Recently, stress-related factors have been linked to the initiation and progression of breast cancer. One such factor is neuropeptide Y (NPY), a highly expressed member of the pancreatic polypeptide family that is mainly found in the sympathetic nervous system. Since breast tissue is highly innervated by the sympathetic nervous system, it has a constant supply of NPY to stimulate its receptors. NPY activates six G-protein coupled receptors, of which, NPY1R, NPY2R, and NPY5R are the most biologically significant in mammals. Activation of these peptide receptors by corresponding peptide hormone binding has been shown to impact tumour proliferation, migration, and angiogenesis. Hypoxia, which is fundamental for tumour progression and metastasis, is associated with increased NPY receptor levels and activity. This research aims to investigate how pharmacological inhibition of NPY and its receptors, NPY1R and NPY5R, impact cellular migration and proliferation of hypoxic human breast cancer cells. This will be evaluated using migration and proliferation assays with pharmacological inhibition of NPY1R and NPY5R. Also, immunofluorescence techniques will be used to examine NPY receptor expression in hypoxic regions of human breast cancer samples and an in vitro spheroid assay will be conducted to examine the role of NPY in tumour formation by receptor stimulation and inhibition. Overall, a greater understanding of how NPY1R and NPY5R antagonists impact breast cancer cell migration, proliferation, and formation, with a focus on hypoxia, will be an asset in the development of novel therapeutics for the treatment of breast carcinomas.