Department of Molecular and Cellular Biology

Graduate Seminar MCB*6500

Friday, February 2,2024 @12:45 p.m.

presented by:

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(Advisor: Dr. Cezar Khursigara)

"Investigating the putative interaction between $FtsK_N$ and peptidoglycan remodeling proteins DacC and MltA"

Bacterial cell division requires the precise coupling of DNA replication and remodeling of the cell envelope to produce two daughter cells. In Escherichia coli, cell division relies on a complex and dynamic network of more than 50 proteins, only 10 of which are essential to cell survival (FtsZ, FtsA, ZipA, FtsK, FtsQ/L/B, FtsW, FtsI, and FtsN). FtsK is a bifunctional multi-spanning transmembrane protein that transports double-stranded DNA by its C-terminus (FtsK_C) and coordinates septation by its N-terminus (FtsK_N) through an unknown mechanism. While there is limited functional data for FtsK, current research suggests that FtsK acts as a cell cycle checkpoint, ensuring the division site is free of genetic material before recruiting downstream cell envelope remodeling proteins. Numerous putative interactive partners of FtsK have been identified by the Khursigara lab, many of which are involved in the modification of peptidoglycan, a component of the cell envelope that provides the cell with structure and shape. These include the penicillin binding protein DacC and the lytic transglycosylase MltA, however evidence of direct protein-protein interaction has yet to be identified. In this capacity, we hypothesize that the role FtsK plays in linking early and late cell division phases is central to the regulation of the cell cycle. This research aims to demonstrate that FtsK forms protein contacts with DacC and MltA and that uncoupling its amino acid interactions results in protein complex dissociation and decreased cellular localization. The proposed function of FtsK as a key regulator of the cell cycle offers a novel method of inhibiting cell division, applicable to the development of effective antibiotics and contributing to the understanding of bacterial cell division.