Dissertation title: Aconitase-mediated posttranscriptional regulation in *Helicobacter pylori*

The gastric pathogen *Helicobacter pylori* is remarkable in that it is able to colonize the acidic environment of the stomach, a niche not chronically colonized by any other bacteria. *H. pylori* is notorious for possessing relatively few regulatory systems. It was once thought that the stomach was a relatively stable environment, and without having competition from other bacteria, it was not necessary for *H. pylori* to possess an intricate regulatory system. However, as more evidence becomes available, it appears that *H. pylori* must be metabolically dynamic, as its response to environmental change is multifaceted and mediated by more than one regulatory system. Posttranscriptional regulation in bacteria has increasingly become recognized as playing a major role in the response to environmental stimuli. Aconitase is a bifunctional protein that acts as a posttranscriptional regulator by controlling mRNA stability. In its apo-form, aconitase binds to sequences in either the 5' or 3' untranslated regions (UTRs) of mRNA transcripts. Binding of apo-aconitase to the 5' UTR inhibits ribosome-binding, thereby decreasing translation of the downstream gene. Transcript stability is enhanced when apo-aconitase binds to the 3' UTR of transcripts, where it prevents ribonuclease degradation. The role of aconitase as a posttranscriptional regulator in *H. pylori* has not yet been explored. Here, I propose a global role for aconitase (AcnB) in modulating expression of proteins in *H. pylori*. Putative aconitase targets for regulation include those related to oxidative stress, urease and hydrogenase activities, motility, DNA replication, transcriptional regulation, translation, protein folding, and others.