The recently emerged concept of cancer stem cells (CSCs) has led to a new hypothesis to explain tumor initiation and progression. The CSCs theory hypothesizes the presence in tumors of a hierarchically organized, relatively rare cell population, which is responsible for tumor initiation, self-renewal, maintenance and resistance to chemotherapy. Recently, we demonstrated that the CD90 stem cell marker is highly expressed in the Hs578-T malignant breast cancer cell line, constituting an interesting marker that should be further studied to assess its actual role in human breast tumor progression. We set out to investigate the functional role of CD90 in normal and breast cancer cell lines by overexpressing its corresponding gene in normal MCF10-A cell line and by knocking down this gene in tumoral Hs578-T breast cancer cell line. For CD90 knockdown in Hs578-T and its overexpression in MCF10-A were used the third generation of lentiviral system following the protocol described for Tiscornia. The doubling time of these cell lines was determined by CyQUANT Cell Proliferation Assay Kit. The levels of RNA and proteins were analyzed by qRT-PCR and Western Blot, respectively. Our results revealed that CD90 overexpressing MCF10-A cell line is able to grow independently of EGF and expresses the Neurokinin 1 (NK1) receptor, which has been shown to exert oncogene functions. In addition, N-cadherin expression is decreased in CD90 knocked down Hs578-T cells, suggesting that this marker might be related with the mesenchymal-epithelial transition. In the present study, we demonstrated that CD90, a marker commonly used to isolate and characterize stem cells, has an important role during breast cancer progression and is a strong and novel candidate for a breast cancer malignancy marker. These results contribute not only to a deeper understanding of the Biology of CSCs but also to the development of optimized therapeutic approaches to breast cancer.

Keywords: breast cancer, cancer stem cells (CSCs), stem cell markers
Support: BNDES, CAPES, CNPq, FAPESP, FINEP, MS-DECIT and MCTI.