Sulforaphane is a cancer chemopreventive agent that belongs to a class of plant-based products that contain isothiocyanate moieties. Isothiocyanates can be found in cruciferous vegetables such as broccoli, cabbage, and arugula\textsuperscript{1-2}. Sulforaphane, also known as 4-methylsulfinylbutyl isothiocyanate, exhibits many bioactive properties, including antimicrobial, antioxidative, and anticancer activities. Synthetic **R,S-Sulforaphane** (**S8044**) is an effective chemopreventive agent; it prevents the development and growth of mammary tumors in animal models\textsuperscript{3}. Naturally-occurring isomer **R-Sulforaphane** (**S8046**) is optically active. Much of sulforaphane's anticancer effect occurs through activation of phase II detoxifying enzymes.

Sulforaphane is an inducer of phase II enzymes such as glutathione-S-transferase and quinone reductase\textsuperscript{4-5}. Sulforaphane increases activity and expression of these enzymes as well as \(\gamma\)-glutamyl-transpeptidase in lymphoblastoid cells and prostate cancer cells, inducing apoptosis and inhibiting cell growth\textsuperscript{6-7}.

Sulforaphane also induces apoptosis in other cancer cells lines. In colon carcinoma cells, this compound increases expression of Bax and induces release of cytochrome C and cleavage of PARP, resulting in cell cycle arrest and apoptosis\textsuperscript{8}. In melanoma cells, sulforaphane increases activation of caspases, Bax, and p53 and decreases expression of Bcl-2, NF-\(\kappa\)B, caspase 8, and Bid\textsuperscript{9}. These signaling modifications result in apoptosis and inhibition of cell proliferation.

Other mechanisms that contribute to the anticancer effects of sulforaphane are currently under investigation. One such mechanism focuses on the modulation of epigenetic markers. In colon cancer cells, sulforaphane inhibits activity and increases turnover of histone deacetylases\textsuperscript{10}. In this study, this compound enhances acetylation and degradation of DNA repair enzymes, preventing them from mending double-stranded DNA breaks; this activity induces cell cycle arrest, autophagy, and apoptosis.

References: