

Damon Runyon Cancer Research Foundation Awards \$4.5M to Top Young Scientists

The four-year fellowship encourages the nation's most promising young scientists to pursue careers in cancer research.

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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 six 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 as they prepare to transition to independence.

2026 Recipients of the Damon Runyon-Dale F. Frey Award for Breakthrough Scientists

Fangtao Chi, PhD, Massachusetts Institute of Technology, Cambridge

“Dietary nutrients and metabolism shape intestinal regeneration and tumorigenesis”

Dr. Chi's research examines how dietary nutrients and cellular metabolism influence intestinal regeneration and tumorigenesis. The intestine renews itself rapidly and relies on intestinal stem cells to repair damage after infection, inflammation, or cancer therapy. Dr. Chi is discovering that specific nutrients can act as signals that boost stem cell activity and accelerate tissue repair. But these same regenerative programs can also be hijacked to support abnormal growth and increase the likelihood of tumor formation under certain conditions. Dr. Chi plans to systematically test nutrients and metabolic pathways to understand when and how diet promotes repair versus when it inadvertently promotes tumorigenesis. Ultimately, this work aims to provide a foundation for designing dietary regimens that improve tissue repair and resilience while reducing the potential

for intestinal cancers, including colorectal cancer.

Cayla E. Jewett, PhD, University of Colorado, Denver Anschutz Medical Campus

“Regulation of programmed DNA damage to drive centriole amplification in multiciliated cells”

Cilia are small, hairlike protrusions found on the surface of cells; some cells in the lining of our organs have many dozens of cilia, which beat in a coordinated manner to drive fluid across the tissue. These multiciliated cells escape many guidelines governing normal cellular behavior. For example, too many copies of a cellular organelle called a centriole can drive tumorigenesis, but multiciliated cells require extra centrioles for normal function. In addition, cancer cells often hijack the DNA damage pathway for survival, but Dr. Jewett has found that multiciliated cells utilize the DNA damage pathway for their development. Given these parallels, how multiciliated cells refrain from becoming cancerous remains a mystery. Dr. Jewett will investigate the multiciliated cell system to understand how centriole overproduction and DNA damage are controlled to prevent tumorigenesis, with the hope of discovering new targetable molecules for cancer therapeutics.

Titas Sengupta, PhD, Princeton University, Princeton

“Epigenetic regulation of neuronal function in response to aging and the environment”

Histones are proteins that provide structural support for chromosomes, and modifications of histones impact gene expression without altering DNA sequence (i.e., epigenetically). Histone modifications play essential roles in normal physiology and are dramatically altered in cancer across tissue types, including the nervous system. Dr. Sengupta is investigating how histone modifications in neurons regulate neuronal functions such as learning and memory. For example, she has discovered a mechanism in the roundworm nervous system by which gene expression changes rapidly to impact short-term memory, demonstrating that even short-term memory is influenced by dynamic gene expression rather than pre-existing proteins. Dr. Sengupta’s work aims to uncover how histone modifications are regulated, targeted to specific genes, and influenced by external and internal cell states, in order to better understand mechanisms of epigenetic dysregulation of gene expression in cancer.

Dylan M. Parker, PhD, University of Colorado, Boulder

“Stress granule regulators and their roles in cancer progression”

Dr. Parker studies the role of molecular assemblies known as stress granules that form when cells are exposed to stressful conditions. The assembly of stress granules upon cellular insult is thought to regulate gene expression and modulate cell survival. Notably, stress granules are present in various cancers, and many chemotherapeutic treatments lead to the formation of stress granules. Dr. Parker aims to determine the mechanisms regulating stress granule assembly and disassembly to understand how stress granule formation supports the development of cancer and chemotherapy-resistant tumors. This research has the potential to discover novel targets to treat cancers as well as sensitize chemotherapy-resistant cancers to existing treatments.

Catherine Triandafillou, PhD, University of Pennsylvania, Philadelphia “Illuminating error correction strategies in early development”

When an organism is developing, it must correct mistakes that might occur at the level of individual cells or tissues. Dr. Triandafillou aims to better understand how error correction systems work, and why they might not work in cases like cancer. To explore these developmental questions, she uses what are called gastruloids, 3D clusters of stem cells that can organize themselves and transform into the basic building blocks of an organism. She has developed a method using microscopy to trace the history of these cells and measure how much their past state influences what they become. Dr. Triandafillou seeks to illuminate how differences in individual cells might impact what those cells eventually turn into, and how such differences affect the correction of mistakes—like abnormal growth, bias in cell types, or missing cell types—as well as how the cells around an error react to it.

Youngmu (Nick) Shin, PhD, University of California, San Francisco “Rewiring cell-cell communication using engineered scaffold proteins”

Cells in our bodies constantly talk to each other, and this cell-to-cell communication controls important processes such as how our immune system detects and kills cancer cells. It is not well understood, however, how cells build the tiny, specialized contact sites where this communication happens. Dr. Shin hypothesizes that many of these contact sites, called synapses, are organized by scaffold proteins that come together into droplet-like clusters, called condensates, to facilitate cell-to-cell communication. Dr. Shin is testing this hypothesis by building artificial versions of this cell-cell interface. He engineers simple, synthetic proteins that act as scaffolds, uses them to create a “synthetic synapse” between cells, and then studies how changing these scaffolds alters the shape and strength of the connection. He plans to combine these experiments with simulations that model how the building blocks of these scaffolds behave and cluster. By stripping cell-cell contacts down to their essential parts, this work aims to uncover the physical rules that cells use to organize communication sites. In the long term, these principles could be used to design immune cells—such as engineered T cells for cancer therapy—that form more precise connections with diseased cells while sparing healthy tissue.

November 2025 Fellows

Duaa H. Al-Rawi, MD, PhD [The Mark Foundation for Cancer Research Fellow], with her sponsors Scott W. Lowe, PhD, and Sohrab P. Shah, PhD, at Memorial Sloan Kettering Cancer Center, New York

High-grade serous ovarian cancer (HGSC) is the most common and deadly form of ovarian cancer, largely because it is often found after it has spread. HGSCs arise in the fallopian tube, where tiny precancerous changes can exist for years before a tumor is detected. Dr. Al-Rawi is investigating what early genetic changes cause a normal fallopian-tube cell to become HGSC. She focuses on two events that appear very early and in most cases: loss of the protective p53 “guardian” pathway and chromosomal instability, in which cells repeatedly gain or lose large pieces of DNA. Using precise genome editing to model specific p53 mutation types, mouse models that track how altered cells expand over time, and single-cell profiling of rare human precursor samples, Dr. Al-Rawi will test how which early changes predict progression. By defining these earliest steps, the

work supports new strategies for risk stratification, early detection, and prevention for HGSC and related serous cancers. Dr. Al-Rawi received her PhD from Massachusetts Institute of Technology, Cambridge, her MD from Stanford University, Stanford, and her BS from Kansas State University, Manhattan.

Tatsat Banerjee, PhD [HHMI Fellow], with his sponsor Ronald D. Vale, PhD, at Whitehead Institute for Biomedical Research, Cambridge

CAR T cell therapy, which involves genetically engineering a patient's own immune cells to seek and destroy cancer, has revolutionized the treatment of certain blood cancers. However, it frequently performs poorly against solid tumors because T cells become exhausted or cannot effectively detect the cancer cells. Dr. Banerjee aims to learn the fundamental molecular, genetic, and biophysical rules of internal signaling in T cells. By decoding these rules, he aims to design next-generation CAR T cells with enhanced sensitivity, persistence, and versatility. Taking an atypical approach, he will combine multiple cutting-edge technologies to dissect and engineer the "immunological synapse," the connection that forms between a T-cell and a tumor cell, to ultimately tune the function of the T cells. This study aims to overcome current limitations in treating leukemias and extend the success of immunotherapy to solid tumors, specifically melanoma. Dr. Banerjee received his PhD from Johns Hopkins University, Baltimore, his MTech from the Indian Institute of Technology, Kanpur, and his BEng from Jadavpur University, Kolkata.

Elizabeth Black, PhD, with her sponsor Iain Cheeseman, PhD, at Whitehead Institute for Biomedical Research, Cambridge

One hallmark of cancer is dysregulation of core cellular processes, including protein production or translation. One crucial element of translation is determining where protein production begins, a process called translation start site selection. It is estimated that a third of human genes contain multiple translation start sites, but because they are invisible in standard experiments, it is not understood how they change in diseases like cancer. Dr. Black aims to identify how leukemia and lymphoma cells control translation start site selection and how this impacts their behavior and disease outcomes. By uncovering how translation control is altered in blood cancers, this research can reveal fundamental insights into cancer biology that have been previously overlooked. Dr. Black received her PhD from Yale University, New Haven, and her BS from Wake Forest University, Winston-Salem.

Sarah W. Cai, PhD [HHMI Fellow], with her sponsors Yifan Cheng, PhD, and David Julius, PhD, at the University of California, San Francisco

Cancer-associated pain can arise directly from tumor growth or as a side effect of chemotherapy drugs. First-line cancer treatments, such as cisplatin and paclitaxel, contribute to pain hypersensitivity by increasing the activity and expression of TRP ion channels—the receptors that detect painful stimuli in sensory neurons and trigger pain sensation. Research thus far has focused on how these receptors look and behave at a single-molecule level (one copy) or at a cellular/organismal level (many thousands of copies per neuron). However, TRP channels are also

proposed to operate in nanoscale clusters (tens of copies) that amplify signaling within a sensory neuron. Dr. Cai's research will use state-of-the-art microscopy techniques alongside biochemical and cell-based approaches to study how receptors are organized on the surface of sensory neurons. She aims to understand how inflammation and injury, including toxicity from chemotherapy drugs, contribute to acute and chronic pain. This work will provide fundamental insights into pain signaling that can inform the development of new pain management strategies. Dr. Cai received her PhD from The Rockefeller University, New York, and her BS from California Institute of Technology, Pasadena.

Esther J. Han, PhD [Robert Black Fellow], with her sponsor Andrew L. Goodman, PhD, at Yale University

Our diets include myriad small molecules known as xenobiotics, which are mainly derived from plants, that influence cancer prevention and progression. When plants experience infection, they chemically modify these compounds to improve disease resistance, but whether parallel processes exist in mammals remains unknown. Given the strong association between inflammation and cancer development, and based on preliminary data indicating that inflammation-induced xenobiotic modifications can occur in the mammalian gut, Dr. Han will dissect how the gut microbiome and the host transform dietary xenobiotics to alter their function during health and inflammation. The findings from these studies will lay foundations to improve health through nutritional interventions. Dr. Han received her PhD from Princeton University, Princeton, and her BS from the University of California, Berkeley.

Qixiang He, PhD [HHMI Fellow], with his sponsor Samuel H. Sternberg, PhD, at Columbia University, New York

Our immune system uses many strategies to defend against viruses. Recent studies have uncovered a surprising bacterial antiviral strategy—instead of cutting DNA to destroy it, this system builds new DNA molecules to stop infections. Dr. He's project will focus on understanding how this bacterial defense system works and explore whether this system can be repurposed to safely make DNA inside human cells. This could offer a new way to address a major challenge in the development of gene therapy and cancer immunotherapy more broadly: safely and efficiently delivering DNA into target cells without triggering harmful immune reactions. Dr. He received his PhD from the University of Wisconsin-Madison, Madison, and his BS from the University of Chinese Academy of Sciences, Beijing.

King L. Hung, PhD [HHMI Fellow], with his sponsor Ardem Patapoutian, PhD, at The Scripps Research Institute, La Jolla

Cancer is fundamentally a disease of lost tissue integrity, in which cells fail to properly coordinate and regulate one another, leading to abnormal cell growth and invasion within tissues. Dr. Hung aims to uncover basic principles of how cells combine chemical and mechanical signals to maintain tissue integrity. Using flatworms that are capable of tissue regeneration as a model, he will employ live whole-worm imaging of tissue regeneration to study mechanical and chemical

signaling at the cellular level. This project will provide key insights about the logic of multicellular signaling circuits for maintaining normal tissue integrity and clues about how these signaling circuits can be dysregulated in cancer. Dr. Hung received his PhD from Stanford University, Stanford, and his BS from the University of Washington, Seattle.

Jinho D. Jeong, PhD [Robert Black Fellow], with his sponsor Liron Bar-Peled, PhD, at Massachusetts General Hospital, Boston

Cancer cells often rely on specific protein complexes—groups of proteins that work together—to grow and survive. Dr. Jeong is using a new chemical technology called Molecular COUPLRs to identify and disable these disease-related protein complexes in lung cancer. This approach is especially relevant for non-small cell lung cancer, as well as for lung cancer brain metastases, which remain the leading cause of death for patients. By mapping which protein complexes each tumor depends on and finding ways to selectively disrupt them, Dr. Jeong aims to uncover new vulnerabilities in lung cancer and help lay the foundation for more effective treatments. Dr. Jeong received his PhD from Stanford University, Stanford, and his BS from Seoul National University, Seoul.

Wenbin Mei, PhD [Robert Black Fellow], with his sponsors James T. Neal, PhD (Broad Institute of MIT and Harvard), and Eliezer M. Van Allen, MD (Dana-Farber Cancer Institute), at the Broad Institute of MIT and Harvard, Cambridge

While much has been uncovered about the specific mutations that arise within a tumor, it is not fully understood how the DNA a person is born with (their inherited genetics) influences how those tumors grow. Dr. Mei's research focuses on a specific, aggressive cancer gene called ERBB2, which is responsible for many breast, lung, and stomach cancers. Dr. Mei aims to discover if a patient's inherited genetics makes them more likely to develop these specific tumor mutations or makes the cancer more dangerous. By understanding the interaction between a patient's natural DNA and their tumor's DNA, we can better predict cancer risks and find more effective, personalized treatments. Dr. Mei received his PhD from The Rockefeller University, New York, and his BS from Peking University, Beijing.

Rishi Kumar Mishra, PhD [Robert Black Fellow], with his sponsor Morgan E. DeSantis, PhD, at the University of Michigan, Ann Arbor

Metastasis, the spread of cancer cells from primary tumors to healthy tissues, accounts for over 90% of cancer-related deaths. A protein called dynein is crucial for cell movement and research indicates that inhibiting dynein can reduce breast cancer cell spread. Typically, dynein moves toward the cell nucleus along microtubules from the plus-end (typically near the cell periphery) to the minus-end (usually near nucleus). However, during cell migration, dynein congregates at the microtubule's plus-end by a process that is poorly understood. Dr. Mishra aims to establish the molecular mechanism underlying dynein's localization at the plus-end of the microtubule. This understanding will help elucidate how dynein facilitates cell migration and metastasis, potentially leading to new cancer treatment strategies applicable to various cancer types. Dr. Mishra received

his PhD from the Indian Institute of Science, Bengaluru, and his BS from Banaras Hindu University, Varanasi.

Christian G. Peace, PhD [Ludwig Institute for Cancer Research Fellow], with his sponsor Joshua D. Rabinowitz, MD, PhD, at Princeton University, Princeton

Cancer cells and certain immune cells inside tumors need a lot of energy to survive and function, creating a kind of “tug-of-war” for nutrients in the tumor’s environment. However, until recently, there has not been a good way to measure how these cells use nutrients for energy inside a living tumor. To tackle this challenge, Dr. Peace developed a new technology that can track which nutrients power a key energy pathway—the TCA cycle—in both cancer cells and immune cells, directly in vivo in tumors. By uncovering these details, his work aims to improve how we design cancer treatments, especially immunotherapies that help the immune system fight cancer more effectively. This work has the potential to be relevant for all cancers. Dr. Peace received his PhD and BA from Trinity College, Dublin.

Juntao Yu, PhD [HHMI Fellow], with his sponsor Yukiko M. Yamashita, PhD, at Whitehead Institute for Biomedical Research, Cambridge

Asymmetric cell division is a mechanism by which adult stem cells generate one self-renewing stem cell and one differentiating daughter cell in order to maintain tissue homeostasis. A fundamental unsolved question is how two daughter cells adopt distinct cell fates (e.g., how one becomes a blood cell and one becomes a blood vessel cell) to prevent unchecked proliferation and tumorigenesis. Using fruit flies as a model system, Dr. Yu will investigate the selective inheritance of chromosomes for cell fate decision during asymmetric divisions of stem cells. This work will reveal a fundamental principle of how stem cells use chromatin-based mechanisms to determine cell fate and maintain homeostasis. Moreover, it will provide key insights into how cancer cells hijack these pathways to sustain their immortality and uncover novel vulnerabilities in cancer that could be targeted therapeutically. Dr. Yu received his PhD from Harvard University, Cambridge, and his BS from the University of Science and Technology of China, Hefei.

Ming M. Zheng, PhD [Robert Black Fellow], with his sponsor Paul C. Blainey, PhD, and C. Sam Peng, PhD, at the Broad Institute of MIT and Harvard, Cambridge

Many cancer treatments kill healthy cells along with cancer cells and tumors frequently adapt to treatment and build resistance. These challenges exist because the most important pathological cancer processes occur through complex interactions inside living cells, and the current models used to study cancer cannot fully mimic these complex living interactions. Dr. Zheng aims to combine large-scale genetic screening, advanced single-molecule imaging, and AI modeling to create detailed maps of how cancer-driving genes behave inside living human cells. These maps will show how networks of genes, as well as small DNA changes, alter the real-time behavior of powerful cancer drivers. This work will guide the development of treatments that cause less harm, stay effective longer, and act with far greater precision. Dr. Zheng received his PhD from Massachusetts Institute of Technology, Cambridge, and his BS from Peking University, Beijing.

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