Regulation of signal transduction pathways by hypoxia in breast cancer subtypes

Breast cancer has a high mortality in women worldwide, and typically metastasis rather than the primary tumor is the cause of death. Tumor cells experience hypoxia, which is accompanied by alterations in cell metabolism and can drive metastasis. YAP and TAZ are two transcriptional co-activators involved in growth, metabolism, and metastasis in cancer. Triple negative breast cancer (TNBC) has a lower survival rate due to the lack of therapeutic targets. Fundamental research exploring the molecular mechanisms in cancer cells and their response to a hypoxic environment may contribute to insights for future clinical treatment. This thesis focused on profiling breast cancer cells belonging to distinct subtypes under acute and chronic hypoxia, investigating the crosstalk between hypoxia regulated pathways and YAP/TAZ signaling in luminal breast cancer versus TNBC cells, and identification of the potential targets of TAZ in breast cancer cells.