Elizabeth Nancy Bess was born in Salt Lake City, Utah. She received her B.S. degree from the University of Utah in Biological Chemistry in 2009. During this time, she performed research on pain perception in the Emergency Department at the University of Utah School of Medicine, worked as an autopsy assistant at the Utah Office of the Medical Examiner, and taught violin lessons to budding musicians.

In 2015, Elizabeth earned her Ph.D. in Organic Chemistry. Under the mentorship of Professor Matthew Sigman at the University of Utah, she developed mathematics-based tools to quantitatively describe and predict the outcomes of catalytic reactions (such as enantio- and site-selectivity) that she was performing in the lab. These tools have been adopted in academia and industry to reduce the trial-and-error associated with chemical-methods development and to garner a quantitative understanding of molecular properties that govern their behavior. Near the end of her graduate career (2014), Elizabeth was a doctoral trainee in the Novartis BioReactions Group in Basel, Switzerland. Here, she first tried her hand at microbiology.

As an HHMI postdoctoral fellow of the Life Sciences Research Foundation, Elizabeth worked in the microbiology lab of Professor Peter Turnbaugh at the University of California, San Francisco in the Hooper Foundation (2015-2018). Here, she elucidated the genetic basis for the cooperative bioactivation of plant lignans by a consortium of bacteria that reside in the human gastrointestinal tract. Using germ-free mice, she demonstrated how strain-level variation in the bacteria colonizing the gut can toggle production of bacterial metabolites that enter the systemic circulation. During her postdoctoral stint, Elizabeth enjoyed leading yoga classes for UCSF scientists.

Elizabeth joined the Department of Chemistry at the University of California, Irvine in Summer 2018. Her lab is fusing chemistry and microbiology to interrogate the chemical mechanisms by which the human gut microbiome impacts human health and disease.

Accumulation of α-synuclein protein aggregates in brain neurons is thought to result in Parkinson’s disease. Despite the clear impact that this disease has on the brain, it seems that the disease as well as α-synuclein aggregates may originate somewhere else—in the gut. Each person’s gut houses trillions of bacteria. Our data show that these gut bacteria start a domino-effect that can cause aggregation of α-synuclein in intestinal cells that natively express this protein. Emerging from our discovery of molecular-level mechanisms detailing how gut bacteria cause α-synuclein aggregation, we identified diet-derived small molecules that inhibit formation of α-synuclein aggregates in intestinal cells. Our discoveries suggest that microbiome-targeted treatments may be developed to slow progression of Parkinson’s disease or even stop the disease in the gut before it impacts the brain.

Elizabeth Bess, PhD (hosted by Jeremy Mills)

Assistant Professor, University of California

Finding Catalysts of Gut Reactions: The Gut Microbiota in Disease Onset and Treatment

*ZOOM option available: https://asu.zoom.us/j/81517529537*