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## The beneficial effects of CAPE and EGCG on breast cancer cell lines

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Oxidative stress is the result of an imbalance between excessive reactive oxygen species and anti-oxidants mechanisms. Recently, the anti-oxidant caffeic acid phenethyl ester (CAPE) and Epigallocatechin Gallate (EGCG) have come to the attention of researchers. CAPE is an active component isolated from the propolis found in honey bee hives. CAPE has been determined to have neuroprotective, anti-oxidant, anti-inflammatory and anti-apoptotic properties. It is nontoxic and is consumed as a traditional medicine. As a potent anti-oxidant and NF- $\kappa$ B inhibitor, CAPE is more effective than the other types of natural flavonoids found in fruits, vegetables and tea. In vivo studies showed that CAPE prevents the formation of ROS, malondialdehyde (MDA) and peroxynitrite. (EGCG) is also an anti-oxidant polyphenol found in green tea. It may have health benefits as a nutritional supplement for cancer, atherosclerosis, diabetes, neurodegenerative diseases and HPV virus infection. Anti-oxidant effects of EGCG protect cells from lipid peroxidation and DNA damage induced by reactive free radicals. EGCG directly interacts with proteins and phospholipids in the plasma membrane and regulates signal transduction pathways, transcription factors, DNA methylation, mitochondrial function and autophagy to show many of its beneficial biological actions. EGCG induces apoptosis in several human cancer cell lines including breast, prostate, lungs, ovaries and liver. In this presentation, the beneficial effects of CAPE and EGCG on MCF-7 and MDA 231 breast cancer cell lines will be discussed.

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## Regulation of iodide secretion to milk by the mammary gland expressed Na<sup>+</sup>/I-Symporter (NIS)

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Activity of the sodium/iodide symporter (NIS) in lactating breast is essential for iodide (I<sup>-</sup>) accumulation in milk. This is an essential activity for the proper development of the suckling newborn, as I<sup>-</sup> in milk is the baby's only source for thyroid hormone synthesis. Independent of lactation, significant NIS up-regulation was also reported in breast cancer, indicating a potential use of radioiodide treatment. All trans-retinoic acid (tRA) is a potent ligand that enhances NIS expression in a subset of breast cancer cell lines and in experimental breast cancer models. Aiming to uncover genetic elements directly regulating NIS expression, we screened evolutionary conserved non coding genomic sequences for responsiveness to tRA in mammary epithelial model cell lines. Here, we report that a potent enhancer in the first intron of NIS mediates direct regulation by tRA stimulated nuclear receptors. In vitro as well as in vivo DNA-protein interaction assays revealed direct association between retinoic acid receptor- $\alpha$  (RAR $\alpha$ ) and retinoid-X-receptor (RXR) with this enhancer. Moreover, using ChIP we uncovered early events of NIS transcription in response to tRA, which require the interaction of several novel intronic tRA responsive elements. These findings indicate a complex interplay between nuclear receptors, RNA Pol-II and multiple intronic RAREs in NIS gene and they establish a novel mechanistic model for tRA induced gene transcription.

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