INTRODUCTION
Breast cancer (BC) is the most common cancer in women worldwide and the second most common cancer overall. Although it is considered postmenopausal disease, genetic predisposition, aging, gender, menarche age, zero ratio, late menopause, and a family history of breast cancer are still major risk factors for breast cancer.

Breast stem / progenitor cell transformation has been implicated in breast carcinogenesis, and many studies have reported the presence of cancer stem cells (CSCs) in malignant BC. CSCs can positively influence tumor survival, spread of metastases and escape of therapy. In particular, the secretion of interleukins 6 and 8 (IL-6 and IL-8) by tumor-associated fibroblasts, mesenchymal stem cells, and macrophages promotes self-renewal of CSCs in BC, which additionally indicates the role of the tumor microenvironment in cancer progression. Estradiol also affects the breast cancer stem cell (BCSC) population in a paracrine manner, as well as other factors, including metalloproteases (MMPs), insulin growth factor (IGF), platelet growth factor (PDGF), secreted by the surrounding cancer cells, which may affect on proliferation, invasiveness and metastatic spread of BC cells.

CONCLUSION
In conclusion, the data discussed indicate that PR isoforms and ERβ are more compelling targets for the reduction of BCSC populations in human BC. Hence, a better and fuller understanding of other SSRs is required to develop new treatments for BC and control the drug resistance that often entails BCSC.

Preclinical and clinical data indicate that BCSC control progression, invasion, metastasis, and drug and radiation resistance. Consequently, the elimination of the BC is strictly dependent on the elimination of the BCSC. New molecules such as GDC0449 or eribulin have undergone clinical trials for their antitumor stem cell activity. Further preclinical and clinical studies are needed to elucidate the significance of CSC signaling in BC recurrence and therapy resistance.