Apoptosis inducing anthraquinone rhein and emodin differentially suppress human dehydroepiandrosterone sulfotransferase (hSULT2A1) and phenol sulfotransferases (hSULT1A1) in Hep-G2 and Caco-2 cells

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Abstract: The anti-cancer and apoptosis-inducing drugs rhein (4, 5-dihydroxyanthraquinone-2-carboxylic acid) and emodin (3-methyl-1, 6, 8-trihydro-xyanthrax-quinone) are clinically very important. They modulate cell cycle via tumor suppressor gene, immuno-receptors and ligand activated nuclear receptors. Our recent observation suggests for the first time that 10 days of treatment of either drug with various concentrations (0.01 to 100 μM) differentially suppressed the sulfotransferases (SULTs) activities and protein expressions in human hepatocellular carcinoma (Hep G2) and intestinal carcinoma (Caco-2) cell lines. SULTs are phase II drug metabolizing enzymes which catalyze the sulfuryl group transfer to hydroxyl containing endobiotics and xenobiotics. In the present investigation, dehydroepiandrosterone SULT (hSULT1A1) was markedly suppressed by these drugs in human cells. This is the first time report which demonstrates that rhein and emodin may regulate human SULTs. Our finding has important physiological and clinical implications. It will help in the understanding of the SULTs regulations by clinically important drugs and xenobiotics. In future, these drugs may be used in a better defined manner, taking into account its SULTs suppression effects with possible physiological consequences.

Keywords: Physiological role, rhein and emodin, sulfotransferase, suppression effect

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