The Effect of Pollutant Bisphenol A on Cancer Cell Proliferation

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Bisphenol A (BPA), an environmental pollutant and ubiquitous endocrine disruptor, is associated with obesity, cancer, and childhood neurological disorders. Because BPA has similar structure as estrogen and androgen, and both hormones can bind with their corresponding receptors to drive cell growth and survival, these observations raise the hypothesis that BPA might increase cancer cell proliferation through both the estrogen receptor (ER) and the androgen receptor (AR). To test this hypothesis, the effect of BPA on proliferation of cancer cells cultured at physiological conditions was measured. At low concentrations similar to what are detected in human bodies, BPA stimulated proliferation of cancer cells expressing either ER or AR, suggesting that the low environmentally relevant dose of BPA is harmful to human health. At high concentrations, BPA increased cell proliferation of ER-positive cancer cells but surprisingly inhibited the growth of AR-positive cancer cells. To explore the molecular mechanisms of BPA regulation, the expression of cell proliferation-related genes was analyzed. Upon BPA treatment, gene expression of apoptosis inhibitors and cell-division progression drivers was up-regulated in ER-positive cancer cells, but was down-regulated in AR-positive cancer cells. It was further confirmed at cell level that BPA inhibited the apoptosis of ER-positive cancer cells but stimulated the apoptosis of AR-positive cancer cells. In conclusion, BPA regulates the proliferation of cancer cells expressing ER and/or AR, and its regulation mechanisms through ER and AR are different. The finding that BPA can stimulate the apoptosis of AR-positive prostate cancer cells also provides the foundation on designing the new treatment for prostate cancers.

Awards Won:

Second Award of $2,000