The Foust laboratory is interested in understanding the mechanisms and developing treatments for the neuromuscular diseases Spinal Muscular Atrophy (SMA) and Amyotrophic Lateral Sclerosis (ALS). In addition to skeletal muscle weakness, these SMA patients suffer from gastrointestinal (GI) motility symptoms. These GI motility disorders could be caused by defects in neuromuscular transmission in the enteric nervous system (ENS) but confounding issues have prevented examination in patients. The Foust Lab is utilizing novel genetic and viral approaches to model SMA to better understand the role of survival motor neuron (SMN), the gene implicated in SMA, in neuromuscular signaling in the gut. Additionally, in their models of ALS, they are using adeno-associated viruses to deliver RNAi to knockdown mutant SOD1 which has shown dramatic slowing of disease progression. They plan to develop this clinically and are exploring genes that can be paired with SOD knockdown to further augment the profound survival effect. Come hear about the discovery of a viral vector that crosses the blood brain barrier and how it has been used to target the central, autonomic and enteric nervous systems!