

Six Damon Runyon Clinical Investigators Awarded \$5.2M for Cancer Research

Recipients of the Damon Runyon Clinical Investigator Award are early career physician-scientists conducting patient-oriented cancer research.

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The Damon Runyon Cancer Research Foundation has named six 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 physicians capable of translating scientific discoveries 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 as a deterrent to pursuing research, Damon Runyon will also retire up to \$100,000 of medical school debt owed by the awardee.

The Foundation also awarded Continuation Grants to four Damon Runyon Clinical Investigators for an additional two years of funding, totaling \$400,000 each. 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.

Physician-scientists have an important role to play in public health research as well.

"In addition to Damon Runyon's funding of basic science, funding projects that are more translational or epidemiologic in their focus is really important," says 2024 Continuation Grantee Kelly L. Bolton, MD, PhD. "Looking at population databases to try to understand cancer risk and prevention, for example—it's really helpful when physicians are involved."

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 committed millions to support the careers of physician-scientists across the United States since 2000.

2024 Clinical Investigators

Steven M. Corsello, MD [Leslie Cohen Seidman Clinical Investigator], with mentors Nathanael S. Gray, PhD, and Ronald Levy, MD, at Stanford University School of Medicine, Stanford

Pancreatic cancer is a devastating disease with limited treatment options. New strategies are urgently needed, but few actionable therapeutic targets are known. By systematically testing diverse molecules against pancreatic cancer cells combined with gene knockout studies, Dr. Corsello has identified a starting point to simultaneously activate inflammatory signaling and cell death pathways. He will determine the efficacy and underlying molecular mechanism of this approach, and potential immunotherapy combinations, using patient-derived tumor models. His goal is to accelerate the development of more effective and less toxic therapies for pancreatic cancer.

Megan L. Insco, MD, PhD, with mentor F. Stephen Hodi, MD, at Dana-Farber Cancer Institute, Boston

Gene expression is a complex process, and sometimes mistakes are made, resulting in the generation of aberrant or “junk” RNAs. Dr. Insco previously discovered that cellular failure to “clean up” this junk RNA can contribute to the development and progression of melanoma. Her work is now focused on targeting aberrant RNA to treat cancer. First, she will identify compounds that specifically target melanomas that are unable to clean up their junk RNAs. Second, she will investigate how immune cells can be activated to attack melanoma cells that have high levels of aberrant RNAs. Many advances in our understanding of RNA biology over the last four decades have resulted in new therapies for patients. As this area of RNA biology is almost completely unexplored, Dr. Insco anticipates that studying mechanisms of aberrant RNA oncogenesis will reveal new therapeutic strategies for patients.

Mary M. Mullen, MD, with mentors Dineo Khabele, MD, and Nima Mosammaparast, MD, PhD, at Washington University, St. Louis

Only up to 20% of patients with advanced ovarian cancer will survive five years after diagnosis. This is largely due to the cancer’s resistance to traditional chemotherapy and the current lack of targeted therapies that work with chemotherapy to improve response. Dr. Mullen’s lab has identified a new target, COP9 Signalosome Subunit 5 (COPS5), to treat ovarian cancer. Her team has found that inhibiting COPS5 with a drug called CSN5i-3 drastically improves ovarian cancer response to chemotherapy. She now aims to test the effectiveness of CSN5i-3 and chemotherapy against patient-derived, therapy-resistant ovarian cancer tumors. She will also investigate the mechanism of COPS5, believed to be involved in the repair of DNA damage caused by chemotherapy. Dr. Mullen hopes this innovative target will transform the care of patients with ovarian cancer.

Erin M. Parry, MD, PhD, with mentor Margaret A. Shipp, MD, at Dana-Farber Cancer Institute, Boston

Histologic transformation, when a cancer's features shift dramatically and it presents as a new cancer type, can occur at any point in the course of disease or arise due to the selective pressure of cancer therapies. One of the most well-recognized examples of histologic transformation is the transformation of follicular lymphoma, a slow-growing cancer of the lymphocytes, to an aggressive lymphoma, typically a large B-cell lymphoma. Despite this being well-recognized in the clinic, understanding of the molecular changes that trigger this transformation remains limited. Dr. Parry seeks to comprehensively study the genetics underlying follicular lymphoma transformation with a goal of improving future recognition and diagnosis of transformation. She also aims to identify unique potential therapeutic targets associated with follicular lymphoma transformation.

John R. Prensner, MD, PhD [Ben and Catherine Ivy Foundation Clinical Investigator], with mentors Sriram Venneti, MD, PhD, and Carl J. Koschmann, MD, at University of Michigan, Ann Arbor

New therapeutic approaches are urgently needed for children suffering from high-risk medulloblastoma, a form of pediatric brain cancer, where half of children will experience disease relapse leading to death. Dr. Prensner's work is focused on understanding the biological underpinnings of high-risk medulloblastoma and developing new treatment options. His team recently found that high-risk medulloblastoma may exploit an imbalance in the production of proteins from the tumor cell genetic material (RNA, DNA). Dr. Prensner aims to define the cancer biology that causes an imbalance in the protein-RNA ratio in medulloblastoma, and investigate specific therapeutic options that may target this biology. His hope is that this work leads to new options for clinical trials for children with high-risk medulloblastoma.

Lachelle D. Weeks, MD, PhD [Damon Runyon-Timmerman Traverse Clinical Investigator], with mentor Benjamin L. Ebert, MD, PhD, at Dana-Farber Cancer Institute, Boston

Dr. Weeks plans to develop computerized models that can review images of blood cells and predict a patient's risk of developing acute myeloid leukemia. Because computers can capture small changes in images better than humans looking at cells under a microscope, such a model could connect data about the shapes and appearance of blood cells to the presence of pre-leukemia genetic changes known as clonal hematopoiesis. This work will inform the extent to which blood cell appearance is associated with the underlying biology of leukemia and pre-leukemia. Ultimately, Dr. Weeks aims to refine existing models of leukemia risk prediction and pave the way for screening programs that can identify individuals with clonal hematopoiesis who are at the highest risk for progressing to cancer.

2024 Continuation Grantees

Kelly L. Bolton, MD, PhD, with mentors Matthew J. Walter, MD, and Eytan M. Stein, MD, at Washington University School of Medicine, St. Louis

Myeloid neoplasms (MN), including acute myeloid leukemia and myelodysplastic syndrome, are lethal blood cancers. The genetic mutations in the blood that lead to MN can occur years before diagnosis and maintain almost normal function before transformation. Certain mutations, including

those in the gene IDH2, have been identified as high-risk for developing MN. Individuals with a reduction in the number of mature blood cells (cytopenias) who harbor acquired mutations in their blood, yet do not meet criteria for a cancer diagnosis, have a condition called cytopenias of undetermined significance (CCUS). These individuals almost invariably develop MN. Dr. Bolton will conduct a clinical trial to evaluate whether the IDH2 inhibitor enasidenib can be used as a therapy for CCUS. She will assess mechanisms of resistance and determine whether enasidenib can prevent the development of MN. This represents the first use of genetically targeted therapy for cancer prevention.

Alexander C. Huang, MD, with mentors Gerald P. Linette, MD, PhD, and E. John Wherry, PhD, at University of Pennsylvania, Philadelphia

Immune checkpoint inhibitors (ICI), like anti-PD-1 therapy (α PD-1), have transformed clinical oncology by inducing long-term remissions, even in metastatic disease. However, fewer than 40% of cancer patients achieve such long-term remission with α PD-1, and immune-related toxicity limits more aggressive combined approaches, such as anti-PD1 and anti-CTLA-4 therapy. The question remains why a large portion of the immune response generated by combination immunotherapy is directed towards toxicity rather than anti-tumor immunity. A better understanding of the T-cell response to ICI is needed to develop safer and more effective treatment strategies. In humans, CD8+ T-cells are responsible for anti-tumor immunity. Dr. Huang is investigating the immune responses of different types of CD8+ T-cells to α PD-1 and whether they play a role in determining clinical efficacy and immune toxicity.

David Y. Oh, MD, PhD, with mentor Lawrence Fong, MD, at University of California, San Francisco

While immunotherapies such as anti-PD-1 therapy have provided an important treatment option for bladder cancer, the majority of patients do not respond to these regimens. This may reflect the distinct activation requirements of other immune T cells besides CD8+ T-cells. In recent work, Dr. Oh and colleagues have identified cytotoxic (cancer cell-killing) CD4+ T-cells in human bladder cancer that are associated with immunotherapy responses. However, the regulation of cytotoxic CD4+ T-cells and how these mechanisms compare with CD8+ T-cells is not understood. Dr. Oh proposes to identify and validate surface receptors that enhance or inhibit the activity of cytotoxic CD4+ T cells in human bladder cancer, and the tumor antigens that are recognized specifically by these cells. He will also compare which of these regulatory mechanisms are unique to cytotoxic CD4+ T-cells relative to their CD8+ T-cell counterparts from the same patients. This work has the potential to increase both the proportion of bladder cancer patients who respond to immunotherapy as well as the quality of their response.

Phillip L. Palmbo, MD, PhD, with mentor Joshi J. Alumkal, MD, at University of Michigan, Ann Arbor

Although immunotherapy results in improved survival for some patients with advanced bladder cancer, most tumors do not respond, and the molecular drivers of this resistance to immunotherapy are poorly understood. Dr. Palmbo's goal is to use advanced bladder cancer models and patient data to identify the molecular drivers of resistance to bladder cancer therapy and to develop therapeutic strategies to reverse therapy resistance. His group has identified a gene, TRIM29, which is expressed in 70% of bladder cancers and is associated with

immunotherapy resistance. TRIM29 is a protein that promotes degradation of STING and other innate immune proteins that drive anti-tumor immune response. Dr. Palmbos is currently investigating the regulation of the TRIM29-STING pathway and developing strategies to sensitize bladder and other cancer types to immunotherapy.

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