Journal of Oncology

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Special Issue on

Mechanisms Implicated in Endocrine Therapy Resistance in Breast Cancer

CALL FOR PAPERS

Breast cancer is a disease of complex multifactorial etiology, and its molecular classification is still an important task, especially in regard to disease progression and therapeutic response. Approximately one-third of breast tumors are classified as hormone-dependent or luminal subtypes A and B. The tumors of the luminal A subtype, which present the best prognosis, are estrogen receptor (ER) positive, progesterone receptor (PR) positive, and ERBB2/HER2 negative and express estrogen-regulated genes present in normal luminal epithelial cells. Tumors classified as luminal B are associated with a worse prognosis, express variable levels of ER, and present with low or moderate expression of genes expressed in luminal cells. They can also be HER2-positive and are affected by p53 mutations with a higher frequency than luminal A tumors.

Given the critical role of the estrogen pathway in breast cancer of the luminal subtype, first-line adjuvant treatment is usually endocrine therapy. Several agents are available and can be used alone or in combination. They include tamoxifen, a selective ER modulator with antagonistic but also partially agonistic action, and fulvestrant, a selective ER degrader (SERD) with a more complete antagonistic action on ER and aromatase inhibitors (AIs), which inhibit the enzyme that converts androgens into estrogens. Regardless of endocrine treatment offered, primary or acquired endocrine resistance ends up affecting the majority of metastatic tumors and about 40% of all luminal tumors.

This special issue aims to provide new insights into the molecular pathways and mechanisms implicated in endocrine therapy resistance in breast cancer. We encourage original research and review articles focusing on the potential mechanisms associated with endocrine therapy resistance and how to overcome it.

Potential topics include but are not limited to the following:

- Classification of breast tumors, with a focus on the identification of luminal tumors
- Epigenetic mechanisms of endocrine resistance in breast cancer
- Cyclin-dependent kinase 4/6(CDK4/6) inhibitors (palbociclib and abemaciclib) and mammalian target of rapamycin (mTOR) inhibitors (everolimus) to overcome endocrine resistance
- ESR1 mutations in the mechanism of endocrine resistance
- Growth factor receptors, PI3K/AKT/mTOR and RAF/MEK/ERK pathway activation in the mechanism of endocrine resistance
- Combination of immunotherapy and hormone therapy to overcome endocrine therapy resistance
- New and emerging biomarkers for endocrine therapy resistance in breast cancer
- Immunotherapy in endocrine therapy resistant tumors

Authors can submit their manuscripts through the Manuscript Tracking System at https://mts.hindawi.com/submit/journals/jo/mitrc/.

Papers are published upon acceptance, regardless of the Special Issue publication date.

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