SUPEROXIDE DISMUTASE PROTECTS HUMAN GL-15 GLIOBLASTOMA CELLS AGAINST THE TOXICITY INDUCED BY 1,2-DIHYDROXYBENZENE

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BACKGROUND: 1,2-dihydroxybenzene (catechol) is a metabolite of benzene that is formed in the presence of oxygen and NADPH by liver microsomes of several mammals, including man. It is important to study the toxicity of this compound because benzene is an occupational hazard and environmental toxicant. AIM: The main objective of this work was to study the toxicity of catechol towards human GL-15 glioblastoma cells. METHODS: Catechol oxidizes spontaneously in 50 mM phosphate buffer, pH 7.4, consuming oxygen and forming free radicals. The rate of the autoxidation was measured following the consumption of oxygen using a Clark electrode, and the quinone formation was observed spectrophotometrically at 320 nm in a cell-free system. The production of superoxide was evidenced by the use of superoxide dismutase (SOD). The toxicity of catechol towards GL-15 cells was studied by the MTT assay. The formation of quinones in culture medium was observed spectrophotometrically at 405 nm. The role of superoxide on the toxicity induced by 200 μM catechol was evidenced by the use of 100 U SOD in cultures treated with this compound for 72 h. RESULTS: Catechol, at 1 mM, oxidizes consuming oxygen in a rate of 1.2 $\pm$ 0.3 μM/min and producing quinones. SOD inhibited the oxygen consumption in 83.7% and the formation of quinones in 37.1%. Catechol was toxic towards GL-15 cells treated with 200 μM of this compound for 72 h, and this toxicity was related to the production of quinones. SOD protected these cells and inhibited the formation of quinones. CONCLUSIONS: The data obtained in this work suggest that the catechol-induced toxicity towards GL-15 cells is due to its autoxidation that produces superoxide radicals. Supported by CNPq