

# Damon Runyon Cancer Research Foundation Names Three New Quantitative Biology Fellows

Their projects use computational approaches to understand cancer risk, improve immunotherapy and identify chromosomal drivers of cancer.

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Damon Runyon has awarded Quantitative Biology Fellowships to three outstanding young scientists applying computational approaches to persistent biological questions. Their projects will use machine learning, spatialomics, advanced network modeling, and other technologies to better understand breast cancer risk, improve response to immunotherapies, and identify new chromosomal drivers of cancer. Each postdoctoral scientist will receive independent funding (\$240,000 total) to train under the joint mentorship of an established computational scientist and a cancer biologist.

Now in its seventh year, this unique award program was created to encourage scientists with backgrounds in 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.

“In an era where technological capacity and access to biological data sets are increasing at a rapid pace, computational expertise is becoming even more important in cancer research,” says Yung S. Lie, PhD, President and CEO of the Damon Runyon Cancer Research Foundation. “Our Quantitative Biology Fellows are already establishing themselves as interdisciplinary pioneers in the field.”

## 2026 Quantitative Biology Fellows

Minsoo Kim, PhD [Breast Cancer Research Foundation Quantitative Biology Fellow], with mentors Nicholas E. Navin, PhD, and Ken Chen, PhD, at the University of Texas MD Anderson Cancer Center, Houston

Cells in healthy individuals were thought to carry the same number of chromosomes, but it was recently discovered that in the breast tissue of healthy women there are rare cell populations that acquire extra or lose copies, a condition called aneuploidy. These abnormal cells mirror the characteristics of breast cancers and may represent early precursors of the disease years before clinical diagnosis. Dr. Kim focuses on finding and characterizing these rare abnormal cells in healthy breast tissues. He hopes to build computational tools to understand what biomarkers set these cells apart from their healthy neighbors and how their surrounding microenvironment may influence their behavior, opening new avenues for early cancer detection and risk stratification. To test whether these signs truly predict cancer development, he plans to apply this approach to breast tissue samples from patients who were monitored over many years, some of whom later developed cancer, with the goal of giving clinicians better ways to detect breast cancer earlier and to identify at-risk patients. He received his BA from Johns Hopkins University in Baltimore, MS from the University of Minnesota in Minneapolis, and PhD from Cornell University in New York.

#### Computational Methodology:

Dr. Kim will develop a heterogeneous graph neural network (GNN) that jointly models single cell copy number alterations and gene expression with genes, cells, and chromosome segments as nodes. This will separate transcriptional changes driven by chromosomal gains and losses from other sources of variation. He will extend the model to spatial transcriptomic data to further isolate microenvironmental influences on gene expression and understand how these rare aneuploid cells interact with their local environment.

Sahana Kuthyar, PhD, with mentors Jared Mayers, MD, PhD, and Michael Wu, PhD, at Fred Hutchinson Cancer Center, Seattle

Dr. Kuthyar studies why cancer patients, especially those receiving treatments like chemotherapy or radiation, are at high risk of developing serious lung infections such as pneumonia. While these treatments are essential for killing cancer cells, they also weaken a key part of the immune system that normally helps the body detect and eliminate bacteria. This weakened defense makes patients more vulnerable to infection. At the same time, many hospitalized patients receive supplemental oxygen, which can change the lung environment in ways that help certain bacteria grow stronger and become more aggressive. In cancer patients, these two factors are closely connected: the weakened immune system cannot effectively control bacteria, while the high-oxygen environment actively promotes bacterial survival and virulence. Together, this creates a perfect storm that increases both the risk of contracting pneumonia and severity of disease. This work is relevant to cancers commonly treated with immune-suppressing therapies, including leukemia, lymphoma, and solid tumors such as lung, breast, and colorectal cancer, and aims to identify better ways to predict, prevent, and treat these life-threatening infections. She received her BS and MS from Emory University in Atlanta and her PhD from the University of California, San Diego.

### Computational Methodology:

This project proposes a framework to dissect pneumonia risk in immunocompromised patients using human and mouse models. Dr. Kuthyar will use hierarchical networks to link gene expression and metabolites. Multi-omics factor analysis will capture microbial and immune variation and models trained on human data will be tested in mice, enabling iterative prediction and validation. This approach integrates species harmonization, metabolite prioritization, and network mapping to reveal hyperoxia-driven microbial adaptation and myeloid immune deficits driving pneumonia risk.

Matthew Leventhal, PhD, with mentors Cheng-Zhong Zhang, PhD, and David S. Pellman, MD, at Dana-Farber Cancer Institute, Boston

The X and Y chromosomes play a crucial role in human sex determination. Females have two copies of the X chromosome, while males have one X chromosome and one Y chromosome. In females, the second copy of the X chromosome is silenced early in development, meaning that only one of the X chromosomes is expressed. As a result, mutations on the activated X chromosome are more likely to change cellular functions and in context of cancer, could lead to more rapid disease progression. Dr. Leventhal proposes a novel computational approach to distinguish between the actively expressed and silenced X chromosomes in females. He hopes to use this method to analyze a dataset of over 8,000 tumors to identify potential new drivers and molecular vulnerabilities within 31 types of cancer. He will model whether these alterations can occur in pre-cancerous cells, indicating that they could be targets for early therapeutic intervention. He received his BA from Bowdoin College in Brunswick and his PhD from Massachusetts Institute of Technology, Cambridge.

### Computational Methodology:

Dr. Leventhal will develop a computational tool that models the error rate of statistical phasing in bulk whole-genome sequencing and corrects these errors to determine accurate haplotype-specific copy number of all chromosomes. This model integrates genomics with RNA-seq data to determine the active and inactive X chromosome. The subsequent error correction will allow him to perform the first pan-cancer analysis in over 8546 tumors to identify recurrent copy number alterations affecting the active or inactive X chromosome.

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