Exploiting the peptidoglycan (PG) sacculus has been an effective antibacterial strategy as the PG biosynthetic machinery is essential in preventing cell lysis. The discovery of novel antimicrobial targets becomes more essential as bacteria gain resistance to these commonly used antibiotics. Endogenous lytic transglycosylases (LTs) catalyze the cleavage of PG to facilitate cell division, and provide insertion sites for cellular machinery. The essential control of autolytic LTs is provided by the decoration of PG via O-acetylation.

PG O-acetylation occurs at the C6 hydroxyl of muramoyl residues of PG. This modification also increases virulence as it inhibits the activity of lysozyme, which serves as the host immune system first line of defense. O-Acetylation in Gram-negative bacteria employs a two-component system of a membrane bound O-acyl transferase (MBOAT) protein, peptidoglycan O-acetyltransferase (Pat) A, and a periplasmic transferase, PatB. PatA remains uncharacterized but is believed to translocate acetyl across the cytoplasmic membrane to subsequently be presented to PatB via an unknown mechanism.

This study aims to express and characterize PatA by complementing it into a PatA deficient strain of \textit{N. gonorrhoeae}, RD5. In addition, conserved potential catalytic residues, such as a conserved MBOAT histidine (H329), will be investigated. Finally, a possible acetyl periplasmic acceptor will be examined by exploiting PatB transferase activity \textit{in vitro}. Understanding the role of PatA in O-acetylation will gain insight into exploiting it as a novel antivirulence target.