Cytotoxicity of dihydroxybenzenes to human glioblastoma GL-15 cells

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1,2-Dihydroxybenzene (catechol), 1,4-dihydroxybenzene (hydroquinone) and 1,3-dihydroxybenzene (resorcinol) are position isomers. Catechol and hydroquinone, metabolites of benzene, oxidize resulting in semiquinones, quinones and reactive oxygen species (ROS). The objective of this work is to study the toxicity of dihydroxybenzenes to human glioblastoma GL-15 cells. Oxidation rates were measured using a Clark-type oxygen electrode. Cultures of GL-15 cells were treated with different concentrations of dihydroxybenzenes for 72 hours. The control group was not treated. In order to study the role of ROS in hydroquinone-induced cytotoxicity, cells were treated with 200 µM of this compound for 72 hours in the presence of 200 U SOD, 500 IU catalase, or 600 µM deferoxamine. The cell viability was quantified by the MTT test. Hydroquinone (1 mM) oxidized consuming 1.4 ± 0.2 µM $O_2.min^{-1}$. No significant difference was observed for oxygen consumption rates between catechol and hydroquinone. However, resorcinol reacted very slowly with oxygen. In cytotoxic studies the IC_{50} value for hydroquinone was 200.3 ± 149.4 µM, and 5579.6 ± 3467.9 µM for resorcinol. Only resorcinol-induced toxicity was not correlated to quinone formation. Antioxidant enzymes and deferoxamine did not prevent hydroquinone-induced toxicity. These results differed from those obtained previously for catechol, in which toxicity was due to the formation of superoxide and quinones. These results suggest that the toxicity of hydroquinone is provided by quinones but not by ROS.