Determining the role of ERα36 in the molecular mechanisms of Inflammatory breast cancer stemness

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Inflammatory breast cancer (IBC) is the most aggressive type of breast cancer with a survival rate of 5% beyond 5 years after treatment. The rapid growth and metastasis of IBC leads to a late diagnosis where in most patients the cancer has already reached the lymph nodes and spread into other tissues. Currently, there are no approved targeted therapies specifically for IBC. The great heterogeneity in breast cancer cells includes a cancer stem cell (CSC) component which is believed to contribute to the metastatic behavior in IBC. It has been shown that breast CSC's express an estrogen receptor (ER) variant, ERα36, but do not express the canonical receptor ERα66. Since ERα36 is expressed in triple-negative and other subtypes of IBC, we hypothesize that it has a role in contributing to the aggressive behavior of IBC by enhancing stemness of breast cancer cells. Therefore, we want to study the role of ERα36 in IBC stemness and the molecular mechanisms behind it. To achieve this, we will knockdown ERα36 in IBC cells and perform molecular studies, as well as biological assays used for CSC's characterization. This study will contribute to the understanding of IBC aggressiveness and identification of novel targets for IBC therapies.