Neurons are large, complex cells with long output projections called axons that are up to a meter long in humans. Thus efficient and precise intracellular transport of proteins and organelles is particularly important in neurons, allowing for complex behaviours such as learning and memory. Indeed, disturbances in transport are key pathological events contributing to neurodegeneration in Huntington, Alzheimer, and Parkinson diseases. In axons continuous, fast transport of cargo by the motor proteins kinesin and dynein on microtubules requires a constant supply of energy in the form of ATP. Interestingly, glycolytic enzymes are attached to fast axonal transport vesicles, where they provide a local energy source for motor proteins. However, how these soluble, cytosolic enzymes are attached to transport vesicles is unknown. Palmitoylation is a reversible posttranslational lipid modification that can influence protein localization and function. Palmitoylation involves the addition of long-chain fatty acids to cysteine residues via a thioester linkage. Interestingly, numerous palmitoyl-proteomic studies suggest that all glycolytic enzymes are palmitoylated. Therefore, I hypothesize that palmitoylation of glycolytic enzymes attaches them to transport vesicles to provide on-board energy to the motors. I will determine which glycolytic enzymes are palmitoylated and if palmitoylation of these enzymes is required for fast axonal transport. The results of this research will provide a greater understanding of the mechanisms involved in glycolytic enzyme palmitoylation and could offer insight into various neurodegenerative diseases.