

Bisphenol A and Alpha-Zeranol Promote the Proliferation and Tumor Growth of Estrogen Receptor-Independent Pre-invasive Breast Cancer

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Bisphenol A (BPA) and alpha-zeranol (aZAL) are endocrine-disrupting compounds (EDCs) with binding affinity to the estrogen receptor, mimicking the effects of beta-estradiol and promoting the growth, invasion, and stemness of estrogen receptor-positive breast cancers. The effect of these EDCs on the invasion, growth, and stemness of estrogen receptor-independent breast cancers has not been widely studied. The purpose of this study was to investigate whether exposure to bisphenol A and alpha-zeranol would result in the increased growth of estrogen receptor-negative ductal carcinoma in situ (DCIS). MCF10DCIS.com cells were injected into the left and right flank of NU/NU mice with implantations containing cholesterol with beta-estradiol (1mg), bisphenol A (1 mg), or alpha-zeranol (1 mg). Tumor volume and weight were recorded, and the tumors were collected for immunohistochemistry analysis (IHC). IHC was performed with the tumors to analyze for a cell proliferation marker, PCNA. Our results showed that in a tumor xenograft model, there was increased tumor growth and cell proliferation in animals treated with bisphenol A and alpha-zeranol compared to control and estrogen groups. Our findings suggest that exposure to BPA and aZAL promotes the growth and development of pre-invasive estrogen receptor-negative breast cancers. Supported by NIH 5R25ES020721-14 and R03 ES 035958.

