The O-acetylation of the essential bacterial cell wall polymer peptidoglycan is known to occur in a large number of bacteria including many important human pathogens, such as \textit{Staphylococcus aureus}, species of \textit{Enterococcus}, \textit{Bacillus anthracis}, \textit{Helicobacter pylori}, \textit{Campylobacter jejuni}, and \textit{Neisseria gonorrhoeae}. This modification to the C-6 position of \textit{N}-acetylmuramoyl residues of peptidoglycan inhibits the action of muramidases (lysozymes) of innate immune systems in a concentration dependant manner and it totally precludes the activity of the lytic transglycosylases, bacterial autolysins that are involved with the insertion of flagella, pili, and secretion/transport systems, as well as the general biosynthesis and turnover of the peptidoglycan sacculus. We discovered a two-component system for the O-acetylation of peptidoglycan in Gram-negative bacteria. An integral membrane protein, peptidoglycan O-acetyltransferase (Pat) A, is proposed to translocate acetate from cytoplasmic pools of acetyl-CoA through the cytoplasmic membrane to the periplasm for its transfer to peptidoglycan by PatB. In Gram-positive bacteria, such as \textit{S. aureus}, a single protein, O-acetyltransferase (Oat), appears to be a fusion of PatA and PatB to catalyze both the translocation and transfer of acetate for peptidoglycan O-acetylation. In this seminar, the discovery of these enzymes together with their biochemical, structural, and mechanistic characterization will be described. Additionally, preliminary evidence supporting the principle that these enzymes may serve as new antibiotic/antivirulence targets will be presented.