immune balance

Sponsor:
Jason Cyster, PhD

In the gut, a special class of immune cells called intestinal intraepithelial lymphocytes (IELs) act as frontline defenders against infection and inflammation. When these cells don't function properly, it can lead to serious diseases like inflammatory bowel disease (IBD) and colorectal cancer. In this project, Dr. Zachary Earley will investigate how IELs develop, migrate, and become "long lived" to maintain gut-immune balance – and the Gα13/GPR132 signaling pathway may be the key.

The Gα13/GPR132 signaling pathway allows cells to sense and respond to environmental signals. Without Gα13, IELs fail to survive in the gut, though they remain healthy elsewhere in the body, making it a unique marker to use for manipulations. Dr. Earley hypothesizes that blocking this pathway may actually protect against gut inflammation, pointing to a possible new treatment for intestinal autoimmune diseases.

"Understanding how these diseases develop and finding novel approaches to treat them is what motivates me to pursue a career in science," Dr. Earley says. In his doctoral work at the University of Chicago, he uncovered how intestinal epithelial cells – cells that line the inside of the intestines – regulate microbial colonization and influence inflammation in diseases like celiac disease. He also revealed how an antibody called IgA maintains immune homeostasis and controls the microbiota. Now, Dr. Earley is focused on uncovering tissue-specific immune signaling pathways, like Gα13, that may hold the key to treating chronic intestinal diseases without systemic immune suppression.

CRI Irvington Postdoctoral Fellow

Samantha Fernandez, PhD

Dana-Farber Cancer Institute

Discovering conserved mechanisms of interferon-dependent cancer immunity using cross-kingdom interactions

Research focus:
Immune responses, viral pathogenesis, interferon signaling

Sponsor:
Philip Kranzusch, PhD

Interferons (IFNs) are powerful immune molecules, but their use in cancer treatment remains a double-edged sword. While IFN signaling can powerfully activate immune responses against tumors, sustained IFN activity can paradoxically lead to immune evasion and cancer relapse.

The key to resolving this contradiction may lie in interferon-stimulated genes (ISGs), a set of more than 300 poorly understood genes induced by IFN signaling. Dr. Samantha Fernandez aims to systematically characterize the immune functions of these genes, using an evolution-informed screening strategy coupled with structural biology and biochemical validation. "By taking a novel, evolutionary approach to studying ISG activity, my research will have a transformative impact on a variety of IFN-dependent cancer treatments and will open new opportunities to harness our immune system to combat disease," she says.

Dr. Fernandez's path into molecular biology began as an undergraduate student. "I was unconventional in many ways as a community college transfer student at UC Berkeley, a first-generation Filipino immigrant, and the first in my family to pursue any kind of doctoral degree," she explains. With perseverance and self-motivation, she pursued evolutionary genomics and virology research at UC Berkeley and the Gladstone Institutes in San Francisco, respectively.

During her graduate training at UC Berkeley, Dr. Fernandez developed genomics-based methods to study how cells control protein synthesis, and she discovered a new mechanism of translation regulation involving ribosome recycling. As a postdoctoral fellow, Dr. Fernandez's focus is on decoding the molecular logic of immunity – where structure, function, and evolution intersect.

CRI Irvington Postdoctoral Fellow

Jesse Garcia Castillo, PhD

University of California, San Francisco

RASGRP1 functions as a rheostat for T-cell fitness

Research focus:
Acute lymphocytic leukemia, T-cell signaling, metabolism

Sponsor:
Jeroen Roose, PhD

T cells rely on finely tuned signaling pathways to remain both vigilant and restrained – strong enough to fight cancer but not so reactive as to cause autoimmunity. One key player in this balance is RASGRP1, a signaling protein that influences T-cell metabolism and activation, but whose full role in immune regulation remains unclear.

Dr. Jesse Garcia Castillo will use genetically engineered mouse models to investigate how RASGRP1 affects CD4+ T cell homeostasis, metabolism, and translation. He will also study the impact of a newly identified genetic mutation in the *RASGRP1* gene on T-cell function and anti-tumor responses using mouse models. Ultimately, Dr. Garcia Castillo's research will provide a deeper understanding of RASGRP1's role in immune regulation and test a central question: can we enhance T-cell fitness for therapy without tipping into autoimmunity?

Dr. Garcia Castillo was born into an immigrant family and became the first in his family to attend college. "I was determined to create my own opportunities and navigated complex funding processes and immersed myself in research early on," he says.

During his undergraduate studies at California State University, Los Angeles, he joined multiple research labs in microbiology, cancer metabolism, and computational modeling. In graduate school at UC Berkeley, Dr. Garcia Castillo focused on the tumor microenvironment and bacterial immunotherapies; his thesis work explored how attenuated listeria can modulate immune responses against cancer. Now, in his postdoctoral training, Dr. Garcia Castillo is defining how RASGRP1 signaling supports T-cell fitness, laying the foundation for more precise and durable immune-based treatments.

CRI Irvington Postdoctoral Fellow

Amina Jbara, PhD

Stanford University

Gut immunocytes in cancer immunosurveillance: from mechanistic insights to rational design with defined communities

Research focus:
Cancer surveillance, gut microbiome, cancer-microbiome connection

Sponsor:
Michael Fischbach, PhD

Why do some patients respond to immunotherapy while others do not? One promising piece of the puzzle lies in the gut. Recent research shows that gut bacteria can shape immune responses to cancer, suggesting that adjusting the microbiome could boost the effectiveness of treatments like immune checkpoint blockade.

Dr. Amina Jbara aims to investigate how gut bacteria affect the migration of specific immune cells from the gut to tumors using synthetic microbial communities – a new system that allows precise manipulation of over 100 gut bacterial strains. Her ultimate goal is to understand what and how specific microbial compositions enhance anti-tumor immunity and whether those effects can be harnessed to improve treatment outcomes. "I anticipate that this approach will help identify specific bacteria that could be used to train the immune system to better fight cancer, offering a more targeted and effective approach to cancer immunotherapy," she explains.

Trained in cancer and RNA biology, Dr. Jbara's graduate studies at the Hebrew University in Jerusalem focused on alternative splicing – a process where different parts of a gene are cut and stitched together to make different proteins – in metastatic pancreatic ductal adenocarcinoma (PDA). Her work identified that a splicing factor called RBFOX2 prevented metastasis in PDA. In parallel, she discovered that the FDA-approved drug azathioprine inhibits metastatic PDA by targeting the Rac1/Cdc42 pathway, findings that helped launch a clinical trial. Now shifting focus to the cancer–microbiome connection, Dr. Jbara is developing next-generation approaches that harness gut bacteria to improve immunotherapy.

CRI Irvington Postdoctoral Fellow

Zhixin Jing, PhD

National Institute of Allergy and Infectious Diseases, NIH

Spatial dissection of bone marrow niches supporting plasma cell longevity

Research focus:
Multiple myeloma, immunological memory, bone marrow niche

Sponsor:
Ronald Germain, MD, PhD

Long-lasting immune memory is essential for protecting the body against recurring infections and sustaining the effectiveness of vaccines over time. A key component of this durable immunity is long-lived plasma cells (LLPCs), which can survive for years or even decades. In multiple myeloma (MM), malignant plasma cells exploit many of the same survival cues as LLPCs. These survival cues are provided by the bone marrow niche (BMN) where the cells reside, but how the cells and tissues within the BMN interact to sustain LLPCs or fuel MM remains poorly defined.

To unravel this mystery, Dr. Zhixin Jing is integrating multiplex immunofluorescence imaging, genetic mouse models, and systems biology approaches. He will create a map of LLPC BMNs, identify key cell-tissue interactions, and determine how BMNs are altered during MM progression. These insights could guide strategies to enhance the durability of vaccine-induced antibody responses and inform new approaches to disrupt tumor-supportive niches in MM.

"The result of this work will deepen our understanding of immunological memory for improving durability of vaccine-induced antibody responses and inform novel niche-targeting strategies for controlling MM progression and/or enhancing currently available therapies," he states.

Dr. Jing is a B-cell biologist with extensive experience in humoral immunity and vaccine-induced antibody responses. In his graduate work, he challenged long-standing assumptions about BM PCs, showing that they are motile and dynamic rather than static. He also developed a genetic model to probe LLPC survival mechanisms. Now as a postdoctoral fellow, Dr. Jing is continuing his passion for understanding LLPC survival niches in the BM, ultimately guiding the development of next-generation vaccines that elicit long-lasting protective immunity.

CRI Irvington Postdoctoral Fellow

Katherine Lindblad, PhD

Boston Children's Hospital

Hijacking dendritic cell-intrinsic NLRP3 inflammasome to drive protective immunity in liver cancer

Research focus:
Hepatocellular carcinoma, dendritic cells, innate immunity

Sponsor:
Jonathan Kagan, PhD

Hepatocellular carcinoma (HCC) remains one of the deadliest cancers worldwide, with limited response rates to current T-cell-boosting immunotherapies. About two-thirds of patients with HCC have impaired activity in another immune cell type called dendritic cells (DCs). "This makes targeting DCs a promising and underexplored avenue in HCC," says Dr. Katherine Lindblad.

Dr. Lindblad's project will focus on a unique activation state of DCs, called hyperactivated (hDCs). Unlike conventional DC activation, hDCs exhibit superior migration, cytokine secretion, and memory T-cell induction. She will establish a rules-based platform for leveraging hDC behavior and assess their potential to induce protective T-cell responses. These findings could inform next-generation DC-based immunotherapies not only for HCC, but across multiple cancer types.

Dr. Lindblad's personal experiences instilled in her "an insatiable drive to understand the defining features of 'healthy' and the etiology of disease." While she had a long-standing interest in translational biomedical research, it was clinical trial work at the National Heart, Lung, and Blood Institute that led her to commit to a career in cancer immunology research. "It gave me a profound appreciation for the impact of bench-to-bedside science. Sample 10 was never just a number – but a person, a family, pain, and hope." Dr. Lindblad subsequently completed her PhD at the Icahn School of Medicine at Mount Sinai, with a focus on how tumor-intrinsic genetics shape immunity in liver cancer.

CRI Carson Family Charitable Trust Postdoctoral Fellow

Andrew MacLean, PhD

The Rockefeller University

Elucidating the mechanisms of post-export plasma cell affinity maturation

Research focus:
Multiple myeloma, antibody responses, B cells

Sponsor:
Michel Nussenzweig, MD, PhD

The body's ability to produce protective antibodies depends on plasma cells, which usually develop from B cells that undergo a selection process to improve their ability to recognize threats. This process, which takes place in structures called germinal centers, favors B cells with high-affinity receptors – receptors that have a strong attraction or binding strength. While it was once thought that only these high-affinity cells could become plasma cells, recent studies have shown that even low-affinity B cells undergo plasma cell differentiation.

Dr. Andrew MacLean's research offers a potential explanation: high-affinity PCs may not be selected more frequently, but instead expand more rapidly through a process of "proliferative bursting." His current project builds on that initial finding, to discover the molecular signals that guide how newly formed plasma cells develop and multiply, helping us better understand how the body produces strong and effective antibody responses.

"An understanding of the factors that regulate this developmental state may inform one of the early cellular risk stages in cancers such as multiple myeloma," he explains.

Dr. MacLean's training has provided him with a strong background in molecular and cellular immunology. During his doctoral studies at the University of Oxford, he used live lung imaging to track memory B cells during influenza infection. His work revealed how memory cells migrate and differentiate into plasma cells at sites of re-infection. Now as a postdoctoral fellow, Dr. MacLean is investigating how B cells undergo affinity-based selection and plasma cell maturation, uncovering fundamental principles that govern antibody strength and longevity.

CRI Irvington Postdoctoral Fellow

Shannon McGettigan, PhD

University of Pennsylvania

Targeting purinergic receptors in normal plasma cells and multiple myeloma

Research focus:
Multiple myeloma, plasma cells, stress response

Sponsors:
David Allman, PhD, and Dan Vog, MD, MSCEI

Multiple myeloma (MM) is a deadly cancer of plasma cells, and despite current therapies that reduce tumor burden, most patients with MM have life-threatening relapses. Understanding the biology of normal and malignant plasma cells could reveal potential novel therapeutic targets. Normal and malignant plasma cells produce large amounts of antibodies, and they have both adapted to survive the stress of high antibody production.

Dr. Shannon McGettigan's project will investigate a previously unrecognized survival mechanism for these cells: the sensing of a molecule called ATP. ATP is released by nearby bone marrow cells, and she hypothesizes that plasma cells sense ATP through a special opening, or channel, in the cell surface. Using *in vitro* cell cultures and *in vivo* mouse models, Dr. McGettigan will inhibit the ability of plasma cells to sense ATP and assess how it impacts them and their neighboring bone marrow cells.

"These studies will provide critical insights into the biology of plasma cells and MM and reveal potential novel therapeutic targets for MM," she says.

Dr. McGettigan's scientific training began in microbiology as an undergraduate student, where she investigated emerging antibiotic resistance genes. She then developed and optimized CAR T cells as an immunotherapy for blood and solid cancers, building a patented diagnostic platform to pre-screen patients for likely responders to therapy. Dr. McGettigan's graduate research focused on B-cell and skin immunology, investigating factors controlling cutaneous IgM secreting cells. Now as a postdoctoral fellow, she is studying plasma cell survival and MM, integrating molecular immunology with translational approaches to identify new therapeutic targets.

CRI Irvington Postdoctoral Fellow

Tara Muijlwijk, PhD

New York University Grossman School of Medicine

Defining tumor and immune co-evolution in cutaneous melanoma lymph node metastasis

Research focus:
Melanoma, lymph node metastasis, cancer-immune cell interactions

Sponsor:
Amanda Lund, PhD

Melanoma is an aggressive form of skin cancer that can become life-threatening when it spreads, or metastasizes, to other parts of the body. While immunotherapy has improved outcomes for some patients, many still fail to respond. Dr. Tara Muijlwijk is investigating how melanoma spreads to the lymph nodes and how the immune system's response within these sites may actually support cancer progression.

The lymph nodes are small immune hubs designed to detect and respond to threats like cancer. However, tumors can co-opt their function to avoid immune attack. Using advanced imaging and molecular tools, Dr. Muijlwijk will map interactions between immune and tumor cells in these metastatic environments. "By uncovering these processes, we hope to identify new strategies to block melanoma spread and improve the effectiveness of immunotherapy," she explains.

Her research could also offer insights into other cancers that metastasize to the lymph nodes, including breast and head and neck cancers.

Dr. Muijlwijk is a translational immuno-oncology researcher with expertise in tumor heterogeneity and immune regulation. Her early work explored genetic diversity in gastrointestinal cancers and immune suppression in sentinel lymph nodes. During her PhD, she characterized a distinct subclass of head and neck cancer with few copy number alterations (a specific type of genetic mutation) and studied how immune landscapes vary across tumor sites. She now investigates tumor-immune interactions in melanoma, combining human specimen analysis with mechanistic studies to inform next-generation immunotherapies.

CRI Irvington Postdoctoral Fellow

Fiona Raso, PhD

New York University Grossman School of Medicine

Targeting tolerogenic dendritic cells during inflammation

Research focus:
Inflammation, gut microbiome, immune tolerance

Sponsor:
Dan Littman, MD, PhD

Every day, the immune system works to protect the body from disease-causing pathogens. To achieve this, immune cells have developed ways to distinguish foreign invaders like bacteria and viruses from harmless substances such as food and coordinate the proper response. When these checkpoints fail, innocuous peanuts, pollen, or even one's own cells can be viewed as dangerous, causing allergies or autoimmune diseases to develop.

Dr. Fiona Raso's research seeks to understand how immune cells misinterpret food or gut bacteria to develop new treatments, and a newly discovered, specialized tolerizing immune cell may be key. These particular immune cells are responsible for helping educate other immune cells to ignore harmless substances like dietary proteins and commensal microbes. Dr. Raso will examine the role that tolerizing immune cells play in different inflammatory conditions and see if they can be manipulated to restore immune tolerance and reverse disease.

"These studies will elucidate if tolerizing immune cells are amendable," she explains, "and would provide the basis for clinical trials to prevent disease or disease progression."

Dr. Raso completed her doctoral training at University of Massachusetts Chan Medical School where she studied B-cell receptor signaling and differentiation and migration of plasma cells to the gut. She also identified key survival niches and migratory cues for memory B cells in the small intestine and contributed to studies on mucosal immune regulation. Now a postdoctoral fellow, Dr. Raso is investigating how tolerogenic dendritic cells regulate immune tolerance to food and microbiota and how these cells can be manipulated to treat autoimmunity and allergy.

CRI Irvington Postdoctoral Fellow

Kristen Witt, PhD

University of Washington

Regulation of nucleic acid sensing in the central nervous system

Research focus:
Neurological disorders, nucleic acid sensing, immune defenses

Sponsor:
Daniel Stetson, PhD

Nucleic acid sensing is a critical immune defense that detects foreign genetic material from viruses or damaged cells. While this process helps fight infections and cancer, it must be tightly regulated, especially in sensitive tissues like the brain. Uncontrolled nucleic acid sensing in infancy can cause catastrophic neurological damage, as seen in conditions like Aicardi-Goutières Syndrome (AGS), yet how this system is normally kept in check remains unclear.

Dr. Kristen Witt aims to uncover how nucleic acid sensing is regulated in the brain and why its misregulation causes such severe damage in infancy. Her work will generate a new mouse model of AGS and use developmental mapping and molecular profiling to pinpoint how and when nucleic acid signals are controlled. "This research will provide insight into neurological disorders linked to nucleic acid sensing," she explains, "and inform how to therapeutically modulate nucleic acid sensing to treat cancer."

Dr. Witt's scientific journey began at Harvard Medical School, "motivated by my love for science and desire to help people". She trained in viral immunology and innate immunity, first studying HIV envelope proteins at the Dana-Farber Cancer Institute and then investigating inflammasome regulation at Boston Children's Hospital.

In graduate school at UC Berkeley, she illuminated exciting new roles for a protein called SP140 in regulation of antiviral immunity, a new regulator and regulatory mechanism of interferon, and a fascinating host-virus arms race in the nucleus. Now as a postdoctoral fellow, Dr. Witt's research focuses on how nucleic acid sensing is regulated in the developing brain and how its disruption contributes to inflammation-linked neurological disease.

CRI Irvington Postdoctoral Fellow

Kai Xu, PhD

Boston Children's Hospital

Role of cohesin-mediated loop extrusion in somatic hypermutation

Research focus:
Blood cancers, B cells, somatic hypermutation

Sponsor:
Frederick Alt, PhD

The immune system's ability to make more effective antibodies depends on a process called somatic hypermutation (SHM), in which B cells undergo a refining process to improve how well they recognize threats. This improvement happens through a special enzyme called AID, which introduces small genetic changes that help fine-tune antibody response. One big mystery is how AID targets only the right parts of the genome, since it has the potential to cause harmful mutations elsewhere.

Dr. Kai Xu hypothesizes that a special "mutation zone" forms around antibody gene regions in B cells, helping AID focus its activity and avoid damaging other parts of the DNA that could lead to cancer. Using new experimental platforms, he will test the structure and function of these zones. "I am excited about the potential of our planned studies to uncover fundamental mechanisms to help pave the way for development of novel approaches to treating or preventing GC B-cell lymphomas," he says.

Dr. Xu's biomedical research journey began in 2009 at Northeastern Forestry University in China, where he was fascinated with the process of designing and performing experiments which inspired his future research trajectory. His training in stem cell and embryonic biology led him to Tsinghua University where his doctoral work focused on uncovering key roles for RNA-binding proteins and chromatin architecture in early embryo development. Now at Boston Children's Hospital, Dr. Xu is developing new platforms to test how genomic architecture restricts AID activity to antibody genes, work that could provide insight into antibody diversification and lymphoma development.

CRI Immuno-Informatics Postdoctoral Fellows

Insights from CRI's Scientific Advisory Council

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

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

CRI Immuno-Informatics Postdoctoral Fellow

Samira Ghazali, PhD

Harvard Medical School

Deciphering transcription factor networks governing Treg identity and function in homeostasis and cancer

Research focus:
Regulatory T cells, transcription factors, immune regulation

Sponsor:
Christophe Benoist, MD, PhD

Regulatory T cells (Tregs) are essential for keeping the immune system in check, however, in cancer, these cells can also suppress the body's natural ability to fight tumors, making it harder for cancer treatments to work. Understanding the elements that control Treg function and adaptability is critical to developing drugs that selectively target tumor-infiltrating Tregs without disrupting their essential role in the rest of the body.

Dr. Samira Ghazali will add to this knowledge-base by using an advanced technique called DAF-sequencing, which maps the molecular switches (called transcription factors, or TFs) that control Tregs. She will compare Tregs from healthy tissues and tumors in mice and identify key differences in TFs. She will also investigate the role of FoxP3, a protein that plays a major role in Treg function, but whose exact role is still unclear.

"Our research will provide new insights into how Tregs are controlled at the molecular level, opening the door to more precise and effective cancer treatments," she explains.

Dr. Ghazali's passion for computational immunology is both academic and personal. "This interest may have been shaped by my childhood experiences. In many ways, merging my love for computational sciences with a desire to contribute to medical research was a natural path," she says. Her PhD work integrated transcriptomic and epigenomic datasets to study CD4+ T-cell differentiation, uncovering novel genetic elements that help maintain T-cell identity. Dr. Ghazali is now investigating the transcriptional regulation of Tregs in tumors, using advanced genomic tools to inform new cancer immunotherapy strategies.

CRI Immuno-Informatics Postdoctoral Fellow

Linglin Huang, PhD

The Brigham and Women's Hospital

Dissecting neuroimmune interactions in the tumor microenvironment to enhance anti-tumor immunity

Research focus:
Cancer, neuro-immune crosstalk, anti-tumor response

Sponsors:
Vijay Kuchroo, PhD, DVM, and Martin Hemberg, PhD

The immune system plays a vital role in detecting and eliminating cancer, but tumors often evolve ways to suppress immune activity and escape control. While extensive research has focused on tumor-immune interactions, a growing body of evidence suggests the nervous system also shapes immune responses in cancer. However, how nerve signals influence immune cells in the tumor microenvironment remains poorly understood.

Dr. Linglin Huang's research investigates how sensory neurons, which detect inflammation and transmit pain signals, interact with immune cells in tumors. Early findings suggest that neuropeptides released by these neurons may suppress or enhance the body's anti-tumor immune response. Using cutting-edge genetic tools, single-cell technologies, and computational analysis, Dr. Huang will map neuropeptide signaling pathways and test whether blocking specific neuropeptides can strengthen immunotherapy.

Because many neuromodulatory drugs are already FDA-approved for other conditions, this research may accelerate the development of new cancer treatments. "If we can repurpose existing neuromodulators to boost immune activity, we could unlock faster, safer ways to improve patient outcomes," she explains.

Dr. Huang is a computational biologist and biostatistician whose research spans multiple fields, with a primary focus on cancer immunology. During her PhD at Harvard University, she developed statistical methods for analyzing single-cell RNA sequencing data, enabling more robust interpretations of immune dynamics. As a postdoctoral fellow, Dr. Huang will use her multidisciplinary background to explore how nerve signaling shapes anti-tumor immunity and to uncover new therapeutic strategies in cancer immunology.

CRI Immuno-Informatics Postdoctoral Fellow

Rongting Huang, PhD

Stanford University

Informatics-driven spatial-omics for cancer immunotherapy discovery in gynecologic cancers

Research focus:
Endometrial cancer, cancer cell clones, tumor mapping

Sponsors:
Brooke Howitt, MD, and Sizun Jiang, PhD

Endometrial cancer is the most common gynecologic malignancy and claims thousands of lives annually. More than 50% of advanced cases relapse due to varied subsets of tumor cells that resist treatment and evade detection by traditional medical tools. These tools average data across tumors, missing critical differences between different regions.

Dr. Rongting Huang plans to tackle this challenge by creating comprehensive maps of tumors and their surroundings, revealing how cancer cells in different clones escape treatment by "rewiring" their local environment. Her pioneering approach combines advanced imaging and sequencing technologies to chart both genetic diversity and immune interactions across the tumor landscape. By unmasking these hidden dynamics, Dr. Huang will empower clinicians to deliver personalized treatments, guiding precision surgery or radiation to high-risk areas and unlocking combination immunotherapies.

"This work will offer patients a roadmap to outmaneuver cancer's complexity and reclaim hope," she explains.

Dr. Huang is a computational biologist "driven by the challenge of bridging cutting-edge computational tools with transformative cancer research." During her PhD, she developed a statistical framework called XClone to detect subclonal genetic mutations. She later expanded this tool to map tumor heterogeneity, revealing how they interact with surrounding cells in the tumor environment. As a visiting researcher at Harvard Medical School, she developed spatialAE, a deep learning model for analyzing immune cell morphology. Now as a postdoctoral fellow, Dr. Huang is deploying her multi-disciplinary training to lead transformative studies in cancer evolution and precision medicine.

CRI Immuno-Informatics Postdoctoral Fellow

Tyler Park, PhD

Memorial Sloan Kettering Cancer Center

Role of AIRE in thymic tolerance to T cells

Research focus:
Immune tolerance, autoimmunity, immune evasion

Sponsors:
Christina Leslie, PhD, and Chrysothemis Brown, MBBS, PhD

Immune tolerance protects the body from autoimmune disease by preventing the immune system from attacking normal tissues. But, this protective mechanism can also shield cancer cells from immune attack. A key player in this process is a gene called *AIRE*, which trains developing immune cells in the thymus to recognize and ignore the body's own proteins.

Dr. Tyler Park's research aims to better understand how AIRE works at the molecular level – specifically, how it helps thymic cells display these "self" proteins and eliminate harmful immune cells before they mature. By using computational and statistical tools, he will map the proteins AIRE interacts with and the pathways it controls. His work will also investigate how dysregulation of these processes might contribute to immune escape in cancer or the development of autoimmune conditions.

"We seek to uncover how AIRE functions and ultimately how immune tolerance is regulated, with the goal of improving immunotherapies while minimizing the risk of autoimmune diseases," he explains.

The foundation of Dr. Park's research lies in "leveraging mathematical and statistical principles to understand biological processes and diseases." He earned his PhD in computational biology at Princeton University, where he developed an algorithm called the SuperDendrix to identify genes essential for cancer survival and the somatic mutations that explain their molecular basis. He also developed a statistical framework to predict drug-target interactions using drug response and CRISPR screens. Now as a postdoctoral fellow, Dr. Park applies machine learning and multiomic analyses to investigate immune tolerance and uncover actionable insights into immune regulation.

CRI Mark Foundation Immuno-Informatics Postdoctoral Fellow

Maryam Pourmaleki, PhD

Stanford University

Spatio-temporal quantification of tumor-immune interactions in colorectal cancer as a determinant of organotropism

Research focus:
Colorectal cancer, metastasis, organotropism

Sponsors:
Christina Curtis, PhD, and Karuna Ganesh, MD, PhD

Colorectal cancer (CRC) is a leading cause of cancer-related death, with most mortality driven by metastasis – typically to the liver or lungs, but rarely to the brain. While organ-specific spread of cancer, or organotropism, has been recognized since the 19th century, the cellular and microenvironmental factors that govern it remain poorly understood.

Dr. Maryam Pourmaleki hypothesizes that primary tumors harbor molecular or cellular features predictive of future metastasis, which may currently go undetected using standard diagnostic methods. In this project, she will develop a tool to model metastasis through space and time and use it to identify interacting tumor-immune cells that predict organotropism. Dr. Pourmaleki will also validate these tumor-immune interactions using a second tool and 3D tumor data from patients with CRC, together with lab experimentation. "This project will identify improved approaches for early detection and treatment of organ-specific CRC metastasis," she explains.

Dr. Pourmaleki's path into cancer research was shaped by personal experience. "My aspirations to improve cancer outcomes began during childhood, stemming from personal experience with cancer in my family. This ignited my passion in medicine," she says.

Her doctoral work in computational biology and medicine focused on relating cancer spatial topology to clinical features. She used machine learning to study how the spatial organization of tumors relates to clinical outcomes, identifying biomarkers and therapeutic targets across melanoma, lymphoma, glioblastoma, and non-small cell lung cancer. Now a postdoctoral fellow, Dr. Pourmaleki is focused on understanding how tumors acquire the ability to metastasize, aiming to create new tools where current methods fall short.

CRI Immuno-Informatics Postdoctoral Fellow

Rajat Punia, PhD

Cornell University

Computational design of vaccine immunogens for broad-spectrum immunity against hepatitis C virus

Research focus:
Hepatitis C virus, cancer vaccine, liver cancer

Sponsors:
Andrew Flyak, PhD, and Joe Grove, PhD

Hepatitis C virus (HCV) is a leading cause of liver cancer and responsible for nearly 300,000 deaths annually. While antiviral therapies can cure HCV, they do not prevent reinfection, and no preventive vaccine currently exists. Developing a vaccine for HCV has been difficult because the virus mutates quickly and exists in many different forms, or strains. In addition, traditional development strategies have failed because they tend to produce immune responses that only protect against specific strains and they often focus the immune system on parts of the virus that don't generate strong, protective antibodies.

Dr. Rajat Punia aims to design vaccine candidates that mimic a powerful natural immune response seen in some patients infected with HCV – one that produces broadly neutralizing antibodies that target conserved regions of HCV that are critical for infection.

Using computational modeling and protein engineering, he will develop a two-step vaccine that first trains the immune system to recognize these conserved targets and then boosts this response. "If successful, this research could lay the foundation for a much-needed preventive vaccine against HCV and its associated cancers," he states.

Dr. Punia trained in chemical engineering at the Indian Institute of Technology Delhi before earning a PhD in computational biology. His work uncovered mechanisms behind viral entry, lipid membrane dynamics, and drug interactions with cancer-related proteins. He later applied AI-driven methods to antibody design at a biotech startup. Now a postdoctoral fellow, Dr. Punia is developing vaccine candidates for HCV by modeling how viral surface proteins interact with the immune system.

CRI Dr. Keith Landesman Memorial
Immuno-Informatics Postdoctoral Fellow

Anna Ralser, MD, PhD

Gladstone-UCSF Institute
of Genomic Immunology

Inducing immune niches to unlock anti-tumor responses in immunotherapy-resistant CRC

Research focus:
Colorectal cancer, immunotherapy, tumor microenvironment

Sponsors:
Karin Pelka, PhD, and Barbara Engelhardt, PhD

Colorectal cancer (CRC) remains a leading cause of cancer deaths and is largely unresponsive to immunotherapy due to its immunosuppressive tumor microenvironment (TME). In tumors that do respond, immune cells cluster into localized "immune niches" and coordinate their anti-tumor activity. These structures are conserved across cancer types, suggesting that therapeutically inducing them could unlock new treatment strategies.

Dr. Anna Ralser is developing a method to induce these niches in CRC by reprogramming tumor cells to recruit and organize immune responses. Using a sophisticated mouse model that mimics the complexity of human CRC, she will identify immune-attracting signals and validate them through a high-throughput screen of genetically barcoded tumor organoids. "This research will reveal new strategies to unleash the power of the body's own defenses and enable the development of effective immunotherapies against CRC and other solid tumors with immunosuppressive TMEs," she says.

As an MD/PhD, Dr. Ralser's academic journey began with medical training, which sparked her interest in the molecular mechanisms of cancer. "I realized that I could contribute to understanding cancer pathophysiology and uncover new therapeutic avenues," she says. During her PhD at the Technical University of Munich, she identified microbiota-immune signatures that promote colorectal tumor development and conducted multiomic analyses to track tumor-immune interactions. Since joining the Gladstone-UCSF Institute of Genomic Immunology, Dr. Ralser has developed a complex murine tumor model and a novel barcoding strategy to track spatial immune responses – tools she now uses to pursue breakthroughs in CRC treatment.

About CRI

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

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

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

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