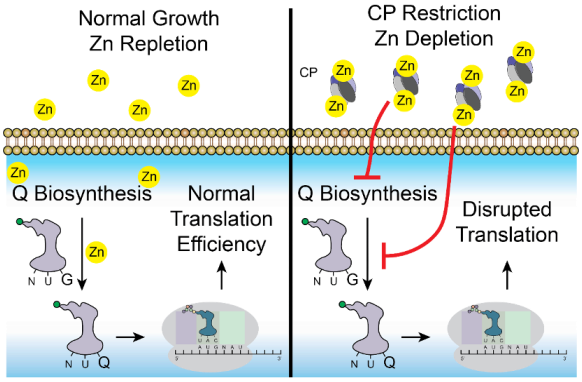
QCB Evenings

Monday, September 27th • 5:00-7:00 pm

The Impact of Zn Limitation on Translation Profiles in *Acinetobacter baumannii*

❖ **Matthew Jordan, Giedroc Group**

Transition metals are required for all forms of life, playing roles as catalytic or structural cofactors. As part of the innate immune response to a foreign pathogen, the vertebrate host recruits the high affinity metal chelator protein calprotectin (CP) to the site of infection to withhold these essential transition metals, notably Zn, thereby starving the invading bacteria. Previously, we observed that CP-mediated Zn sequestration of the major human pathogen *Acinetobacer baumannii* lead to increases in protein abundance of enzymes in the queuosine biosynthesis pathway, suggesting that transition metal restriction reduces flux through the pathway. Queuosine (Q) is an extensive 7-deazaguanine tRNA modification found in certain anticodons that stabilizes near-cognate codon-anticodon interactions, thereby enhancing translation rates. Here, we show that *A. baumannii* has a high requirement for Q modification and exhibits slower growth rates in the absence of Q. Additionally, CP-induced Zn starvation inhibits Q biosynthesis, likely leading to decreased protein translation rates. We further structurally and enzymatically characterize the protein responsible for the committed step of the pathway, 6-carboxypterin synthase QueD2, and evaluate it as the possible point of undermetallation in the Q biosynthesis pathway. Ultimately, we identify the requirement of an essential nutrient, Zn, for the fidelity of protein translation rates in a major human pathogen.



Roles of c-di-AMP in the Cellular Physiology of *Streptococcus Pneumoniae*

❖ **Abigail Garrett, Winkler Lab**

Second messengers such as cyclic-di-AMP (c-di-AMP) impact a variety of cellular processes in many bacterial species. Although c-di-AMP has been implicated in pathogenesis, peptidoglycan synthesis, biofilm formation, competence, growth, and morphogenesis in the human pathogen *Streptococcus pneumoniae*, the mechanism behind the effects of c-di-AMP has not been determined. While many reviews and previous literature suggest that changes in ion homeostasis are responsible for these effects, I will present data indicating that this is likely not the case. I will also discuss novel observations of the effects of c-di-AMP on peptidoglycan synthesis, capsule production, overall cellular growth, cellular morphology, and competence. Finally, I will explain my strategy for determining the mechanism behind the effects of c-di-AMP.