Breast cancer is the leading cause of cancer deaths in women worldwide. A recently uncovered mechanism by which cancers promote growth and metastasis is by the release of exosomes, small extracellular vesicles (EVs) carrying molecular cargo that aid in transferring phenotypic traits to recipient cells from cells of origin. This project aims to investigate the role of the ShcD adaptor protein in these processes. ShcD has unique properties among the larger Shc family (ShcA-D), such as the ability to induce ligand-independent hyperphosphorylation of EGFR (epidermal growth factor receptor) and regulate non-canonical EGFR trafficking. ShcD is upregulated in the triple negative breast cancer (TNBC) subtype which has a particularly poor prognosis owing to its high rates of metastasis. We have found that ShcD promotes EGFR hyperphosphorylation and invasion in TNBC cells. ShcD has also been implicated as a regulator of EV secretion in prostate cancer cells and our preliminary findings have identified ShcD interactions with exosome-associated proteins in TNBC cells. My project therefore aims to evaluate a potential role for ShcD in the formation, secretion, and function of exosomes derived from breast cancer cells. I propose to characterize how ShcD overexpression alters the exosome secretion profile and exosomal cargo from TNBC cells as well as effects of seeding recipient cells with these ShcD-overexpressing TNBC cell-derived exosomes. A better understanding of the drivers of cancer progression and metastasis will ultimately enable the development of therapeutics to improve clinical outcomes for patients with TNBC.