Abstract

Breast cancer is the most commonly diagnosed form of malignant disease in women. Its progression is influenced by the steroid hormone estrogen, which acts through three receptors: estrogen receptor alpha (ERα), estrogen receptor beta (ERβ), and G protein-coupled estrogen receptor (GPER). ERα and ERβ are nuclear receptors that regulate the expression of target genes, while GPER is a membrane receptor that mediates rapid non-genomic signaling. From a therapeutic standpoint, analyzing estrogen receptor expression is crucial for successful treatment. The presence of estrogen receptors significantly affects treatment outcomes. The expression of GPER in cancer cells has been shown to worsen prognosis. The main signaling pathways activated by classical estrogen receptors and GPER in breast cancer cells, and their influence on proliferation and cancer progression, have been summarized based on available literature. In addition, this text focuses on the mechanisms by which GPER may contribute to the development of resistance to tamoxifen, the most commonly used drug against ER+ breast carcinomas.

Key words: GPER, estrogen receptor, ERα, ERβ, breast carcinoma, G protein-coupled receptor