Breast cancer is the most common cancer among women and a leading cause of female cancer deaths worldwide. While breast cancer survival rates have been increasing in developed countries, about 25%-40% of patients eventually develop metastatic cancer – the spread of malignant cells from a primary tumor to distant organs – which remains largely incurable. Central to metastasis is the ability of cancer cells to move and degrade the extracellular matrix. This occurs via the rearrangement of focal adhesions and the formation of degradative structures called invadopodia, both of which require changes in the organization of the actin cytoskeleton. The Nck family (Nck1 and Nck2, collectively Nck) of adaptor proteins is involved in actin cytoskeleton remodeling and were identified as oncogenes 30 years ago. Since then, multiple studies have demonstrated a role for these proteins in cancer invasion and metastasis. In particular, overexpression of Nck is seen in many cancers including ovarian, breast, skin, lung, brain and colorectal cancers. Preliminary data suggests Nck levels are elevated in some breast cancers, and that loss of Nck delays tumor onset in a mouse model of breast cancer. Despite these observations, the mechanism by which Nck1 and Nck2 are involved in cancer remains to be established. My project aims to investigate the signaling mechanisms of Nck in breast cancer using Nck-overexpressing and CRISPR-derived Nck-knockout breast cancer cells as a complementary approach. A better understanding of signaling pathways involved in breast cancer is crucial in developing therapies to prevent and treat it.