The bacterial cell envelope is an essential component to maintain cellular viability, and is composed of the insoluble macromolecule, peptidoglycan. O-Acetylation of peptidoglycan is a vital defence mechanism used by pathogenic bacteria against a host’s innate immune system, as the addition of an acetyl group to the hydroxyl located at the sixth carbon of N-acetylmuramic acid will sterically prevent lysozyme from binding to peptidoglycan. Gram-negative microbes utilize a two protein system to O-acetylate their peptidoglycan: peptidoglycan O-acetyltransferase A and B (PatA & PatB), and it is the later which is responsible for conducting the O-acetylation reaction. Past work has shown that PatB does not readily bind peptidoglycan without the presence of an acetyl group. To circumvent this, using a covalent inhibitor such as methylsulfonyl fluoride to act as an acetyl mimetic may cause PatB to undergo the conformational change allowing it to bind to peptidoglycan. I hypothesize that the active site architecture of PatB is altered in some manner to facilitate the binding and acetylation of peptidoglycan in order to carry out the enzyme’s catalytic function. First, I will grow co-crystals of MSF bound PatB, and collect structural data of the inhibitor bound enzyme. I will then identify a peptidoglycan analog that PatB can bind to with the use of differential scanning fluorometry, due to peptidoglycan’s poor solubility and difficulty to crystalize. Finally, once both of the aforementioned structures have been elucidated, I will compare any structural differences that may exist between these two models to the previously solved apoenzyme.