Gerald Shadel

In humans, as in most animal cells, genetic information is housed not only in the nucleus, but also in mitochondria. Mitochondrial DNA (mtDNA) encodes thirteen essential proteins of the ATP-producing oxidative phosphorylation complexes as well as 22 tRNAs and 2 rRNAs required to translate these thirteen mRNAs in the mitochondrial matrix. Mutations in mtDNA cause maternally inherited disease syndromes that involve complex pathology, including neuromuscular and cardiac pathology, deafness, and blindness. In addition, mtDNA mutations accumulate in normal aging tissues, certain tumors, and have been implicated in late-onset diseases such as Alzheimer's, Parkinson's, diabetes, and cancer, indicating that the pathology of dysfunctional mitochondria is only beginning to be unraveled. The research in the Shadel laboratory is directed toward understanding the mechanism of gene expression in human mitochondria and its impact on human disease and aging. The ultimate goal is to understand the full impact of dysfunctional mitochondrial gene expression on human health and use this information to design specific interventions to treat mitochondriabased disease and age-related pathology.

Katerina Politi

The research in the Politi laboratory focuses on lung cancer, the most common cancer worldwide. In particular, we study a subset of lung tumors that have mutations in the Epidermal Growth Factor Receptor (EGFR). EGFR mutations are found in approximately 10% of non-small cell lung cancer cases in the U.S.—most commonly in never-smokers—and are associated with sensitivity to a class of drugs called EGFR tyrosine kinase inhibitors. Our research aims to understand which signaling pathways underlie EGFR mutant lung tumor development, identify novel therapeutic strategies to treat the disease, and uncover mechanisms of drug resistance.

John Sinard

John Sinard's research focuses on designing and developing informatics solutions which can be deployed into the dayto-day practice of pathology and medicine. Dr. Sinard has led the development of several custom software solutions addressing workflow issues in the Department. At the institutional level, he is interested in the design and building of data repositories that can deliver clinical data directly to faculty investigators, enabling clinical research. He also has an interest in the use of computers in medical education.

Yuval Kluger

The research in the Kluger computational biology and bioinformatics laboratory involves analysis of genomics and proteomics experiments. This includes computational analysis of output from high-throughput datasets generated from experiments involving melanoma, breast cancer, hematopoeisis, cell cycle genomics, and protein-protein interactions. The computational activities in our laboratory currently include the following areas: 1) application of signal processing approaches for identification of relevant biological signals in high-throughput experiments, such as identification of aberrations in multi-subclonal cancer samples, signal denoising in next generation platforms, and de-mixing of cell types in heterogeneous samples; 2) developing approaches to analyze high dimensional data from genomics platforms for biomarker discovery and personalized medicine. In particular, we use advanced applied mathematical methods to search complex local and non-local genomic patterns across the genome that may discriminate cancer patients with good vs. poor outcomes in CNA studies employing next generation sequencing or SNP platforms; and 3) uncovering direct and collective regulatory relationships between regulators (TFs, epigenomic factors, and miRNAs) and their target genes by integration of heterogeneous Omics datasets and DNA sequences.

Michael Robeck

Some viruses, such as HIV and the hepatitis B (HBV) and C viruses, establish chronic infections that persist for the lifetime of their hosts and are associated with a large degree of worldwide mortality. The Robek laboratory studies the host-pathogen interactions related to HBV infection. Our lab is exploring three unique therapeutic approaches to treat this disease: 1) we are investigating the relationships between HBV and cellular signaling pathways, as these may be exploited pharmacologically to block virus replication; 2) we are characterizing the ability of novel immunoregulatory proteins to inhibit HBV replication and prevent liver damage; and 3) we are studying new methodologies for therapeutic vaccination to boost the immune response to HBV in people who are chronically infected with the virus.



