

Meet the 16 Early Career Scientists Awarded \$4.8M by the Damon Runyon Cancer Center Research Foundation

These 2025 Damon Runyon cancer research fellows study bone-deep pain, bacterial secretions, re-engineered viruses, gamma delta T cells and more.

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The Damon Runyon Cancer Research Foundation has named 16 new Damon Runyon Fellows, brilliant postdoctoral scientists conducting basic and translational cancer research in the laboratories of leading senior investigators. This prestigious Fellowship encourages the nation's most promising young scientists to pursue careers in cancer research by providing them with independent funding (\$300,000 total over four years) to investigate cancer causes, mechanisms, therapies, and prevention.

"Damon Runyon gave me the freedom to pursue a high-risk project," said current Fellow Zeda Zhang, PhD. "But it also brings together smart people with very different expertise. And by creating this network, it fosters interdisciplinary research—because no one can single-handedly cure cancer."

"We are thrilled to be funding these innovative, young scientists with the brilliance and passion to push boundaries and make breakthroughs. Damon Runyon Fellows are the future leaders of their respective fields," said Yung S. Lie, PhD, President and CEO of Damon Runyon.

Spring 2025 Damon Runyon Fellows

Carissa Chan, PhD, with her sponsor Daniel A. Portnoy, PhD, at the University of California, Berkeley

Dr. Chan's research focuses on gamma delta T cells, an unusual and understudied population of immune cells. While gamma delta T cells have strong antitumor activity, they are most highly stimulated not by cancer cells but by signals produced by microorganisms. Dr. Chan's work examines the mechanisms by which gamma delta T cells detect and respond to leukemia versus pathogenic microorganisms, and how infection with these microorganisms subsequently impacts the trajectory of leukemia. This is particularly relevant to patients undergoing conventional cancer treatments (e.g., chemotherapy) that suppress the immune system, rendering them susceptible to

infection. Furthermore, gamma delta T cells are capable of both rapid and long-term responses against their targets, which positions them as a tool to treat initial cancer as well as prevent disease recurrence. Dr. Chan received her PhD from Yale University, New Haven, and her BS from the University of California, Los Angeles.

Yuxuan Chen, PhD, with his sponsor Daniel G. Anderson, PhD, at Massachusetts Institute of Technology, Cambridge

Dr. Chen is genetically re-engineering cancer-infecting viruses so that, once inside a tumor cell, they flip on a built-in “self-destruct” circuit called pyroptosis. This explosive form of cell death not only wipes out the infected cell but also broadcasts an alarm that rallies the immune system against the whole tumor. By pairing this viral upgrade with an ultrasound trigger, Dr. Chen aims to turn treatment-resistant pancreatic cancer—and, ultimately, other solid tumors—into diseases the immune system can eradicate. Dr. Chen received his PhD and MS from Zhejiang University, Hangzhou, and his BS from Southwest Jiaotong University, Chengdu.

Leslie A. Day, PhD, with her sponsor George A. O’Toole, PhD, at the Geisel School of Medicine at Dartmouth, Hanover

Dr. Day is investigating how bacteria called *Prevotella* contribute to oral squamous cell carcinoma (OSCC), the most common type of oral cancer. These bacteria are found in higher numbers in the mouths of people with OSCC compared to healthy individuals, and studies suggest they may promote tumor growth and make cancer treatment more difficult. When patients undergo surgery to remove oral tumors, these bacteria can form resistant communities called biofilms that survive antibiotic treatment, increasing the risk of post-surgical infections. By developing genetic tools to study *Prevotella*, Dr. Day aims to identify how these bacteria persist in cancer environments and potentially discover new ways to improve OSCC treatment outcomes. Dr. Day received her PhD from the University of Minnesota, St. Paul, and her BS from the University of Missouri, Columbia.

Phaedra C. Ghazi, PhD, with her sponsor Tyler Jacks, PhD, at Massachusetts Institute of Technology, Cambridge

An emerging hallmark of cancer is phenotypic plasticity, which enables cancer cells to change their traits throughout tumorigenesis and in response to targeted therapy treatment. While new targeted therapies are emerging for the treatment of lung cancers, it has been demonstrated that lung tumors with heterogeneous cell populations are especially resistant to current treatments. One reason that lung cancer cells with the same cancer-causing mutation but different cellular identities may have differential sensitivities to targeted therapies is altered gene expression. Dr. Ghazi aims to characterize how these hybrid lung tumors are innately resistant to treatment by determining differences in gene expression and regulation. She is engineering a novel mouse model that reports the cellular identity of lung tumors in live animals. Together these efforts aim to improve our ability to treat particularly aggressive lung tumors. Dr. Ghazi received her PhD and MS from the University of Utah, Salt Lake City, and her BS from the University of Massachusetts, Amherst.

Jane E. Lodwick, PhD, with her sponsor Rebecca L. Lamason, PhD, at Massachusetts Institute of

Technology, Cambridge

Many bacteria naturally colonize tumors, inspiring an emerging class of microbial therapeutics that can be modified to specifically deliver drugs into cancer tissue. In one promising drug delivery strategy, bacteria are engineered to efficiently transfer bespoke molecular cargo into human cells through bacterial membrane channels called secretion systems. Although many kinds of bacterial secretion systems exist, few have been studied well enough to be used in delivery platforms. Dr. Lodwick plans to investigate the structure and function of a specific secretion system in a bacterium called *Rickettsia parkeri*. Understanding how and when its components assemble into a cargo delivery machine may aid the development of new bacteria-based cancer therapies. Dr. Lodwick received her PhD and MS from the University of Chicago, Chicago, and her BA from Wellesley College, Wellesley.

Ling S. Loh, PhD, with her sponsor Joseph Parker, PhD, at California Institute of Technology, Pasadena

In simple terms, cancer arises when some cells in our body stop cooperating with the rest and start growing uncontrollably, threatening the whole organism. This breakdown in cooperation is similar to how certain beetles (called myrmecophiles) infiltrate ant colonies and selfishly use their resources, acting like a “cheater” in a cooperative society. Both cancer cells in healthy tissue and these beetle invaders in ant colonies represent a failure of cooperation, whether among cells in an organism or individuals in a colony. Ant colonies, like multicellular organisms, rely on strict controls to function properly, and when those controls are bypassed, the whole system is at risk. By recreating a key “cheating” trait in beetles—disabling their surface chemical signals to let them sneak into ant colonies—the project aims to reveal universal principles about how cooperation breaks down and how systems might evolve defenses against such threats. These insights could help to understand the fundamental properties of cancer and how to design better strategies to stop it. Dr. Loh received her PhD from George Washington University, Washington D.C., and her BS from National University of Singapore, Singapore.

Kate M. MacDonald, PhD [Connie and Bob Lurie Fellow], with her sponsor Karlene A. Cimprich, PhD, at Stanford University School of Medicine, Stanford

Despite being necessary for life, gene expression is dangerous. It forces the double-stranded DNA helix to unwind and separate, so that one strand can be used as the template to synthesize an RNA molecule—the cousin of DNA that brings genes to life. This process leaves the opposite strand exposed and vulnerable to accidental damage and mutation, which can then cause cancer. Dr. MacDonald’s work will systematically check various features of RNA molecules, looking for characteristics that cause an RNA sequence to aberrantly stabilize on its template DNA, prolonging the vulnerable exposure of the opposite DNA strand. Using a new kind of microscopic RNA imaging that she developed, she will find the cellular proteins responsible for removing pathologically stable RNA molecules from DNA. Uncovering the molecular features that promote gene expression-driven DNA damage will deepen our understanding of the origins and development of all cancers. Dr. MacDonald earned her PhD from the University of Toronto, Toronto, and her BS from the University of British Columbia, Vancouver.

Hannah R. S. Martin, PhD [Connie and Bob Lurie Fellow], with her sponsor David Julius, PhD, at the University of California, San Francisco

“Bone-deep pain” is more than a metaphor. Bones and joints are constantly monitored by sensory neurons (nociceptors) that detect damage and trigger protective pain responses. However, in bone cancer and osteoarthritis, this pain can become chronic and debilitating—especially when the bone’s rich environment is colonized by migrating metastatic tumor cells originating in the prostate, breast, or lung. Dr. Martin’s research aims to uncover how skeletal sensory neurons are activated and remodeled in these conditions. Using neurophysiology techniques, she is mapping how skeletal neurons respond to potential triggers and testing a new hypothesis: that these neurons not only detect tumors but also influence their growth. This work may uncover new strategies for treating chronic skeletal pain. Dr. Martin received her PhD from the University of Chicago, Chicago, and her BS from St. John’s University, New York.

Matthew L. Miller, PhD, with his sponsor Susan M. Kaeck, PhD, at the Salk Institute for Biological Studies, San Diego

Glioblastoma is a highly aggressive form of brain cancer that, unfortunately, resists most existing treatments, including immunotherapy. This resistance largely stems from the brain’s protective barriers that restrict immune cells from effectively reaching and attacking tumors. Dr. Miller’s research focuses on overcoming these barriers by understanding how immune cells, specifically T cells, can better navigate into brain tumors and survive in the harsh, nutrient-poor environment surrounding them. Dr. Miller is identifying the chemical signals (chemokines) that direct immune cells from the brain’s borders (the meninges) into the tumor area. By mapping these pathways, he aims to engineer immune cells to recognize and respond more effectively to these signals, improving their ability to reach and attack tumors. Secondly, he will study how the unique metabolic conditions in the fluid surrounding the brain (cerebrospinal fluid) influence the function of immune cells. This fluid contains substances that might limit immune cells’ ability to attack cancer cells. By analyzing these conditions, Dr. Miller intends to design immune cells that are better adapted to survive and function effectively within the brain’s challenging environment. Ultimately, his goal is to develop strategies that significantly improve the effectiveness of immunotherapy for glioblastoma, offering new hope to patients with this devastating cancer. Dr. Miller received his PhD from the University of California, Los Angeles, and his BS from the University of California, Berkeley.

Léa Montégut, PhD [National Mah Jongg League Fellow], with her sponsor Miriam Merad, MD, PhD, at Icahn School of Medicine at Mount Sinai, New York

Early detection of lung cancer is associated with significantly better clinical outcomes. For this reason, CT-scan-based screenings in at-risk populations have been widely adopted, notably in people over 50 with a history of smoking. Still, other contributing factors include chronic exposure to environmental carcinogens, advanced age, and preexisting lung conditions. Compounding this complexity, not all precancerous lesions will evolve into invasive tumors, emphasizing the need to understand the mechanisms that govern the shift from benign to malignant states. To address this gap, Dr. Montégut will focus on decoding the early immune system alterations that occur within the lung microenvironment during the pre-cancer-to-cancer transition. By doing so, she aims to

identify molecular markers that indicate high-risk patients and pinpoint potential molecular targets, with the goal of intercepting tumors at a non-invasive stage. Dr. Montégut received her PhD from Paris-Saclay University, Paris, her MS from Polytechnique Montréal, Montréal, and her MEng from Ecole Polytechnique, Palaiseau.

Parker J. Nichols, PhD [Merck Fellow], with his sponsor Brenda L. Bass, PhD, at the University of Utah, Salt Lake City

Cells have a built-in defense system that detects double-stranded RNA (dsRNA), a molecule often associated with viruses. Although this system evolved to fight microbial infections, activating it can also help the body recognize and attack cancer, especially when combined with treatments that make tumors easier for immune cells to detect. However, tumors can escape this immune pathway by producing high levels of a protein called ADAR, which edits dsRNA by changing one of its building blocks, adenosine, into inosine. These edits prevent the dsRNA from being recognized, allowing cancer cells to stay hidden even during treatment. Dr. Nichols' research aims to understand how these RNA changes block immune detection and to identify which RNA molecules are most likely to trigger an immune response. By uncovering how cancer cells use this editing process to escape detection, he hopes to support the development of better immunotherapy treatments. Dr. Nichols received his PhD from the University of Colorado, Anschutz Medical Campus, Aurora, and his BA from Lewis and Clark College, Portland.

Anna Karen Orta, PhD, with her sponsor Gabriel C. Lander, PhD, and her co-sponsor Danielle A. Grotjahn, PhD, at the Scripps Research Institute, La Jolla

Mitochondria are best known as the cell's power plants, but they also help cells respond to stress and repair their own DNA. A mitochondrial protein called ATAD3A plays a key role in these processes and is found at abnormally high levels in many aggressive cancers such as glioblastoma, breast cancer, and colorectal cancer, where it contributes to tumor growth and resistance to treatment. Dr. Orta studies how ATAD3A acts as a sensor of mitochondrial DNA damage—detecting trouble inside the mitochondria and helping signal to other parts of the cell, like the endoplasmic reticulum, that stress responses are needed. Using cryo-electron microscopy along with biochemical and cell-based approaches, she aims to uncover how ATAD3A is regulated and how its function supports cancer cell survival. Ultimately, she hopes to expose new ways to target mitochondrial stress pathways in cancer. Dr. Orta received her PhD from California Institute of Technology, Pasadena, and her BS from the University of Texas at El Paso, El Paso.

Rebecca Pasquarelli Rios, PhD [HHMI Fellow], with her sponsor Stephen J. Elledge, PhD, at Brigham and Women's Hospital, Boston

In order to survive, cancer cells must evade or disable the immune system. Many cancers are caused by chronic infection with viruses that modulate the host cell by influencing gene expression to achieve this same goal. Dr. Pasquarelli Rios is studying how viral proteins alter gene expression to create a tumor microenvironment that prevents the immune system from killing cancer cells. Since many viral proteins achieve this by changing the expression of secreted proteins, which can be therapeutically targeted, Dr. Pasquarelli Rios is also exploring the effect of secreted proteins on tumor immune evasion. This work will deepen our understanding of how

cancers evade the immune system and has the potential to uncover new targets for cancer treatment. Dr. Pasquarelli Rios received her PhD from the University of California, Los Angeles, and her BS from Brown University, Providence.

Denis Torre, PhD, with his sponsors Danwei Huangfu, PhD, and Thomas M. Norman, PhD, at Memorial Sloan Kettering Cancer Center, New York

Genetic disturbances can disrupt normal cellular programs, promote unrestricted proliferation (i.e., tumor growth), and expose vulnerabilities that can be targeted therapeutically. However, how cells dynamically respond to such changes over time remains incompletely understood. Dr. Torre will use cutting-edge genetic tools, such as CRISPR and single-cell RNA sequencing, to study the precise sequence of molecular events triggered upon silencing of key regulators of cell identity and proliferation in human cells. By combining single-cell data with advanced statistical modeling, this work will reveal how gene perturbations dynamically alter cellular networks and drive survival or cell death, thus helping inform the development of novel cancer treatments. Dr. Torre received his PhD from the Icahn School of Medicine at Mount Sinai, New York, and his BS from the University of Trieste, Trieste.

Daniel A. Waizman, PhD [Connie and Bob Lurie Fellow], with his sponsors Ari B. Molofsky, MD, PhD, and Richard M. Locksley, MD, at the University of California, San Francisco

Dr. Waizman is trying to understand how we can apply principles of resilience in the context of colorectal cancer. He is investigating how a cellular alarm signal known as Interleukin-25 triggers the immune system to create protective layers within barrier tissues, such as the intestine, to increase their environmental defenses and capacity for tissue regeneration after injury. These layers could serve two-fold functions—in prevention of colorectal cancer and the prevention or reduction of adverse side effects of cancer treatment, which serve as a barrier to continued treatment. Dr. Waizman received his PhD from Yale University, New Haven, and his BA from Cornell University, Ithaca.

Longfu Xu, PhD [HHMI Fellow], with his sponsor Carlos J. Bustamante, PhD, at the University of California, Berkeley

Many cancers develop when crucial “cellular machinery” malfunctions. One component of this machinery is the ring ATPase, which harnesses the energy from ATP to perform essential tasks such as maintaining protein homeostasis and ensuring genome stability—processes vital for preventing uncontrolled cell growth. Understanding precisely how these complex human ring ATPases operate and coordinate their actions remains a significant challenge. Dr. Xu’s research focuses on a mechanically similar, yet structurally simpler, ring ATPase found in the $\phi 29$ bacteriophage. By filming high-resolution “movies” of this viral ring ATPase in action using advanced single-molecule techniques, Dr. Xu aims to uncover the fundamental principles of its mechanochemical cycle. This will reveal, step-by-step, how it converts chemical energy into the precise mechanical forces and coordinated movements required to stabilize DNA. This work is relevant to a range of cancers where cellular ring ATPases are dysregulated, and the insights gained could pave the way for novel therapeutic strategies targeting these essential molecular machines. Dr. Xu received his PhD from Vrije Universiteit, Amsterdam, his MS from the University

of Chinese Academy of Sciences, Beijing, and his BS from Northeast Agriculture University, Harbin.

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