VANDERBILT
School *of* Medicine Basic Sciences

Houra Merrikh, PhD

Professor of Biochemistry
Professor of Pathology, Microbiology and Immunology

houra.merrikh@vanderbilt.edu Twitter: @merrikh_houra
617-451-1628
https://lab.vanderbilt.edu/merrikh-lab

Biosketch

Houra was born in Iran and grew up in Turkey, and then North Cyprus. She immigrated to the US alone in 1997 at the age of 16. Starting from community colleges, she made her way to Brandeis University where she obtained her PhD in 2009. She then spent less than two years as an NIH postdoctoral fellow at MIT. She started her independent laboratory as an Assistant Professor at the University of Washington, Seattle, in 2011 before moving her lab to Vanderbilt University in 2019. Houra became a full Professor with tenure at the age of 38; a remarkable achievement.

Key Publications

"Inhibiting the Evolution of Antibiotic Resistance," Ragheb MN, Thomason MK, Hsu C, Nugent P, Gage J, Samadpour AN, Kariisa A, Merrikh CN, Miller SI, Sherman DR, Merrikh H. *Mol Cell.* 2019 Jan 3;73(1):157-165.e5

"Replication-Transcription Conflicts Generate R-Loops that Orchestrate Bacterial Stress Survival and Pathogenesis," Lang KS, Hall AN, Merrikh CN, Ragheb M, Tabakh H, Pollock AJ, Woodward JJ, Dreifus JE, Merrikh H. *Cell.* 2017 Aug 10;170(4):787-799.e18

"Accelerated gene evolution through replication-transcription conflicts," Paul S, Million-Weaver S, Chattopadhyay S, Sokurenko E, Merrikh H. *Nature*. 2013 Mar 28;495(7442):512-5

"Inhibiting the evolution of drug resistance & investigating the consequences of conflicts between the replication and transcription machineries"

My research program has two branches: 1) investigating the molecular underpinnings of the conflicts between the replication and transcription machineries, and 2) identifying and inhibiting of evolutionary mechanisms that promote drug resistance.

The focus of the first branch is to determine the impact of replication-transcription conflicts on DNA replication, mutagenesis, and evolution, and determining how cells overcome this problem. Over the years, we have made major breakthroughs in this area, including: (i) identifying the critical factors that make conflicts detrimental, (ii) determining that conflicts induce replisome disassembly multiple times every cell cycle, (iii) showing that conflicts have a powerful impact on the evolution of genome architecture. (iv) demonstrating that conflicts promote temporally and spatially-controlled evolution.

The second branch of the lab is focused on the mechanisms that promote drug resistance, which is a serious problem both in the context of infections and during cancer treatment. Although many groups are working on developing new treatments that kill cells to resolve this problem, history has shown that drug resistance arises regardless of the nature or potency of the treatments administered to patients. We are working on resolving the problem by inhibiting the key driver of drug resistance development; mutagenesis and subsequent evolution.