

Damon Runyon Cancer Research Foundation Awards \$4.2M to Clinical Cancer Researchers

Meet the five new Damon Runyon Clinical Investigators and learn about the projects of these early career physician-scientists.

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The Damon Runyon Cancer Research Foundation has named five new Damon Runyon Clinical Investigators. The recipients of this prestigious award are outstanding, early-career physician-scientists conducting patient-oriented cancer research at major research centers under the mentorship of the nation's leading scientists and clinicians.

The Clinical Investigator Award program was designed to increase the number of physician-scientists capable of translating discoveries in the lab into new treatments for cancer patients. Each Awardee will receive \$600,000 over three years, as well as assistance with research costs such as the purchase of equipment. Because the need to repay medical school loans is often cited by physicians as a deterrent to pursuing research, Damon Runyon will also retire up to \$100,000 of medical school debt.

The Foundation also awarded Continuation Grants to three Damon Runyon Clinical Investigators, totaling \$1,000,000 in additional funding. The Continuation Grants are designed to support Clinical Investigators who are approaching the end of their original award and need more time to work on a promising avenue of research or a clinical trial.

This program is possible through the support of the William K. Bowes Jr. Foundation. Through partnerships with generous donors, industry sponsors, and its Accelerating Cancer Cures initiative, the Damon Runyon Cancer Research Foundation has invested over \$88 million to support the careers of 136 Clinical Investigators across the United States since 2000.

2025 Clinical Investigators

Jonathan Chou, MD, PhD, with mentor James A. Wells, PhD, at University of California, San Francisco

Nearly all of the FDA-approved therapies in the last decade for bladder cancer target cell surface

proteins. Despite enormous progress in targeted therapy development, however, only five unique targets (out of thousands) have been explored. In addition, because current tumor targets are often also expressed on normal tissues, toxic side effects are common and can even be life-threatening. Therefore, identifying cancer-specific, targetable proteins is critical to enhancing efficacy and safety of bladder cancer drugs. In this project, Dr. Chou will utilize a new technique to identify novel drug targets from patient tumor samples, develop molecules that bind them, and engineer these molecules into cellular therapies. He will also evaluate a strategy to target a surface protein called CDCP1 and explore the role of several proteases (enzymes that break down proteins) in therapy resistance. Dr. Chou hopes that his work will reveal a new class of targetable surface proteins for bladder cancer and pave the way for future clinical trials.

Bridget P. Keenan, MD, PhD, with mentors Matthew H. Spitzer, PhD, and Lawrence Fong, MD (Fred Hutchinson Cancer Center), at University of California, San Francisco

Hepatocellular carcinoma (HCC), a type of liver cancer often caused by liver disease related to viral infections or metabolic disease, is a leading cause of cancer deaths globally. Treating HCC with immunotherapy and targeted therapies shows promise, but liver damage can make these treatments challenging to administer and less effective. Dr. Keenan's preliminary data suggest that certain immune cells, known as myeloid cells, become suppressive in patients with HCC and worsen liver function. However, it is possible that the correct combinations of immunotherapy treatments could partially reverse this myeloid cell suppression and result in better outcomes for patients with HCC. Dr. Keenan will focus on understanding exactly how liver disease affects the immune system and finding ways to counteract the suppressive effects of myeloid cells. By studying blood samples and liver tissues from patients with HCC undergoing immunotherapy treatment, she aims to identify the best combinations to enhance the immune system's ability to fight liver cancer. This research could lead to new, more effective treatments for patients with liver cancer, potentially improving survival rates and quality of life.

Peter G. Miller, MD, PhD, with mentors Timothy A. Graubert, MD, and David T. Scadden, MD, at Massachusetts General Hospital, Boston

The goal of Dr. Miller's research is to determine how mutations in blood cells give rise to pre-malignant blood conditions such as clonal hematopoiesis (CH), which drive the development of blood cancers. To this end, Dr. Miller will study patients with rare inherited diseases and use experimental models in the laboratory. He ultimately seeks to use the data generated through this research to develop new strategies to predict, prevent, and treat highly lethal blood cancers.

Srivatsan Raghavan, MD, PhD, with mentors William C. Hahn, MD, PhD, and Brian M. Wolpin, MD, MPH, at Dana-Farber Cancer Institute, Boston

Pancreatic cancer is a highly lethal disease with relatively few treatment options. A new class of inhibitors that target the KRAS gene, which is altered in approximately 90% of pancreatic cancer patients, are showing great promise in the clinical setting as a new therapeutic option for these patients. However, nearly all patients develop resistance and experience tumor regrowth after a relatively short period of treatment with these drugs. Dr. Raghavan aims to investigate how cancer cells adapt and become resistant to these KRAS inhibitors and develop combination therapies to

overcome this resistance. He anticipates that these studies will uncover fundamental mechanistic insights into cancer drug resistance and identify novel therapeutic strategies that will improve outcomes for patients with pancreatic cancer.

Tanaya Shree, MD, PhD, with mentor Brian J. Druker, MD, at Oregon Health and Science University, Portland

T-cell engaging bispecific antibodies, which bring T cells close to tumor cells and induce them to kill the tumor cell, are a new class of immunotherapy that have demonstrated efficacy in lymphoma and myeloma and are now in development for many other cancers. In diffuse large B cell lymphoma, bispecific antibodies have proven very effective, but approximately 60% of patients derive no long-term benefit. Dr. Shree is working to understand the requirements for generating an effective bispecific antibody response in patients. This knowledge could result in novel improved treatment approaches for patients with lymphoma and inform the design of bispecific T cell-engaging strategies for other types of tumors.

2025 Continuation Grantees

Daniel J. Delitto, MD, PhD, with mentor Michael T. Longaker, MD, DSc, at Stanford University, Stanford

Pancreatic cancer develops in the midst of intense scarring and fibrous connective tissue (fibrosis). The architects of this scarring are cells called fibroblasts, known to fuel cancer growth and promote treatment resistance. Dr. Delitto's research is focused on the interface between cancer-induced fibrosis and the immune system. He has shown that fibroblasts play a significant role in shielding cancer cells from immune cells. By altering how fibroblasts sense tissue damage, Dr. Delitto has uncovered a mechanism that reactivates the immune system to fight the tumor. He aims to further develop these findings into a novel immunotherapy regimen for pancreatic cancer.

Nathan Singh, MD [Bakewell Foundation Clinical Investigator], with mentor John F. DiPersio, MD, PhD, at Washington University, St. Louis

Chimeric antigen receptor (CAR) T cell therapy, in which a patient's own immune cells are engineered to target their cancer, has changed the treatment landscape for many blood cancers. Despite promising early results, however, long-term follow-up has revealed that nearly half of patients treated with CAR T cells eventually experience cancer recurrence. Using a variety of techniques in cell lines and patient samples, Dr. Singh aims to understand how interactions between engineered T cells and blood cancer cells in some cases lead to long-term remission, and in others to therapeutic failure. The broad goals of his lab are to understand the biological signals that cause these therapies to fail, and to use this knowledge to design next-generation immunotherapies that can cure more patients.

Aaron D. Viny, MD, with mentors Emmanuelle Passegué, PhD, and Joseph G. Jurcic, MD, at Columbia University, New York

Up to 50% of patients with acute myeloid leukemia (AML) have a genetic alteration called DNA

methylation, in which a carbon methyl group is added to the DNA molecule, typically turning the methylated gene “off.” A mainstay of therapy is the use of hypomethylating agents, which prevent the copying of these modifications during cell division, but this therapy is effective in only 20–30% of patients. Using chemical and genetic manipulation in mouse bone marrow, Dr. Viny aims to determine the effect of DNA methylation on the ability of specific regions of the genome to be accessible to proteins involved with gene expression and other regions to be inaccessible and “silenced.” In a prospective phase II clinical trial, he will treat relapsed AML patients with dual hypomethylating agents. By studying these patients’ genetic profiles, he aims to determine the genetic features that contribute to therapy response, paving the way for more effective interventions to be developed for patients with acute myeloid leukemia. Dr. Viny was previously a Damon Runyon Fellow.

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