VANDERBILT
School *of* Medicine Basic Sciences

David Cortez, PhD

Richard Armstrong Professor for Innovation in Biochemistry
Chair, Department of Biochemistry
Associate Director of Basic Sciences Vanderbilt-Ingram Cancer Center
Co-Leader, Genome Maintenance Program

david.cortez@vanderbilt.edu Twitter: @davidkcortez
615-322-8547
https://lab.vanderbilt.edu/cortez-lab

Biosketch

Dr. Cortez earned degrees from the University of Illinois and Duke University before completing postdoctoral studies at the Baylor College of Medicine as a Jane Coffin Childs fellow. In 2002, he joined the faculty at Vanderbilt University.

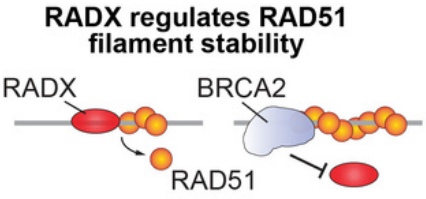
In 2007, he founded and continues to lead the Genome Maintenance Program in the Vanderbilt-Ingram Cancer Center (VICC). He was promoted to Ingram Professor in 2009 and named the Richard Armstrong Professor in Biochemistry in 2020.

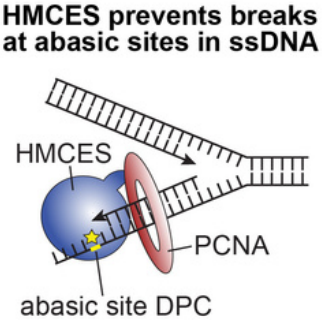
He currently is the Associate Director of Basic Research in the VICC and the Chair of the Department of Biochemistry in the Vanderbilt School of Medicine.

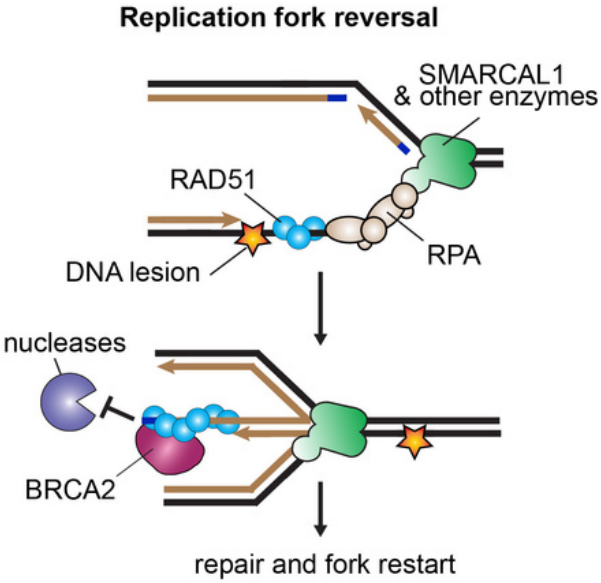
"Exploring DNA Replication Stress Responses and Mechanisms"

Dr. Cortez's research is dedicated to understanding the mechanisms that maintain genome integrity with a particular emphasis on DNA replication stress responses. The lab utilizes a multi-disciplinary approach that includes biochemistry, cell biology, genetics, proteomics, and structural biology. Research topics being addressed include:

- Identification and characterization of replication and repair proteins that help overcome varying types of replication obstacles including DNA damage and conflicts with transcription.
- Define how abasic sites in single-stranded DNA are managed. Studies are underway to define how the evolutionarily conserved protein HMCES acts to protect abasic sites from unwanted processing using a DNA-protein crosslink mechanism.
- Characterize signaling responses to DNA damage and replication stress. The lab is working to understand how the ATR kinase is regulated, and how it signals to control the cell division cycle, DNA repair, and replication fork stability.
- Determine how alternative DNA damage tolerance mechanisms including fork reversal act to maintain genome stability.
- Develop strategies to target DNA damage responses to improve cancer therapy.







Key Publications

"HMCES maintains genome integrity by shielding abasic sites in single strand DNA" 2019, *Cell* 176:144-153

"RADX prevents genome instability by confining replication fork reversal to stalled forks" 2021, *Molecular Cell*, 81:3007-3017

"RAD51 bypasses the CMG helicase to promote fork reversal" 2023, *Science*, 380:382-387