

Damon Runyon Awards Over \$4.3M to Top Young Cancer Scientists

Meet the newest class of Damon Runyon Fellows, 14 exceptional postdoctoral scientists conducting cancer research.

February 21, 2023 By [Damon Runyon Cancer Research Foundation](#)

The Damon Runyon Cancer Research Foundation has announced its newest class of Damon Runyon Fellows, 14 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 (\$260,000 total) to investigate cancer causes, mechanisms, therapies, and prevention.

The Foundation has also named seven new 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 Dale Frey Award gives me a big head start as I transition to an independent career," said Xin Zhou, PhD, a 2022 Awardee and current Assistant Professor at Harvard Medical School. "The award will help my lab to jump-start some of our most adventurous projects and will surely facilitate the transformation of these ideas into reality."

Learn about the new Breakthrough Scientists and Fellows below.

2023 Recipients of the Damon Runyon-Dale F. Frey Award for Breakthrough Scientists

Liudmila Andreeva, PhD [Damon Runyon-CRIS Cancer Foundation Breakthrough Scientist],
Eberhard Karl University of Tübingen, Tübingen

"Making an inflammasome: Structural and biochemical elucidation of NLRP3 inflammasome activation"

Dr. Andreeva investigates the role of a molecule called NLRP3 in the assembly of inflammasomes, multiprotein complexes that form in response to cellular infection or stress. NLRP3 acts as a sensor inside the cell that detects danger signals and activates the inflammasome complex to trigger inflammation and cell death. Dr. Andreeva aims to uncover the step-by-step mechanism of NLRP3 activation and regulation to understand how to prevent “false alarms” that cause disease. This research has the potential to aid the development of drugs that specifically turn off the NLRP3 inflammasome and treat a variety of inflammatory disorders, from osteoarthritis to Alzheimer’s disease and cancer.

Erin E. Duffy, PhD, Harvard Medical School, Boston

“Activity-dependent changes in RNA stability as a mechanism for synaptic plasticity”

Dr. Duffy is investigating how neuronal activity can regulate gene expression through a potentially novel mechanism in the developing brain, called RNA turnover. This mechanism may enable gene expression to be rapidly and locally controlled at individual connections between neurons based on neuronal activity. There is evidence that neuronal activity may contribute to pediatric malignant glioma brain tumors. Dr. Duffy aims to characterize this process and identify new therapeutic targets for pediatric brain cancer.

Courtney Ellison, PhD, University of Georgia, Athens

“The regulation and function of type IV pili in *Acinetobacter* biofilm formation”

Dr. Ellison is investigating how single bacterial cells join together to form complex, multicellular structures called biofilms. Biofilms protect bacterial cells from antibiotics and antimicrobial agents, making them difficult to eliminate. Some biofilm-forming species may cause certain cancers, and biofilms of infectious bacteria threaten immunocompromised patients such as those undergoing chemotherapy. Dr. Ellison focuses on bacterial appendages called type IV pili that play a crucial role in biofilm formation. Understanding the role of pili and their contribution to biofilm progression may lead to novel therapies to eliminate biofilms.

Esteban Orellana Vinueza, PhD, Boston Children’s Hospital, Boston

“tRNA dysregulation and cancer”

Dr. Orellana Vinueza is investigating whether changes that modify the shape, stability and function of transfer RNAs (tRNAs) play a role in the development of cancer. The tRNA molecules are involved in the process that translates messenger RNA into a protein. Dr. Orellana Vinueza focuses on a tRNA methyltransferase complex that malfunctions in glioblastoma and liposarcoma. He will assess how alterations in the activity of this enzyme affect global patterns of methylation in normal and human cancer cells. Methylation is the process that controls the timing and amount of proteins that are produced in cells. Understanding how this process breaks down may help decipher the mechanisms that drive cancer and guide the development of new treatments.

Abigail E. Overacre-Delgoffe, PhD, UPMC Hillman Cancer Center, Pittsburgh

“Harnessing the immune response to cancer through the microbiota”

Dr. Overacre-Delgoffe is utilizing unique tools to address how the interaction between the host’s immune system and gut microbes affects colon tumor progression and patient responsiveness to current immunotherapies. Currently, colon cancer patients show an extremely limited response to immune-based therapies and have very poor survival rates. The colon is a unique environment that is composed of host cells and numerous bacteria and microbes that have evolved with the host. Dr. Overacre-Delgoffe aims to understand the basic mechanisms of immunotherapy resistance due to microbiome-immune system interactions, which may aid in developing more effective therapeutics for colon cancer.

Tyler Starr, PhD, University of Utah, Salt Lake City

“Protein evolution at the host-virus interface”

Dr. Starr focuses on the process of how the immune system produces antibodies that specifically recognize and bind to antigens on pathogens and cancer cells. He is combining computational analyses of antibody sequences with experimental methods to test the effects of possible mutations on antibody function. This iterative process, similar to what the body does naturally, will help identify the most effective antibodies for an infection. The results of this research may lead to HIV therapeutics that reduce the incidence of AIDS-related cancers and improve the development of antibody-based cancer immunotherapies. Since the onset of the COVID-19 pandemic, Dr. Starr has extended these approaches to understand the natural evolution of SARS-CoV-2 and how it is targeted by antibodies, with implications for therapeutic antibody development, vaccine design, and preparation for future pandemic emergence from within this viral lineage.

Lexy von Diezmann, PhD, University of Utah, Salt Lake City

Assistant Professor (as of 7/23), University of Minnesota, Minneapolis

“Single-molecule dynamics of DNA repair assemblies in live cells”

Dr. von Diezmann studies how cells control the pathways used to repair DNA. Errors in DNA repair contribute to the development of many cancers, such as breast, ovarian, and pancreatic cancers. Dr. von Diezmann will explore the basic principles governing the DNA damage response by examining how a specific subtype of homologous repair enzymes functions in reproductive cells of the model organism *C. elegans*. Her project will provide mechanistic insight into how changes in the liquid-like organization of proteins at sites of DNA damage regulate repair, supporting the development of novel chemotherapies that modulate the DNA damage response.

November 2022 Damon Runyon Fellows

Ben F. Brian, PhD [HHMI Fellow], with his sponsor Gregory M. Barton, PhD, at the University of California, Berkeley

Abnormal interactions between our immune system and our gut microbes can lead to inflammation that drives colon and gastric cancer growth. Dr. Brian is investigating how the immune system recognizes and responds to these microbes, and how these interactions contribute to abnormal inflammation that can fuel cancer growth. Microbiota-immune interactions have been generally studied in the context of “clean” laboratory mice, but these models do not fully capture human immunology and the complex interplay between host cells and foreign microbes. To overcome this, Dr. Brian plans to study these interactions in “dirty” mice, colonized by a diverse community of microbes as well as pathogens. He will then use laboratory mice with more defined microbial communities to test how recognition of specific microbes by the immune system is regulated and how disruptions to this regulation contributes to inflammation. Dr. Brian received his PhD from the University of Minnesota, Twin Cities and his BS from the University of California, Santa Barbara.

Hui (Vivian) Chiu, PhD, with her sponsor Ruslan Medzhitov, PhD, at Yale University, New Haven

Fatigue is the most common symptom experienced by patients with cancer or undergoing cancer treatment. While chronic inflammation and hormonal imbalance have been suggested as possible causes, the roots of cancer-related fatigue remain unclear and thus we lack effective treatments. Dr. Chiu seeks to illuminate the physiological basis of fatigue using interdisciplinary approaches that combine the strengths of neuroscience, immunology, and computational biology. Through the lens of brain-body interactions, Dr. Chiu aims to identify key molecular and cellular components of fatigue with the goal of improving treatments for cancer and other severe diseases, such as long COVID. Dr. Chiu received her PhD from the California Institute of Technology, Pasadena and her MS and BS from the National Taiwan University, Taiwan.

Mingjian Du, PhD, with his sponsor Charles S. Zuker, PhD, at Columbia University, New York

Global increases in metabolic syndrome, obesity, and diabetes are likely related to the overconsumption of hyper-palatable, cheap, ultra-processed food containing high amounts of added sugar and fat. Intriguingly, the vagus nerve has been discovered as the key conduit relaying information about sugar or fat ingestion from the gut to the brain, where a preference for sugar or fat is then developed and reinforced. Dr. Du aims to understand how the neurons are organized in the gut-brain vagal axis to sense sugar and fat, and to identify and characterize the neural circuits downstream of the gut-brain vagal axis that produce an insatiable appetite for sugar and fat. Understanding the basic biology of the gut-brain axis can provide important insights and strategies to help combat overconsumption of highly processed foods rich in sugar and fat, which may contribute to lowering the risk of metabolic diseases and cancer. Dr. Du received his PhD from The University of Texas Southwestern Medical Center, Dallas and his BS from the Tsinghua University,

Beijing.

Archana Krishnamoorthy, PhD, with her sponsor David S. Pellman, MD, at Dana-Farber Cancer Institute, and Johannes Walter, PhD, at Harvard Medical School, Boston

Cancer initiation and progression stems from cell division errors that promote chromosome breakage and accumulation of mutations. Dr. Krishnamoorthy will use cutting-edge, cross-disciplinary approaches to provide insights into the fundamental question of how cell division shapes the cancer genome. Understanding the mechanisms of cancer genome complexity will help identify better diagnostics and treatments for cancers linked with high levels of genome alterations. Dr. Krishnamoorthy received her PhD from Vanderbilt University, Nashville and her MS from Middle Tennessee State University, Murfreesboro and her BS from PES Institute of Technology, Bangalore.

Nicholas P. Lesner, PhD, with his sponsor M. Celeste Simon, PhD, at the University of Pennsylvania, Philadelphia

Ammonia, a waste product of cellular activity, is cleared from the body by the liver and kidneys through a process known as the urea cycle. During the urea cycle, ammonia is converted to urea, and arginine (an amino acid) is generated. When liver cells become cancerous, the urea cycle pathway stops functioning and cancer cells must import arginine from outside the cell. When cancer cells are prevented from importing arginine (via removal of arginine from the diet or genetic removal of the transporter), tumors do not grow, suggesting that arginine is critical for cells. However, the function of arginine in the cell is unclear. Using mass spectrometry and mathematical modeling, Dr. Lesner will identify the fate of arginine as it is metabolized by liver cancer cells in mouse models, and investigate how this is altered by various genetic mutations. Additionally, he will examine how restricting arginine from the diet genetically alters the liver and tumor cells. By understanding how disruption of this metabolic pathway influences liver cancer growth in the context of specific cancer drivers, Dr. Lesner aims to inform new therapeutic strategies. Dr. Lesner received his PhD from The University of Texas Southwestern Medical Center, Dallas and his BA from the University of Wooster, Wooster, Ohio.

Ryan Y. Muller, PhD [HHMI Fellow], with his sponsor David P. Bartel, PhD, at Whitehead Institute for Biomedical Research, Cambridge

Epstein-Barr Virus (EBV) is known to cause several human cancers, including nasopharyngeal cancer, gastric cancer, and B-cell lymphomas. During the early stages of viral infection, EBV induces a state of rapid cell division in host cells that promotes oncogenesis. Dr. Muller studies specific regions of RNA, known as stable introns, which are expressed at abundant levels during early infection but whose role in the viral lifecycle and during oncogenesis is unknown. Investigating how stable introns influence the host cell's biology may reveal insights into EBV-driven oncogenesis and provide a general understanding of the mechanisms that drive cancer progression. Dr. Muller received his PhD from the University of California, Berkeley and his BS from Arizona State University, Tempe.

Christopher Noetzel, PhD, with his sponsor Boris Striepen, PhD, at the University of Pennsylvania, Philadelphia

As different tissues in the body form, cells need to undergo a complex, precisely timed series of differentiation programs to form specialized cell types. Importantly, premature or delayed initiation of these programs can contribute to cancer formation. However, how timing of cellular differentiation is encoded on a molecular level is poorly understood. Dr. Noetzel is using the protozoan parasite *Cryptosporidium parvum* as a simplified model of eukaryotic differentiation. After infecting the intestinal lining of a mammalian host, these single-celled parasites undergo exactly three rounds of asexual replication before collectively differentiating into gametes. These studies will investigate how this hard-wired, intrinsic developmental timer is encoded. In his project, Dr. Noetzel aims to understand how these parasites “count to three,” which will inform our basic understanding of how eukaryotic cells keep track of time during development. Dr. Noetzel received his PhD from the Weill Cornell Medical College, Cornell University, New York and his MSc and BSc from Georg-August-University, Göttingen.

James Osei-Owusu, PhD, with his sponsor Andrew C. Kruse, PhD, at Harvard Medical School, Boston

The relaxin-2 receptor (RXFP1), which binds a hormone aptly known as relaxin, plays a key role in reproductive and cardiovascular physiology by increasing blood flow and facilitating childbirth. Abnormal signaling of the receptor leads to a variety of cancers, particularly tumors of reproductive tissues. James aims to establish a comprehensive understanding of RXFP1 molecular signaling and how it can be inhibited for cancer treatment. This study will facilitate the discovery of candidate therapeutic agents as well as aid the rational design of drugs for reproductive cancer treatment. Dr. Osei-Owusu received his PhD from Johns Hopkins University, Baltimore and his MSc and BSc from the University of Ghana, Legon.

Claudia A. Rivera Cifuentes, PhD [Lorraine W. Egan Fellow], with her sponsor Yasmine Belkaid, PhD, at the National Institute of Allergy and Infectious Diseases, Bethesda

Endogenous retroviruses are viral elements of the human genome derived from retroviral infections of distant ancestors. Recent findings support the idea that these elements can cause immune system activation and inflammation. However, the crosstalk between endogenous retroviruses and the gut microbes that control immunity within the gut—and how abnormalities in this dialogue lead to inflammatory disorders—is not well understood. Further, although endogenous retroviruses have been proposed as potential targets for immunotherapy, we lack a mechanistic understanding of their interactions with the gut microbiota and how these interactions influence cancer development. Dr. Rivera Cifuentes aims to uncover how multi-kingdom interactions in the gut control intestinal health and colorectal cancer development. This work may have important clinical implications for the treatment of gut inflammatory disorders and gastrointestinal cancers. Dr. Rivera Cifuentes received her PhD from the University of Paris, Paris and her BSc from The Pontifical Catholic University of Chile, Santiago.

Joshua Sheetz, PhD [HHMI Fellow], with his sponsor Michael Rape, PhD, at the University of

Cancer cells adapt their metabolism to achieve rapid growth and proliferation. Much of their metabolic malleability hinges on mitochondria, subcellular hubs for energy transformation and biosynthesis. As a key means to control mitochondrial composition and meet metabolic demands, cells mark mitochondrial proteins for degradation by a process called ubiquitylation. How both cancerous and healthy cells direct and monitor mitochondrial ubiquitylation remains poorly understood. Dr. Sheetz aims to dissect the cellular machinery that performs mitochondrial ubiquitylation and determine how this process promotes metabolic adaptability in cancer cells. A major translational goal is to identify approaches for tuning the levels of mitochondrial ubiquitylation in tumors and in metabolic disorders that put patients at risk for cancer. Dr. Sheetz received his PhD from Yale University, New Haven and his BS from the University of North Carolina, Chapel Hill.

Marie R. Siwicki, PhD [Dale F. and Betty Ann Frey Fellow], with her sponsor Paul Kubes, PhD, at the University of Calgary, Alberta

Neutrophils are important anti-microbial cells within the innate immune system. Recently, it has been shown that neutrophils can perform diverse functions, taking on both pro-inflammatory and pro-healing roles in response to tissue injury or insult. Dr. Siwicki's goal is to understand how different neutrophil subtypes or states function to balance inflammatory versus regenerative processes, ultimately influencing tissue health and cancer. This work has the potential to uncover the basis of neutrophils' pro-tumor versus anti-tumor functions and could open the door to therapeutic targeting of specific neutrophil behaviors in order to improve clinical outcomes in cancer. Dr. Siwicki received her PhD from Harvard Medical School, Boston and ScB from Brown University, Providence.

James W. Swann, VetMB, DPhil [William Raveis Charitable Fund Fellow], with his sponsor Emmanuelle Passegué, PhD, at Columbia University, New York

A key question in cancer biology is how genetic mutations, acquired over time, interact with environmental factors to affect emergence and progression of disease. This is particularly relevant in blood cancers because many people acquire genetic mutations in blood-forming stem cells in the bone marrow but only a small proportion go on to develop acute myeloid leukemia (AML). Dr. Swann is investigating whether inflammatory signals alter the behavior of stem cells that have already acquired an initial mutation, causing them to acquire features of cancer that will hasten the onset of AML. Specifically, Dr. Swann is interested in whether pre-cancerous stem cells change their gene expression in response to inflammation, which might allow them to outcompete normal cells in the bone marrow. He is utilizing cutting-edge techniques such as CRISPR editing of blood stem cells to investigate the molecular pathways responsible for these biological changes. This project has the potential to identify molecular pathways activated by inflammation that might promote AML development, offering new targets for therapeutic interventions. Dr. Swann received his VetMD (DVM) from the University of Cambridge and his DPhil (PhD) from the University of Oxford.

Erik Van Dis, PhD [Robert Black Fellow], with his sponsor Daniel B. Stetson, PhD, at the University of Washington, Seattle

The innate immune system is the body's first line of defense against pathogens. The innate immune sensor MDA5 detects nucleic acids derived from pathogenic genomes or damaged cells and drives the production of cytokines, an important signaling molecule in the immune inflammatory response. MDA5 can be aberrantly activated by host nucleic acids, however, leading to autoimmune activation. Hyperactive MDA5 alleles are associated with the development of autoimmune diabetes. Dr. Van Dis aims to define the innate immune signaling pathways that initiate autoimmune diabetes to better understand immune activation pathways in the pancreas and guide the development of novel immunotherapies for pancreatic cancer. Dr. Van Dis received his PhD from the University of California, Berkeley and his BA from Carleton College, Northfield.

McLane Watson, PhD, with his sponsor Russell G. Jones, PhD, at Van Andel Research Institute, Grand Rapids

Cancer immunotherapy has revolutionized the way we treat cancer; however, it is only successful in a small subset of patients. Optimally functioning CD8 T cells, the specialized killers of the immune system, are key to the success of cancer immunotherapies. While CD8 T cell function is highly influenced by their metabolism, little is understood about how metabolism changes the function of these cells. Dr. Watson hypothesizes that metabolism affects CD8 T cell function by altering how tightly its DNA is packaged (its epigenetics), leading to altered gene expression. Using a mouse model of adoptive T cell therapy, a widely used immunotherapy in humans, and epigenetic techniques, Dr. Watson proposes to uncover how metabolism influences CD8 T cell epigenetic landscapes to control their function. He plans to apply these findings to improve T cell function and enhance tumor clearance. Dr. Watson received his PhD from the University of Pittsburgh, Pittsburgh and his BS from Hope College, Holland, Michigan.

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