



# Kivanç Birsoy, Ph.D.

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**The Birsoy lab studies how metabolic pathways regulate biological processes and contribute to diseases including cancer, mitochondrial disorders, and inborn errors of metabolism. Using genetic and metabolomic tools, Birsoy studies the mechanisms by which human cells alter their uptake and use of nutrients to adapt to the genetic and environmental stresses observed in these disorders.**

Through a series of chemical reactions collectively known as metabolism, an organism extracts and harnesses energy from organic matter. While the core components of this process are relatively well understood, little is known about how an individual cell rewires its metabolic pathways under varying circumstances, including disease. Using forward genetic approaches, Birsoy's lab studies the regulation of metabolism in mammalian cells with the long-term goal of developing therapies for relevant diseases. His group studies cellular metabolism in the contexts of cancer and inborn errors of metabolism.

There is increasing evidence that genetic alterations modify the metabolic program of cells. Since cancer cells are dependent on these changes in metabolism for proliferation, there has been a great interest in exploiting these metabolic liabilities for cancer therapy. Using functional genomics approaches based on CRISPR, Birsoy's lab is systematically mapping out cancer cell dependencies on nutrients, such as amino acids and lipids, while simultaneously looking for opportunities to exploit them for cancer therapy. Understanding the molecular basis for these dependencies will help unveil new metabolic programs and may aid in the development of innovative strategies for cancer treatment, including traditional compounds designed to inhibit intracellular enzymes as well as nutritional approaches to eliminate cancer-feeding metabolites from the blood.

The Birsoy lab is also interested in understanding spatial organization of metabolic pathways in human cells. Compartmentalization of metabolic pathways within membrane-enclosed organelles provides optimal chemical environments for specific reactions and enables efficient utilization of nutrient resources. As the major oxidative organelle, mitochondria are home to critical anabolic and catabolic processes and maintain cellular redox balance, limiting generation of free radicals. However, how concentrations of different metabolites are maintained in organelles is mostly unknown. This question is particularly relevant for cancer as well as mitochondrial disorders, which are characterized by multi-organ dysfunction. Using a combination of genetic and organellar metabolomic tools, Birsoy's lab is examining how mitochondrial dysfunction affects cellular metabolism to give rise to these various disease phenotypes.

## EDUCATION

B.S. in molecular biology and genetics, 2004  
Bilkent University

Ph.D., 2009  
The Rockefeller University

## POSTDOC

Whitehead Institute for Biomedical Research, 2010–2015

## POSITIONS

Assistant Professor, 2015–2022  
Associate Professor, 2022–2025

Professor, 2026–  
The Rockefeller University

## AWARDS

Sidney Kimmel Foundation Scholar, 2016

Searle Scholar, 2016

Irma T. Hirschl/Monique Weill-Caulier Trust Research Award, 2016

Sabri Ülker Science Award in Metabolism, 2016

NIH Director's New Innovator Award, 2017

March of Dimes Basil O'Connor Scholar, 2017

AACR NextGen Award for Translational Cancer Research, 2017

Pershing Square Sohn Prize, 2018

Pew-Stewart Scholar for Cancer Research, 2018

The Rockefeller University Distinguished Teaching Award, 2019

Vilcek Prize for Creative Promise in Biomedical Science, 2020

Mark Foundation Emerging Leader Award, 2021

ASCB Innovation in Research Award, 2021

Blavatnik National Award Finalist in Life Sciences, 2023

Pew Innovation Fund Investigator, 2023

Chan-Zuckerberg Investigator Award, 2025

## SELECTED PUBLICATIONS

Soula, M. et al. Glycosphingolipid synthesis mediates immune evasion in KRAS-driven cancer. *Nature* 633, 451–458 (2024).

Khan, A. et al. Metabolic gene function discovery platform GeneMAP identifies SLC25A48 as necessary for mitochondrial choline import. *Nat. Genet.* 56, 1614–1623 (2024).

Liu, Y. et al. Autoregulatory control of mitochondrial glutathione homeostasis. *Science* 382, 820–828 (2023).

Wang, Y. et al. SLC25A39 is necessary for mitochondrial glutathione import in mammalian cells. *Nature* 599, 136–140 (2021).

Garcia-Bermudez, J. et al. Squalene accumulation in cholesterol auxotrophic lymphomas prevents oxidative cell death. *Nature* 567, 118–122 (2019).