Spy 1: A Potential Driving Force of the Breast Cancer Stem Cell Population

Nick Joseph Philbin
University of Windsor, philbin@uwindsor.ca

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Nick Philbin, Bre-Anne Fifield, Lisa A. Porter

Breast cancer is the most commonly diagnosed cancer in women. Despite recent improvements in diagnostics and treatment options, the tremendous heterogeneity of the disease often complicates treatment. Triple Negative Breast Cancer (TNBC) occurs in 10-15% of breast cancer diagnoses and typically has poorer outcomes. This is largely due to lack of targeted therapies and the existence of a population of cells known as breast cancer stem cells (BCSCs); a population known to be high in TNBC. BCSCs are more resistant to therapy and capable of driving patient relapse. Cell cycle mediators may play a key role in driving expansion of this population of dangerous cells. Spy1, a cyclin-like protein, promotes cell cycle progression through the G1/S, and the G2/M phase of the cell cycle and has been shown to be elevated in TNBC patients. Additionally, Spy1 is known to expand the brain tumour initiating cell population in brain cancers. Using an *in vitro* model of TNBC (MDA-MB-231 cell line), the relative abundancy of the BCSC population can be assessed to determine if increased levels of Spy1 can expand the BCSC population resulting in more aggressive, invasive and resistant cancers. BCSCs can be identified using markers known to label this population such as the CD44 high/CD24 low, and ALDH isoforms. This work seeks to determine if Spy1 is capable of regulating the BCSC population and may allow for the development of targeted therapy to increase the survival rate of those diagnosed with TNBC.