VANDERBILT SCHOOL OF MEDICINE | Basic Sciences

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Biosketch

Jason obtained his undergraduate degree at The University of Chicago and received his PhD in Biochemistry and Biophysics from UCSF. He conducted postdoctoral research at Cornell University using biochemical and imaging approaches to study how cellular proteins are targeted for degradation. In 2013, Jason started his lab in the Department of Cell and Developmental Biology at Vanderbilt to investigate mechanisms of cellular protein degradation in the context of human disease. Jason has been the recipient of the Blavatnik Award for Young Scientists, the NIH Pathway to Independence Award, and a Junior Investigator Award from the American Federation for Aging Research.

Key Publications

"Ubiquitin turnover and endocytic trafficking in yeast are regulated by Ser57 phosphorylation of ubiquitin," *eLife*, 2017, e29176

"COPI mediates recycling of an exocytic SNARE by recognition of a ubiquitin sorting signal," *eLife*, 2017, e28342

"TORC1 Regulates Endocytosis via Npr1-mediated Phosophinhibition of a Ubiquitin Ligase Adaptor," *Cell*, 2011, 147(5): 1104-17

"Seeking a healthy balance among the many paths leading to protein destruction"

The main objective of the MacGurn Lab is to **dissect cellular mechanisms of protein degradation** and ultimately to leverage this knowledge towards the **development of strategies to fight human disease**.

Objective #1: Harness protein degradation pathways to fight cancer.

In the MacGurn Lab, we are learning how to **manipulate protein degradation machinery to target destruction of cancer-driving proteins**. This has led to the identification of novel chemical strategies for **inhibition of important signaling pathways that promote cancer progression**. For example, we have identified one protein degradation switch that we are currently exploring as a possible therapeutic target for **treatment and prevention of advanced forms of prostate cancer**.

Objective #2: Develop strategies for "tuning up" global protein degradation.

In the course of aging, and particularly in neurodegenerative states like **Alzheimer's disease and Parkinson's disease**, our cells suffer a dramatic decline in protein degradation capacity. Furthermore, there is an emerging consensus that restoring degradation capacity can reverse cellular pathologies associated with neurodegeneration and aging. **We have discovered a novel mechanism for "tuning up" global protein degradation in eukaryotic cells, and we are actively investigating how this affects aging in eukaryotic cells.**