Damon Runyon Awards Fellowships to 15 Brilliant Young Scientists

Grants totaling nearly $3.5 million give early career investigators independence to pursue brave and bold cancer research.

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Fifteen scientists from across the country were named Damon Runyon Fellows. The recipients of this prestigious, four-year award are outstanding postdoctoral scientists conducting basic and translational cancer research in the laboratories of leading senior investigators. The Fellowship encourages the nation's most promising young scientists to pursue careers in cancer research by providing them with independent funding ($231,000 total) to work on creative, high-risk projects.

"We are thrilled to be funding these innovative, young scientists with the brilliance and passion to push boundaries and make breakthroughs. They are committed to understanding the fundamental processes driving cancer, which may ultimately lead to new therapeutic approaches for patients," said Yung S. Lie, PhD, President and CEO of the Damon Runyon Cancer Research Foundation.

Spring 2020 Damon Runyon Fellows:

Nicholas M. Adams, PhD [Marion Abbe Fellow], with his sponsor Boris Reizis, PhD, at the New York University School of Medicine, New York, studies a specialized subset of immune cells that secrete potent antitumor cytokines called type I interferons (IFN-I). Within a tumor, these cells, called plasmacytoid dendritic cells (pDCs), are impaired, which contributes to immune suppression and cancer progression. Dr. Adams aims to uncover the molecular mechanisms that govern IFN-I production and pDC dysfunction in cancer. As dendritic cells are a promising cell therapy for cancer, understanding the regulation of pDC-IFN-I production can guide strategies to harness and integrate their anti-tumor function in new immunotherapies.

Yiming Chen, PhD, with his sponsor Karl Deisseroth, MD, PhD, at Stanford University School of Medicine, Stanford, is developing platforms to conduct high-throughput screens of protein-based fluorescent biosensors. Biosensors allow visualization of otherwise invisible biological processes such as communication between cells. Cells use diverse peptides to send messages to each other, and this has important implications for tumor growth and side effects of cancer treatments. This project will increase understanding of the complex signaling networks among cells and may lead to rational design of new cancer therapies targeting faulty cellular communication.

Junhong Choi, PhD [HHMI Fellow], with his sponsor Jay A. Shendure, MD, PhD, at the University of

Washington, Seattle, is examining how the control of DNA replication plays a role in cell growth and proliferation. Cellular growth is tightly orchestrated around DNA replication, where the genome must be faithfully copied before the cell divides. Different cell types each have a distinct DNA replication program, dictating which parts of the genome are copied before others. Dr. Choi aims to develop a high-throughput method to profile DNA replication in many cells at once to gain a better understanding of the relationship between DNA replication and cell growth. This research has the potential to uncover critical insights into cancer development.

Pragya Goel, PhD [Dale F. and Betty Ann Frey Fellow], with her sponsor Pascal Kaeser, MD, at Harvard Medical School, Boston, is investigating structural and functional aspects of dopamine transmission in the brain, a key neuromodulator for motor and cognitive processes. Dopamine receptors have also been implicated in a variety of cancers, and recent evidence suggests that brain cancer (glioma) cells can form synaptic connections with neurons that drive tumor progression. To better understand the molecular organization that supports dopamine signaling, Dr. Goel will use super-resolution microscopy, modern genetic approaches, and functional measurements to assess how major dopamine receptors are organized in the brain and determine the interplay between dopamine release and reception. This research aims to better understand the basic mechanisms of dopamine signaling, which may ultimately enable the design of novel therapies.

Anita Gola, PhD, with her sponsor Elaine V. Fuchs, PhD, at The Rockefeller University, New York, is investigating how tissue regenerates the right cell type, at the right place. Effective cell-cell communication and cell-spatial organization are critical to maintaining organ function and homeostasis. Dr. Gola will use skin as a model tissue to understand how immune cells are organized and how they communicate with resident stem cells while maintaining tolerance and providing protection. When these interactions are disrupted, they can lead to cancers and other hyper-proliferative disorders. Unraveling the mechanisms that govern healthy immune-stem cell crosstalk and what goes wrong in disease may lead to new therapeutics for skin cancers.

Rachel Segal Greenberg, PhD [HHMI Fellow], with her sponsor Stephen Liberles, PhD, at Harvard Medical School, Boston, is focusing on how sensory neurons that innervate internal organs develop and function under changing environmental conditions. Our ability to sense and respond to fluctuations in blood-oxygen levels or exposure to airway irritants is controlled by the sensory neurons that comprise the vagus nerve. These neurons detect changes in numerous organs including the heart and lungs, and mediate responses. Understanding how vagal neurons respond to these microenvironments may provide new insights into how certain conditions contribute to tumor growth and identify targets for the development of cancer therapies.

Shuo Han, PhD [Fayez Sarofim Fellow], with his sponsor Philip A. Beachy, PhD, at Stanford University School of Medicine, Stanford, is developing novel methodologies to fine-tune cellular signaling pathways that may prevent tumor formation and promote regeneration through the Hedgehog pathway. This pathway plays a central role in regulating embryonic tissue patterning and tissue renewal after birth. Dr. Han will take an interdisciplinary approach combining chemical biology, protein engineering, and computational modeling to examine the Hedgehog pathway in a

cell-type specific and spatiotemporally resolved manner. This work aims to provide mechanistic insights into the function of Hedgehog signaling in tissue repair and establish new therapeutic approaches for regenerative medicine.

Balint Z. Kacsoh, PhD [Rebecca Ridley Kry Fellow], with his sponsors Shelley L. Berger, PhD, and Christopher J. Lengner, PhD, at the University of Pennsylvania, Philadelphia, is studying how social environment can affect disease initiation or progression. Empirical evidence suggests that extreme social environments—such as overcrowding or isolation—can induce or accelerate disease states such as cancer, but little is known about the underlying biology. Dr. Kacsoh proposes to dissect the molecular processes underlying disease progression as a function of social structure by using the very social model organism, the ant species Camponotus floridanus, and by generating a colorectal-like tumor model in these ants. Studies of model organisms with conserved cancer pathways may provide clues to the links between social environment and disease.

Haoxin Li, PhD [The Mark Foundation for Cancer Research Fellow], with his sponsor Benjamin F. Cravatt, PhD, at The Scripps Research Institute, La Jolla, is mapping the positions of the amino acid cysteine in cancer-relevant proteins. He will perform functional screens that reveal the cysteine residues that are essential to the progression of cancer. Since the unique chemistry of cysteine makes it an attractive target for therapeutic development, this map can guide the discovery and optimization of drugs that can bind to and inhibit cancer-promoting proteins. His research has the potential to greatly accelerate the discovery of new cancer targets and their corresponding therapeutics.

Jingchuan Luo, PhD [HHMI Fellow], with her sponsor Jonathan S. Weissman, PhD, at Whitehead Institute for Biomedical Research, Cambridge, is focusing on the interplay between energy-producing mitochondria and the nucleus inside mammalian cells. Mitochondria contain their own small genome that encodes some proteins, but the vast majority are encoded in the cell's nucleus. The communication between mitochondria and the nucleus to produce the proteins necessary to properly function is tightly controlled, and its dysregulation has been implicated in human diseases including cancer. Dr. Luo is using ribosome profiling in parallel with CRISPR to quantitatively monitor translation (the process of protein production from RNA) on the mitochondrial surface and identify key regulators of this process. She hopes gaining an understanding of the underlying mechanism will yield fundamental insights into mitochondrial biology and its role in disease.

Tristan Wold Owens, PhD [Suzanne and Bob Wright Fellow], with his sponsor David A. Agard, PhD, at the University of California, San Francisco, focuses on heat shock proteins (HSPs) and their "master regulator" called heat shock transcription factor 1 (HSF1). The transformation and growth of cancers causes a wide array of cellular stresses including metabolic changes, genomic instability, and protein misfolding that would halt the growth of a normal cell. Tumor cells, however, depend on cellular stress response machinery, like HSPs, for their survival. HSF1 is critical to tumor development and progression, and HSF1 activity is strongly correlated with poor prognosis in many common cancers. For decades, efforts to develop cancer therapies targeting HSPs have failed. Dr. Owens aims to understand how HSPs and HSF1 interact to regulate activity,

and how this regulation is co-opted to promote tumor growth and progression.

Cristina Puchades, PhD, with her sponsors Yifan Cheng, PhD, and Lily Y. Jan, PhD, at the University of California, San Francisco, studies ion channels – proteins embedded in the membrane surrounding a cell. They act as molecular gates, opening in response to diverse stimuli to allow ions to flow into cells. The essential ion channel TMEM16A is required for many fundamental physiological processes, including neuronal signaling, muscle contraction, and salivary gland secretion. In cancer cells, increased activity of TMEM16A is closely linked to metastatic progression in esophageal, gastric, and pancreatic cancers. Dr. Puchades aims to understand how TMEM16A functions and how drug molecules hinder its activity. This research has the potential to guide the pharmacological targeting of TMEM16A as a novel approach for the development of anti-cancer therapeutics.

Jiao Sima, PhD [HHMI Fellow], with her sponsor Yang Dan, PhD, at the University of California, Berkeley, is investigating the relationship between sleep disturbances and cancer development. She is dissecting how neurons controlling the sleep-wake cycle affect immune functions that impact cancer. Dr. Sima will also examine the complementary problem of how tumor growth and chemotherapy contribute to sleep issues by analyzing gene expression patterns in neurons that regulate the sleep-wake cycle. Understanding the cellular mechanisms linking sleep and cancer could pave the way for drugs that help prevent cancer-induced sleep problems and therapeutic approaches that boost immune function to fight cancer.

Mark R. Sullivan, PhD [Merck Fellow], with his sponsor Eric J. Rubin, MD, PhD, at the Harvard T. H. Chan School of Public Health, Boston, studies the processes that lead to opportunistic infections affecting cancer patients. The human body, which is a hostile environment for pathogens, is well-equipped to fend off infections from most bacteria. However, cancer and chemotherapy can cause inflammation, tissue damage, and impairment of the immune system in ways that leave patients vulnerable to bacterial infection. These opportunistic infections are challenging to treat, as antibiotics often have little effect on these bacteria. Dr. Sullivan aims to identify the bacterial components that allow opportunistic pathogens to live within the lung and survive antibiotic treatment. This research will be critical to discovering more effective therapies to eradicate these infections.

Yunxiao Zhang, PhD [Merck Fellow], with his sponsor Ardem Patapoutian, PhD, at The Scripps Research Institute, La Jolla, is investigating how abnormal “mechanical loading” resulting from obesity, repeated joint use, and structural deformity affects the development of osteoarthritis, most common form of arthritis. Over time, the physical force on joints is converted to chemical signals that lead to wearing down of the protective cartilage in joints that cushions the ends of a person’s bones. Understanding the underlying signaling pathway will facilitate the development of therapies for osteoarthritis, and may also shed light on cancer, which involves similar pathways. The importance of mechanical stress on disease progression is now being revealed.

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