

Damon Runyon Cancer Research Foundation Awards \$4.4M to Top Young Scientists

Meet 13 new Damon Runyon postdoctoral fellows — plus five recipients of the Damon Runyon-Dale F. Frey Award for Breakthrough Scientists

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The Damon Runyon Cancer Research Foundation has named 13 new Damon Runyon Fellows, exceptional postdoctoral scientists conducting basic and translational cancer research in the laboratories of leading senior investigators. The prestigious, four-year Fellowship encourages the nation's most promising young scientists to pursue careers in cancer research by providing them with independent funding (\$300,000 total) to investigate cancer causes, mechanisms, therapies, and prevention.

The Foundation has also named five recipients of the Damon Runyon-Dale F. Frey Award for Breakthrough Scientists. This award recognizes Damon Runyon Fellows who have exceeded the Foundation's highest expectations and are most likely to make paradigm-shifting breakthroughs that transform the way we prevent, diagnose, and treat cancer. To catapult their research careers—and their impact—Damon Runyon makes an additional investment of \$100,000 in these exceptional individuals.

"The Fellowship's emphasis on innovation has inspired me to pursue bold, high-risk ideas that I might have otherwise hesitated to explore," said 2025 Dale Frey Awardee Fangyu Liu, PhD. "This ambitious approach has exceeded my expectations, yielding invaluable insights that I believe will culminate in a landmark [discovery] for the field."

2025 Recipients of the Damon Runyon-Dale F. Frey Award for Breakthrough Scientists

Rongxin Fang, PhD, Stanford University, Stanford

Assistant Professor

"Genome-scale imaging of enhancer-promoter interactions in cancer at single cell resolution"

Cells must communicate with each other to maintain homeostasis and respond to external stimuli. This communication typically occurs through chemical signals or via direct physical contact. Dr. Fang plans to develop genomic tools to understand how different types of cells communicate with each other in the healthy brain and how communication goes awry in brain tumors. His goal is to determine whether and to what extent it is possible to manipulate specific genes or pathways underlying cell-cell communication to reverse disease progression in brain cancer patients.

Xin Gu, PhD, Dana-Farber Cancer Institute and Harvard Medical School, Boston

Assistant Professor

“The midnolin-proteasome pathway catches proteins for ubiquitination-independent degradation”

Dr. Gu’s lab studies how cells regulate the destruction of proteins without using the typical “ubiquitin” tag, which signals that a protein should be transported to the proteasome for digestion and recycling of amino acids. The lab has discovered a new pathway, the midnolin-proteasome pathway, that helps degrade key proteins involved in cancer, including several linked to blood cancers like multiple myeloma. The lab’s goal is to understand this pathway better and explore how it might be used to develop new treatments, especially for blood cancers, by targeting specific proteins that drive disease.

Fangyu Liu, PhD, University of Texas Southwestern Medical Center, Dallas

Assistant Professor

“Discovery of novel ligands that treat metabolic disorders”

Dr. Liu’s research focuses on discovering new drug candidates to treat pancreatic, colorectal, breast, and prostate cancers. Using advanced computational techniques to screen billions of chemical compounds, she aims to identify and develop highly specific molecules that target critical pathways in cancer cells while sparing healthy tissues. For example, she has uncovered compounds that modulate calcium-sensing receptors, which play a role in certain cancers, with reduced side effects compared to the current standard-of-care. She is now applying these insights to improve treatments that boost immune responses against tumors. Dr. Liu’s work not only strives to create new cancer therapies but also deepen our understanding of the complex interactions within tumors, paving the way for precision medicine tailored to individual patients.

Akanksha Thawani, PhD, University of California, Berkeley

“Mechanisms of retrotransposon spread and regulation”

Dr. Thawani studies how so-called “selfish DNA” elements copy and paste themselves within the human genome. Using advanced methods such as cryo-electron microscopy to reveal the atomic structures of various molecules associated with these selfish elements, she aims to delineate their mechanism of mobility. She is also interested in understanding how selfish DNA elements are recognized and silenced within the human genome. Dr. Thawani plans to harness these discoveries to engineer new genome editing technologies to precisely insert large genes at user-specified sites in a variety of human cell types. This general technology will translate directly into new gene therapy tools that will enable treatment of loss-of-function genetic diseases, including

many cancer types, and provide a path to improving CAR-T therapies for blood cancers.

Qinheng Zheng, PhD, University of California, San Francisco

Incoming Faculty Member, Harvard Medical School, Boston

“Gain-of-function rescue of somatic mutations in cancer”

There are two key types of cancerous mutations: one that turns on growth signals too strongly, like a car with a stuck accelerator, and the other that turns off safety mechanisms, like a car with broken brakes. While some cancers can be treated with drugs that block overactive growth signals—such as Gleevec for chronic myeloid leukemia—there are currently no effective treatments for cancers caused by the loss of these safety mechanisms, also known as tumor suppressor genes. Notably, mutations in TP53, one of the most common tumor suppressor genes, are abundant in almost all cancers, including breast, lung, and ovarian cancers. Dr. Zheng’s research focuses on reactivating these impaired tumor suppressor genes, such as TP53 and FBXW7, to develop new treatment options for a wider range of cancers and to address resistance to existing therapies.

November 2024 Damon Runyon Fellows

Saket Rahul Bagde, PhD, with his sponsor Timothy A. Springer, PhD, at Boston Children’s Hospital, Boston

Most cancers develop in the epithelial tissue, which includes the skin and internal organ linings. Hemidesmosomes (HDs) are adhesive structures that anchor epithelial cells to the underlying base layer and maintain tissue integrity. While HD disassembly occurs normally during wound healing, tumor cells can exploit this process to detach and spread to other parts of the body. Dr. Bagde is studying how HD components interlock like Lego blocks to form stable HDs in healthy tissues and how they disassemble in cancerous tissues. To investigate this phenomenon, Dr. Bagde plans to develop organoids—self-organizing mini-organs grown in a petri dish to study disease progression. By creating simple base layers that simulate the supportive properties of the native organ base layer, he plans to promote the growth of both normal and cancerous organoids. This work has the potential to support the development of personalized cancer therapies based on patient-derived tumor samples. Dr. Bagde received his PhD from Cornell University, Ithaca and his MS and BS from the Indian Institute of Science Education and Research, Pune.

Longyue Lily Cao, MD, PhD, with her sponsor Jonathan C. Kagan, PhD, at Boston Children’s Hospital, Boston

Hepatocellular carcinoma (HCC) is the most common liver cancer and has one of the highest cancer-related mortality rates. Conventional cancer immunotherapies, which largely focus on enhancing T cell activity, are unfortunately effective in only a small minority of HCC patients. Though dendritic cells (DCs) are essential for T cell activation, their potential as an immunotherapeutic target remains poorly understood. Dr. Cao is investigating how a unique,

hyperactivated state of DCs can be harnessed to enhance anti-tumor immunity in a genetically engineered mouse model of HCC. Her work aims to uncover how hyperactivated DC responses generate stronger and longer-lasting protection against HCC and hopefully other cancers that are poorly responsive to conventional therapies. Dr. Cao received her MD, PhD from Albert Einstein College of Medicine, New York and her BS from Cornell University, Ithaca.

Teng Gao, PhD [HHMI Fellow], with his sponsor Vijay G. Sankaran, MD, PhD, at Boston Children's Hospital, Boston

Hematopoietic stem cells, which are found in the bone marrow and give rise to all other blood cells, maintain lifelong blood production and immune function. Due to their remarkable ability to regenerate the entire blood system, medical uses of HSCs have provided cures for many previously incurable diseases, including blood cancers. However, several unanswered questions limit our ability to fully harness their therapeutic potential for cancer treatment. What regulates HSC regeneration? Why does their function decline with age? How does HSC behavior vary in healthy individuals? Using cutting-edge single-cell analyses and computational biology, Dr. Gao aims to identify the molecular and cellular factors involved in HSC regeneration, as well as possible targets for enhancing their regenerative potential. This work could enable significant improvements in stem cell-based therapies for cancer treatment. Dr. Gao received his PhD from Harvard University, Cambridge and his BS from Washington University, St. Louis.

Rodrigo Gier, PhD [HHMI Fellow], with his sponsor Kevan M. Shokat, PhD, at University of California, San Francisco

Drug therapies that selectively target proteins that drive the growth of tumor cells are rapidly becoming the standard of care for many cancers. However, tumors are often able to evade inhibition by targeted anti-cancer drugs by activating other proteins, leading to drug resistance. Dr. Gier is developing a new therapeutic approach that repurposes existing drugs to release highly toxic cargoes, known as payloads, that aggregate in drug-resistant cancer cells and kill them. As a general platform, it is applicable to a wide range of solid and liquid cancers. Dr. Gier received his PhD from University of Pennsylvania, Philadelphia and his BA from Swarthmore College, Swarthmore.

Hasreet K. Gill, PhD [HHMI Fellow], with her sponsor Bonnie L. Bassler, PhD, at Princeton University, Princeton

Dr. Gill is studying cell-cell communication via quorum sensing in developing biofilms. Biofilms are communities of bacteria that take on a three-dimensional structure and often develop striking visual features like wrinkles. Resident bacteria exploit this complexity to resist antimicrobial treatments and cause disease, particularly in healthcare settings, where biofilms pose serious threats to immunocompromised chemotherapy patients. Quorum sensing is the process by which bacteria orchestrate collective behaviors, including the assembly and dissolution of biofilm communities. Using quantitative microscopy at single-cell resolution, Dr. Gill is investigating how these signals between bacteria result in the construction of spectacular 3D biofilms at the air-liquid interface. A deeper understanding of how bacteria thrive in dynamic environments will contribute to new strategies to combat infections, which will positively impact treatments for all

cancer types. Dr. Gill received her PhD from Harvard University, Cambridge and her BS from Drexel University, Philadelphia.

Dhiraj Indana, PhD, with his sponsor Michael B. Elowitz, PhD, at California Institute of Technology, Pasadena

Successful immune responses against cancer require immune cells of various types to control each other's proliferation, differentiation, and death. These interactions collectively constitute a set of intercellular signaling circuits. A fundamental challenge in cancer research is to understand the relationship between the architecture and functions of these circuits. Dr. Indana will quantitatively model and synthetically construct intercellular circuits with immune-like functions to understand how circuit architectures empower immune behaviors such as threat detection and cancer elimination while maintaining the ability to return to a non-inflammatory state. This work promises to uncover the "design principles" underlying various immune functions, providing a foundation for engineering novel immunotherapies. Dr. Indana received his PhD and MS from Stanford University, Stanford and his BTech from Indian Institute of Technology, Roorkee.

Kashish Jain, PhD, with his sponsor Valerie M. Weaver, PhD, at University of California, San Francisco

A majority of pancreatic cancer cases harbor a mutation in the KRAS gene, which is involved in cancer initiation, progression, and chemotherapy resistance. Drugs targeting KRAS mutations are often met with resistance due to limited drug penetration into the tumor. Since pancreatic cancer progression involves increased tissue stiffening, KRAS signaling might be controlled by tissue stiffness. Dr. Jain is studying the mechanisms that underlie tissue stiffness-dependent KRAS signaling at the molecular level. Understanding these mechanisms will uncover new ways to block aberrant KRAS signaling or reduce the effects of tissue stiffness on cancer progression, ultimately informing new combination therapies with KRAS-targeting drugs. Dr. Jain received his PhD from the National University of Singapore, Singapore and his BTech and MTech from Indian Institute of Technology, Kanpur.

Sangin Kim, PhD, with his sponsor Roger A. Greenberg, MD, PhD, at University of Pennsylvania, Perelman School of Medicine, Philadelphia

The cellular response to DNA damage is coordinated by an enzyme known as ATM kinase. Mutations in ATM are found in approximately 1% of the population and contribute to an increased risk of both hereditary and sporadic cancers, including breast cancer. Dr. Kim's research investigates how ATM suppresses the production of double-stranded RNAs (dsRNAs) in response to DNA damage. These dsRNAs play a critical role in tumor progression. Dr. Kim aims to identify the key molecular players involved in ATM-mediated suppression of dsRNAs and elucidate how the loss of ATM function triggers inflammatory responses through dsRNA sensing pathways. By uncovering these mechanisms, Dr. Kim aims to deepen our understanding of how ATM mutations drive cancer development and uncover novel therapeutic strategies for ATM-associated cancers. Dr. Kim received his PhD and BS from the Ulsan National Institute of Science and Technology, Ulsan.

Antonio J. LaPorte, PhD, with his sponsor Eric N. Jacobsen, PhD, at Harvard University, Cambridge

Sugar molecules on the surface of cells can be arranged in many different patterns, and it is the exact structure of a sugar that gives it its unique function. In sugars on the surface of cancer cells, the location of sulfate (SO₄⁻) groups changes as the tumor grows and spreads. The study of sulfated sugars has been stymied, however, by the difficulty of synthesizing them in the laboratory. Dr. LaPorte's research aims to remove this roadblock by developing a chemical reaction that easily constructs biologically important sugars with a sulfate group bound to them. These sulfated sugars will then be used to test how specific sulfation patterns affect their interaction with other biological proteins. The results of these experiments will lay the groundwork for new cancer treatments that use complicated sugar structures to selectively target cancer cells without harming healthy cells. Dr. LaPorte received his PhD from University of Illinois, Urbana-Champaign, Champaign and his BS from North Central College, Naperville.

Anita Reddy, PhD [Connie and Bob Lurie Fellow], with her sponsor Justin L. Sonnenburg, PhD, at Stanford University School of Medicine, Stanford

The Western diet, characterized by low dietary fiber and high saturated fat content, is strongly correlated with incidence of CRC. This diet rapidly alters the composition and function of the gut microbiome, inducing microbial imbalance and inflammation, two major hallmarks of CRC. These changes are thought to be caused by diet-induced oxygenation of the gut environment. Most beneficial gut bacteria cannot survive this oxygenated environment, but harmful microbes can thrive in this setting and further exacerbate inflammatory responses. The response of bacteria to oxygen is varied and poorly understood. Dr. Reddy aims to elucidate the mechanism underlying oxygen tolerance in bacteria and find interventions to restore normal oxygen levels. The findings from this project will delineate the contributions of exercise and diet to establishing the oxidative environment of the gut and illuminate the mechanism allowing certain microbes to withstand redox stress. Dr. Reddy received her PhD from Harvard University, Cambridge and her BS from University of Connecticut, Mansfield.

Ariën Schiepers, PhD [HHMI Fellow], with his sponsor Alexander Y. Rudensky, PhD, at Memorial Sloan Kettering Cancer Center, New York

T lymphocytes, an important component of the immune system, recognize infected or cancerous cells with great specificity, ensuring targeted elimination. These potent cells are kept in check by regulatory T cells, the guardians of the immune system. While essential for curtailing excessive inflammation and preventing autoimmunity, their immunosuppressive properties can promote the development and progression of cancer. Regulatory T cells are distinguished by the presence of a protein called Foxp3, which plays a critical role in their differentiation, function and fitness. Foxp3 deficiency results in fatal autoimmune inflammatory disease, underscoring its importance for maintaining organismal health. Despite its significance, however, the reliance of regulatory T cells on Foxp3 in disease contexts like infection and cancer remains incompletely understood. Dr. Schiepers will study the fate and function of regulatory T cells in these settings using mouse genetics approaches and disease models of melanoma and colorectal cancer. Dr. Schiepers received his PhD from The Rockefeller University, New York and his MS and BS from Utrecht University, Utrecht.

Sue Im Sim, PhD [Connie and Bob Lurie Fellow], with her sponsor Orion D. Weiner, PhD, at University of California, San Francisco

To power directional movement, cells build dynamic sheet-like protrusions at their leading edge. How individual molecules are coordinated to produce these changes in cell morphology is poorly appreciated. Dr. Sim uses immune cell migration as a model system to investigate the self-organization of a protein assembly known as the WAVE complex, which facilitates the formation of these protrusions in migratory cells. Her work will harness recent advances in electron microscopy and protein prediction and design to study the mechanism of the WAVE complex. As a critical player in cell migration, the dysregulation of the WAVE complex is associated with tumor cell invasion and metastasis in several cancer types. This aberrant migration enables cancer cells to travel to and infiltrate adjacent tissue sites. Understanding the fundamental mechanisms of cell migration can thus better inform the development of therapeutics that limit the progression of cancer. Dr. Sim received her PhD from the University of California, Berkeley and her BA from Bowdoin College, Brunswick.

Yichi (Tony) Zhang, PhD, with his sponsor Matthew Vander Heiden, MD, PhD, at Massachusetts Institute of Technology, Cambridge

Cancer cachexia, characterized by progressive muscle wasting and weight loss in cancer patients, is a common and multifaceted syndrome that negatively impacts patient quality of life. Cachexia has no available treatments to date, due to insufficient knowledge about the underlying mechanisms. Cachexia is particularly prominent in pancreatic cancer patients. Previous work from the Vander Heiden lab has identified that there is diminished secretion of pancreatic enzymes due to the pancreatic cancer, which contributes to tissue wasting because breakdown of muscle tissue can release amino acids into circulation. Dr. Zhang is interested in understanding whether amino acids released from muscle are fueling tumor growth or sustaining the host organism. Specifically, as circulating amino acids are often used by the liver to produce glucose, Dr. Zhang wants to study how liver glucose metabolism is affected in pancreatic cancer cachexia. This work seeks to improve the quality of life of cancer patients by reducing cachexia while seeking to understand how cancer impacts the whole-body metabolism of the host. Dr. Zhang received his PhD from University of Texas Southwestern Medical Center, Dallas, his MS from Carleton University, Ottawa, and his BScH from Queen's University, Kingston.

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