Metastatic breast cancer is the leading cause of cancer-related mortality in women worldwide. An important component of the metastatic process is the formation of actin-rich membrane protrusions, known as invadopodia, that mediate the degradation of the extracellular matrix (ECM) surrounding the tumour cells. Degradation is mediated by the targeted secretion of matrix metalloproteinases (MMPs), specifically MT1-MMP, which can proteolytically cleave components of the ECM and promote tumour growth and metastasis. The molecular machinery responsible for transport of MMPs to the invadopodia during cell invasion remains poorly defined, but current models have identified soluble N-ethylmaleimide sensitive fusion attachment protein receptors (SNAREs) as key players in membrane trafficking of MMPs. Specifically, the formation of a Syntaxin 4 (Stx4), Synaptosomal-associated protein 23 (SNAP-23), and vesicle-associated membrane protein 2 (VAMP2) SNARE complex have been implicated in the delivery of MT1-MMP to the surface of invadopodia in breast cancer cells to promote invasiveness. This project aims to characterize the proteins involved in regulating the function of the Stx4-SNAP23-VAMP2 complex during invadopodium formation and tumour cell invasion by isolating this complex from breast cancer cells, under invadopodia-promoting conditions, and identifying associated proteins. Candidate proteins will be identified using a mass-spectrometry-based proteomics approach. Additional research will then be conducted on promising candidates, and will focus on characterizing their function and association with the SNARE complex during invadopodia formation and cell invasion. Overall, the proposed research will serve to improve our understanding of tumour cell invasion and possibly contribute to the development of therapies to target metastatic cancer.