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MISSING CARCINOGENIC LINK BETWEEN BISPHENOL A (BPA) EXPOSURE AND BREAST CANCER

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Bisphenol A (BPA), used in the manufacture of clear plastic bottles and lining of food and beverage containers, has been implicated as a class 2B "Suspected" carcinogen and a teratogen by several countries. Within the cell, BPA interacts with MAPK and NFκB pathways that can lead to several tumorigenic events. Previous studies have either stopped at determining BPA induced DNA damage or cited the involvement of MAPK and NFκB pathways and only high dose BPA exposures have been reported that present non-conclusive tumorigenic evidence. These *in vitro* experiments demonstrate that low dose BPA not only causes single strand DNA breaks (SSBs) at 9nM but also causes more error prone double strand breaks (DSBs) at 17nM in the target cell lines. Author further used MCF-7 Human breast cancer and MCF-10A normal breast epithelial cell lines to compare tumorigenic events of BPA exposure. Being metabolized quickly by the liver to form DNA adducts, it can cause direct DNA damage and also act as an inhibitor of secretory pathway calcium ATPase1 (SPCA1). SPCA1 inhibition impacts the post-translational modification and intra-cellular transportation of insulin like growth factor 1 receptor (IGF1R) to the surface. Collectively these events raise the *in vitro* risk of a normal cell line MCF-10A to become tumorigenic.