Degradation of the extracellular matrix (ECM) is a key step in cancer metastasis. One method by which invasive tumour cells migrate and invade through the ECM is by small plasma membrane protrusions, termed invadopodia. These structures are highly concentrated in proteolytic enzymes that cleave and degrade components of the ECM. Membrane-type 1 matrix metalloproteinase (MT1-MMP) is the major contributor to ECM degradation in invasive breast cancer cells and is highly localized at the invadopodial membrane. Trafficking of MT1-MMP to the cell surface has been shown to be regulated by 3 phosphorylation sites on its cytoplasmic tail: Thr$^{567}$, Tyr$^{573}$, and Ser$^{577}$. Additionally, the endocytosis and recycling of MT1-MMP is critical for the maintenance of an invasive phenotype. Phosphorylation of the cytoplasmic tail of MT1-MMP induces its endocytosis from the plasma membrane; however, it is unclear as to what mechanisms regulate the trafficking and recycling of MT1-MMP to invadopodia. I hypothesize that the phosphorylation of the cytoplasmic domain of MT1-MMP influences its intracellular trafficking and recycling to invadopodia, thereby influencing cellular invasion. Nonphosphorylatable mutants of MT1-MMP mutants will be created and transiently expressed in MDA-MB-231 cells. I will characterize the invasion and migratory abilities of MDA-MB-231 cells expressing nonphosphorylatable mutants of MT1-MMP, as well as analyze its cell surface expression at invadopodia. Additionally, the endosomal localization and trafficking of nonphosphorylatable MT1-MMP will be visualized to assess its recycling. This work aims to highlight key residues of MT1-MMP responsible for its trafficking in order to further understand how MT1-MMP function affects invadopodium formation.