Cytotoxicity of Natural Hydroxyanthraquinones: Role of Oxidative Stress

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In order to assess the role of oxidative stress in the cytotoxicity of natural hydroxyanthraquinones, we compared rhein, emodin, danthron, chrysophanol, and carminic acid, and a series of model quinones with available values of single-electron reduction midpoint potential at pH 7.0 (E^1_7), with respect to their reactivity in the single-electron enzymatic reduction, and their mammalian cell toxicity. The toxicity of model quinones to the bovine leukemia virus-transformed lamb kidney fibroblasts (line FLK), and HL-60, a human promyelocytic leukemia cell line, increased with an increase in their E^1_7. A close parallelism was found between the reactivity of hydroxyanthraquinones and model quinones with single-electron transferring flavoenzymes ferredoxin: $NADP^+$ reductase and NADPH: cytochrome P-450 reductase, and their cytotoxicity. This points to the importance of oxidative stress in the toxicity of hydroxyanthraquinones in these cell lines, which was further evidenced by the protective effects of desferrioxamine and the antioxidant *N*,*N*′-diphenyl-*p*-phenylene diamine, by the potentiating effects of 1,3-bis-(2-chloroethyl)-1-nitrosourea, and an increase in lipid peroxidation.