Despite advances in breast cancer treatment, metastatic breast cancer remains incurable, and the molecular signals that promote the invasion of cancer cells from primary tumors to secondary sites are poorly understood. During invasion, cells remodel their attachments to the matrix of proteins that surrounds cells. Invading cells also form structures, called invadopodia, that degrade this matrix of proteins so they can move through it. Both of these processes require rearrangement of the actin cytoskeleton. Nck is an adaptor protein that links transmembrane proteins to the actin cytoskeleton. The first SH3 domain of Nck contains a unique DY binding pocket that increases its binding specificity to certain proline rich sequences. Interaction between Nck and the transmembrane protein ADAM19 (a disintegrin and metalloprotease 19) through the DY pocket could play a role in invadopodia formation. Nck is also overexpressed in metastatic breast cancer subtypes and preliminary data shows that overexpression of Nck in a breast cancer cell model increases invadopodia formation and invasion. This project aims to further characterize this cell model to determine how overexpression of Nck increases invadopodia formation and invasion. Nck will also be overexpressed in other breast cancer cells lines to verify the mechanism in distinct breast cancer subtypes. The interaction between ADAM19 and Nck and its role in invadopodia formation will also be examined, including using AX-024, a small molecule inhibitor that binds the DY pocket of Nck. Overall, Nck and specifically its interaction with ADAM19 offer potential targets for the reduction of metastatic breast cancer.