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Polyphenols can regulate signaling cascades to stop conversion from normal to cancer cells

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Abstract

Polyphenols, such as curcumin, stevia, genistein and resveratrol, has been demonstrated to be effective inhibitors of tumor promotion in mouse skin carcinogenesis with antioxidant properties. We report that polyphenols induce cell shrinkage, chromatin condensation, DNA fragmentation, and cytochrome C induction, characteristics of apoptosis, in immortalized mouse embryo fibroblast NIH 3T3, mouse sarcoma S180 and human hepatocellular carcinoma Hep G2 cells. Many cellular and biochemical effects of polyphenols in mouse fibroblast cells have been reported, such as inhibition of protein kinase C (PKC) activity induced by phorbol 12-myristate 13-acetate treatment, inhibition of tyrosine protein kinase activity, and inhibition of arachidonic acid (AA) metabolism. Here using MCF-7 immortalized cells we demonstrate that phenolic compounds can trigger signal transduction pathways linked to apoptosis, such as caspases, p53, and bcl-2 genes. This programmed-cell death may be considered actually one of the important targets in a preventive approach against cancer. How flavonoids do regulate and control the intracellular signaling cascades considered as relevant targets in a cancer preventive approach remains to be elucidated. The results suggest that, in some immortalized and transformed cells, blocking the cellular signal transduction might trigger the induction of apoptosis; also polyphenols can regulate signaling cascades to stop conversion of a normal cell to a malignant one.