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The Role of Beta-Adrenergic Receptor on Epinephrine induced Metastasis in Breast Cancer

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Letter to the editor

I am writing this letter to bring your concern on "The role of beta-adrenergic receptor on epinephrine-induced metastasis in breast cancer". Breast cancer is one of the most common cancers that affect women globally. Metastasis is a major factor in the morbidity and mortality of breast cancer. Nerve fibers from the sympathetic nervous system are present in organs that serve as key targets for breast cancer metastasis, including the lymph nodes, lung, and bone [1]. A few major molecular subtypes of breast cancer are primarily distinguished based on factors such as growth factor and hormone receptor expression. Despite several developments in the treatment of breast cancer, metastatic disease development continues to be an incurable cause of cancer-related mortality in female patients and almost 20% of patients still experience metastasis and recurrence. For several decades, researchers have been studying the effects of immunotherapy on cancer treatment in preclinical and clinical settings. Their goal is to boost, adjust, and speed up the immune system's ability to recognize and destroy cancer cells [2].

It has been documented that a variety of breast cancer cell lines and tumor samples from patients with the disease exhibit adrenoreceptors (ARs). In a recent study, it was discovered that AR overexpression, specifically β2-AR overexpression, was associated with a poor prognosis for patients with estrogen receptor (ER) positive breast cancer. Immune biomarkers, including the expression of programmed cell death ligand 1 and the grades of tumor-infiltrating lymphocytes, were also significantly lower in these patients [3]. In a small cohort of patients with HER2 breast cancer, the β2-AR level was associated with greater rates of lymph node metastases and worse disease-free survival [4].

After briefing the critical role that beta adrenergic receptors play in the metastasis of breast cancer, particularly under the influence of stress hormones like epinephrine, I want to highlight the use of betablockers. After assessing a study on inhibition of AR receptors and breast cancer, it was suggested that propranolol and terbutaline sulfate combination treatment reversed terbutaline's enhanced effects on all three metastasis features in brain metastatic cells. Mice injected with cells pretreated with propranolol at a non-cytotoxic concentration and treatment duration showed a substantial reduction in brain metastasis in vivo. They demonstrated that triple negative breast-to-brain metastatic cells' in vitro proliferation, motility, and invasion were enhanced with activation of their β2-adrenergic receptors. Propranolol prevented these effects. These results contribute to an increasing amount of data indicating a complicated connection between β-adrenergic inhibition and activation and breast cancer metastasis [5].

A high β2-AR level has been related to lymph node metastases and a negative outcome. In addition to being a potential selective therapeutic target for the aggressive subtype of breast cancer, β2-AR may potentially be a novel biomarker for prognosis prediction in Her2-positive breast cancer. The clinical implications of this could be significant and could represent a novel, cost-effective strategy to improve outcomes for patients with breast cancer, particularly those at high risk of metastasis.

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