

# Damon Runyon Awards \$3.2M to Innovative Early-Career Scientists

Meet the eight innovative scientists and learn about the research Damon Runyon is funding.

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The Damon Runyon Cancer Research Foundation has announced eight recipients of the 2023 Damon Runyon-Rachleff Innovation Award, established to support “high-risk, high-reward” ideas with the potential to significantly impact the prevention, diagnosis, or treatment of cancer. Five extraordinary early-career researchers will receive initial grants of \$400,000 over two years, and each will have the opportunity to receive two additional years of funding (for a potential total of \$800,000). This year, this “Stage 2” continuation support was granted to three current Innovators who demonstrated significant progress on their proposed research during the first two years of the award.

The Innovation Award is designed to provide funding to exceptionally creative thinkers with a revolutionary idea who lack sufficient preliminary data to obtain traditional funding. The awardees are selected through a highly competitive and rigorous process by a scientific committee comprised of leading cancer researchers with their own history of innovative work.

Previous Damon Runyon-Rachleff Innovators have pioneered the development of CAR T therapies, revolutionized the biomedical sciences with CRISPR gene editing tools and single-cell DNA sequencing methods, and developed computational methods for analyzing large-scale datasets that continue to yield lifesaving discoveries.

This program was established thanks to the generosity of Andy and Debbie Rachleff.

## New 2023 Damon Runyon-Rachleff Innovators

Lucas Farnung, PhD, Harvard Medical School

“Understanding the mechanistic basis of gene expression regulation by MLL complexes in cancers”

About 70% of pediatric leukemias and 10% of adult leukemias are caused by a genetic disruption in which the mixed lineage leukemia (MLL) 1 gene breaks off and attaches to a different chromosome. This event, known as a chromosomal translocation, gives rise to a distinct subset of leukemias called MLL-rearranged acute myeloid and lymphoblastic leukemias (AML or ALL). Novel treatments for these cancers represent a major unmet medical need. However, the development

of therapeutics is hampered by a lack of basic understanding of how the MLL translocations disrupt the function of affected cancer cells. Dr. Farnung will use biophysical and structural biology approaches to visualize how MLL translocations function at the atomic level and influence the important process of gene transcription. His work will elucidate the precise molecular mechanisms that drive acute leukemias and provide a platform for the development of novel therapeutic strategies against these cancers.

Ryan A. Flynn, MD, PhD, Boston Children's Hospital

"Tools to target novel cell surface ligands in cancer"

Many cancer diagnostic and treatment strategies use markers on the cell surface to find and kill cancer cells in a sea of healthy tissue. Dr. Flynn's research aims to expand our knowledge of what molecules are found on the surface of cancer cells. He will focus on acute myeloid leukemia (AML), as there is a major unmet clinical need for new curative treatments. Specifically, he aims to define RNA as a new cell surface molecule that could have unique structures on AML cells. With this knowledge he will develop antibodies to selectively detect cancer cells and enable tumor killing. Because tumors from other parts of the body also express RNA on their surface, this strategy is expected to be broadly applicable to other cancer types.

Max Jan, MD, PhD, Massachusetts General Hospital

"Programming next-generation NK cell therapies using targeted protein degradation"

Genetically engineered immune cell therapies have emerged as breakthroughs in the treatment of certain blood cancers. However, these advances have been limited to the minority of cancers that express a cell surface protein on all tumor cells; this protein is absent from essential normal tissues and can be recognized and targeted by therapeutic immune cells. Dr. Jan seeks to develop synthetic biology tools to engineer immune cells to recognize the heterogeneous tumor proteins present on many advanced cancers and then activate the body's tumor clearance mechanisms. His goal is to develop cell therapy candidates for direct translation to the care of people with advanced prostate cancer.

(Kathy) Fange Liu, PhD, University of Pennsylvania

"Y chromosome proteins in sex bias of cancers in non-reproductive organs"

Sex differences are markedly evident in many types of cancer, and one of the major contributors to sex-biased differences lies in the sex chromosomes. In contrast to the traditional view that Y chromosome-encoded proteins only function in male reproductive organs, recent evidence suggests that select Y chromosome-encoded proteins are also expressed in male non-reproductive tissues. Furthermore, dysregulation of the Y chromosome-encoded proteins has been implicated in cancers in non-reproductive organs. Upon closer examination, this subgroup of Y chromosome proteins each has corresponding proteins on the X chromosome. Dr. Liu will study the function of

the Y chromosome-encoded proteins and whether and how protein sequence differences from their X chromosome-encoded counterparts lead to functional distinctions in cancer development.

Elvin Wagenblast, PhD, Icahn School of Medicine at Mount Sinai

“Untangling the evolutionary dependency of childhood leukemia”

Age is the greatest risk factor for developing cancer due to the continuous and life-long accumulation of DNA mutations. Although we have identified causes of childhood cancer, including the inheritance of cancer-predisposing genes, other major contributing factors have not yet been identified. Blood cancer is the most common cancer in children and sequencing data indicate that the first genetic mutations occur during fetal development. Dr. Wagenblast will use human blood stem cells and CRISPR/Cas9-mediated genome engineering to model leukemia evolution and identify biological processes that specifically contribute towards cancer development in children. The goal is to leverage this understanding to identify novel therapeutic targets against childhood blood cancer.

#### 2023 Stage 2 Damon Runyon-Rachleff Innovators

Luisa F. Escobar-Hoyos, PhD, Yale University School of Medicine

Damon Runyon-William Raveis Charitable Fund Innovator

“Understanding RNA splicing in tumor-cell adaptation and anti-tumor immunity”

Current pancreatic cancer chemotherapies are not effective, and targeted therapies are only applicable in about 5% of cases. Furthermore, pancreatic cancers cause immune cell stress, limiting the success of immunotherapies in this disease. Using animal models and tumor samples from pancreatic cancer patients, Dr. Escobar-Hoyos has discovered that changes in RNA splicing, a process that controls protein diversity in cells, are crucial for pancreatic cancer development, therapy resistance, and disruption of anti-tumor immunity. She plans to dissect the molecular role of RNA splicing in pancreatic cancer, which likely drives the disease’s lethality. She seeks to develop a novel anti-RNA splicing therapy with dual action—a targeted therapy against tumor cells coupled with an immunotherapy to restore immune cell anti-tumor activity—to more effectively treat pancreatic cancer patients.

Danielle Grotjahn, PhD, Scripps Research

Damon Runyon-Nadia’s Gift Foundation Innovator

“Uncovering structural mechanisms of mitochondrial fragmentation in cancer by cellular cryo-electron tomography”

A unifying hallmark of several types of cancer is the uncontrolled fragmentation of mitochondria, the microscopic compartments that generate energy for the cell. Although many key players have

been implicated in this process, the manner by which these factors assemble to modify the mitochondrial architecture and induce the unrestricted fragmentation associated with cancer is unknown. Dr. Grotjahn uses cutting-edge instrumentation, powerful electron microscopes, and pioneering image processing techniques to visualize this process inside cancer cells. Her work has the potential to identify new targets to block mitochondrial fragmentation as a future therapeutic strategy to prevent cancerous cell proliferation and tumor growth.

Mandar D. Muzumdar, MD, Yale University School of Medicine

“Targeting endocrine-exocrine signaling in pancreatic ductal adenocarcinoma progression”

Obesity is a major risk factor for over a dozen cancer types, including pancreatic cancer, the third leading cause of cancer-related death in the United States. Despite the rising prevalence of obesity worldwide, surprisingly little is known about how it promotes cancer development. Using animal models that closely mimic human pancreatic cancer, Dr. Muzumdar showed that obesity could provoke abnormal signals sent by the hormone-producing cells of the pancreas to their neighboring tumor-forming cells. With this project, he aims to understand how these hormones are induced and act to drive cancer formation in obesity. Targeting pancreatic hormone signaling could provide a new approach for the prevention and treatment of pancreatic cancer and other obesity-associated cancers.

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