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Chemopreventive effects of sulforaphane is mediated by p38 MAP kinase and caspase-7 activation in ER+ and inflammatory human breast cancer cells

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Objectives

The molecular mechanisms involved in the chemopreventive effects induced by sulforaphane in the MCF-7 human breast cancer cell line and the M13SV1 immortalized human breast luminal epithelial cell line.

Materials and Methods

1. Material

Cell line MCF-7, human breast cancer; M13SV1, human breast luminal epithelial

2. Methods:

p38 MAPKinase assay, western blot, proliferation assay

Results and Discussion

Sulforaphane (SFN) is an antioxidant and a potent stimulator of natural detoxifying enzyme and associated with lowered risk of cancer that is associated with the consumption of cruciferous vegetables. The molecular mechanisms involved in the chemopreventive effects induced by SFN in the MCF-7 human breast cancer cell line and the M13SV1 immortalized human breast luminal epithelial cell line. And SFN reduced proliferation in MCF-7 cells and inhibited the 12-O-tetradecanoylphorbol-13-acetate (TPA)-dependent cyclooxygenase-2 (COX-2) expression in M13SV1 cells. The chemopreventive effects of SFN were associated with p38 mitogen-activated protein (MAP) kinase suggests its important role in cell survival/apoptosis regulation and stabilizationCOX-2. SFN up-regulates p38 in MCF-7 cells and prevented TPA-reduced phosphorylation of p38 in M13SV1 cells, but activated caspase-7 associated with apoptosis in MCF-7 cells. These results suggest that SFN may be alternative candidates for targeted prevention of ER+ and COX-2-induced phenotypes and breast cancer.

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