

Damon Runyon Cancer Research Foundation Awards Quantitative Biology Fellowships to Five Cutting-Edge Scientists

The award program was created to encourage quantitative scientists to pursue careers in cancer research.

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Damon Runyon has announced a new cohort of Quantitative Biology Fellows, five exceptional early-career scientists who are bringing cutting-edge computational tools to bear on some of the most important questions in cancer biology. Whether designing new proteins or mapping DNA structure, their projects aim to shed light on these fundamental questions through large-scale data collection, mathematical modeling, and quantitative analysis.

“In the five years since we named the first class of Quantitative Biology Fellows, it has only become more evident that these scientists bring fresh perspectives and creative approaches to cancer research in addition to their robust computational skills,” said Yung S. Lie, PhD, President and CEO of the Damon Runyon Cancer Research Foundation.

Each postdoctoral scientist selected for this unique three-year award will receive independent funding (\$240,000 total) to train under the joint mentorship of an established computational scientist and a cancer biologist. The award program was created to encourage quantitative scientists (from fields such as mathematics, physics, computer science, and engineering) to pursue careers in cancer research. By investing in research that combines techniques from “wet” and “dry” labs, Damon Runyon aims to highlight the importance of these specially trained scientists in the era of precision medicine.

2025 Quantitative Biology Fellows

Simone Bruno, PhD, with mentors Franziska Michor, PhD, and Karen Cichowski, PhD, at Dana-Farber Cancer Institute, Boston

Triple-negative breast cancer (TNBC) is one of the most aggressive and difficult-to-treat forms of breast cancer. Dr. Bruno’s research focuses on how changes in DNA packaging, known as

chromatin, affect cancer progression and treatment response. Using advanced computational models, Dr. Bruno will investigate the interplay between chromatin modifications and resistance mechanisms, with the goal of identifying strategies to mitigate therapy resistance and reduce cancer recurrence. While Dr. Bruno's primary focus is TNBC, this research provides a generalizable framework that could be applied to other cancers where chromatin modifications play a key role, leading to better, more durable treatments for a broader range of patients.

Computational Methodology:

In this project, Dr. Bruno will develop a computational framework that integrates mathematical models of chromatin modification dynamics with pharmacokinetic and pharmacodynamic models to study the role of these modifications in triple-negative breast cancer (TNBC) progression. Dr. Bruno will adapt existing chromatin modification circuit models to TNBC dynamics and, using Bayesian inference methods, will parameterize them with experimental data. By integrating pharmacokinetic models for specific drugs, this framework will enable the evaluation of diverse treatment strategies.

Paul C. Klauser, PhD, with mentors Jason S. Lewis, PhD, and Caleb A. Lareau, PhD, at Memorial Sloan Kettering Cancer Center, New York

Radiopharmaceuticals, or drugs that contain radioactive forms of chemical elements, have transformed cancer diagnosis and treatment. Radioactive copper and manganese, for example, play a crucial role in PET imaging, while radioactive lutetium is used to deliver targeted radiation to cancer cells. While these radiometals have tremendous potential, however, their application is hindered by a lack of efficient "chelators," or molecules that can securely bind radiometals in the human body. Computational protein design offers a solution by engineering protein-based chelators optimized for radiometal coordination, stability, and biocompatibility. Using advanced protein modeling, Dr. Klauser will develop chelators for radiometals, improving diagnostic imaging and advancing lutetium-based radiotherapies. While this work is applied to HER2-positive gastric cancer, these strategies have broad applications across various cancer types, ultimately enhancing precision oncology and expanding radiopharmaceutical utility.

Computational Methodology:

This research develops a computational strategy to design stable, compact metal-binding proteins for radiopharmaceuticals, enabling fusion with therapeutic antibodies. Using diffusion models, such as RFDiffusion, thousands of protein backbones are generated for metals like copper and manganese. Sequences are assigned via ProteinMPNN, filtered for stability and binding with AlphaFold 3. For rare lanthanides, symmetric duplication of known binding motifs is used. This approach streamlines the discovery of stable scaffolds for radiopharmaceutical applications.

Sohyeon Park, PhD, with mentors Alexandar Hoffmann, PhD, and Frank Alber, PhD, at the University of California, Los Angeles

Macrophages are a major component of the body's first line of defense, acting as sentinel cells that detect and respond to threats. One powerful trait of macrophages is their ability to modulate their response based on previous exposure to stimuli. In cancer, this adaptability can steer macrophages either to fight tumors or to protect them, depending on prior experiences. This phenomenon is often referred to as "macrophage memory." Though macrophage memory is increasingly recognized as a factor influencing cancer progression and treatment outcomes, the mechanisms that allow macrophages to retain this memory remain unclear. Dr. Park hypothesizes that exposure of macrophages to certain stimuli leads to lasting changes in the structure of their DNA. She will combine both experimental and computational approaches to elucidate how this memory forms and how it affects the expression of immune-related genes. By uncovering the principles of macrophage memory formation, she will lay the groundwork for strategies to reprogram macrophages, potentially enhancing anti-tumor immunity and improving cancer therapy.

Computational Methodology:

Dr. Park will use machine learning-aided genome modeling, leveraging bulk Hi-C data, to reconstruct 3D chromosome structures and validate them with deep learning-based image analysis. This framework will infer single-cell 3D chromosome structure in macrophages. Dr. Park will also quantify nuclear speckle-mRNA spatial relationships using microscopy and develop mathematical models of gene regulation that incorporate transcription factor activity. This integrative approach will reveal how chromosome structure shapes macrophage immune memory and functional response in cancer.

Ruoyu Wang, PhD, with mentors Jian Zhou, PhD, and W. Lee Kraus, PhD, at the University of Texas Southwestern Medical Center, Dallas

Many cancer mutations occur in regions of the human genome that do not code for proteins. These non-coding regions serve as vital regulators of gene expression; mutations in these regions contribute to various hallmarks of cancer. Elucidating these regulatory elements and their malignant variants is critical for advancing our understanding of cancer biology and fostering precision medicine. Deep learning sequence models can substantially enhance our grasp of the regulatory genome in both health and disease. To this end, Dr. Wang aims to combine generative AI models with single-molecule regulatory genomics to uncover the principles that underlie the cancer regulatory genome at unprecedented resolution and precision.

Computational Methodology:

With single-molecule regulatory genomics, Dr. Wang will develop a deep generative AI model to learn the probability landscape of the single-molecule regulatory genome. By taking any DNA sequence as input, the deep generative AI model can generate diverse configurations of single-molecule chromatin states.

Aaron Zweig, PhD, with mentors David A. Knowles, PhD, and Elham Azizi, PhD, at the New York Genome Center, New York

Dr. Zweig is modeling how gene regulation changes across time and space using advanced geometry, deep learning models, and CRISPR gene-editing technology. With these tools, it is possible to learn how RNA expression evolves in a cell over time and, ideally, predict evolution in regions susceptible to cancerous mutation(s). This work has the potential to apply to many types of cancer but is especially applicable for acute myeloid leukemia (AML). This is in part because there are known “precursor” states—when cells are at higher risk of becoming cancerous—where understanding gene regulation is most impactful, and in part because a common element of AML treatment is a stem cell transplant, which may attack host cells inconsistently in different regions of the same organ.

Computational Methodology:

The project models temporal gene dynamics via stochastic differential equations, parameterizing the drift vector field with linear networks or shallow neural networks to guarantee provable identifiability, trained via differentiation through the adjoint method. Spatial interactions between spot clusters are characterized with graph neural networks, symmetric functions parameterizing summary statistics, and self-attention modules on latent gene embeddings. The latent embeddings are also defined through a variational autoencoder integrating RNA with other modalities.

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