Quinone citotoxicity in *Saccharomyces cerevisiae* cells

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The cytotoxic effects exerted by quinones are of great interest in therapy of many diseases as well as in understanding posterior collateral effect of these drugs. Quinones in general exert their toxicity in biological systems by generating reactive oxygen species (ROS) or by binding to glutathione (GSH). In this study we used two naphtoquinones, menadione (2-methyl1,4-naphtoquinone: vitamin K3) and plumbagin (5-hydroxy-2-methyl-1,4-naphtoquinone), and as a eukaryotic model cells of *Saccharomyces cerevisiae* harboring deficiencies in Gtt1 and Gtt2. In order to understand the toxicity exerted by these two drugs we carried out experiments of cellular viability, followed by determination of reduced (GSH) and oxidized (GSSG) glutathione. Activity of aconitase was also monitored. The results of cellular viability showed that all strains were very sensible to lethal concentrations of plumbagin (7µM) and menadione (20mM). However, after pre-treatments with a low concentration of plumbagin (0,5µM) and menadione (0,5mM), all strains acquired tolerance. With respect to GSH and GSSG levels we observed a decrease in GSH contents followed by an increase of GSSG caused by both drugs albeit with different patterns. In addition, the pre-treatments lead to 50% reduction in GSSG levels thus explaining the acquisition of tolerance. No aconitase activity was observed in treated and non treated cells suggesting that lack of activity can be related to the increase of ROS in cells due to stress conditions.