



Silybin–Phosphatidylcholine Complex Protects Human Gastric and Liver Cells from Oxidative Stress

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Abstract

Background/Aim: Silybin is the main component of silymarin with antioxidant, anti-inflammatory and cytoprotective actions. Our aim was to compare the effect of silybin used as single substance, silybin-phosphatidylcholine complex (SilPho), and derivatives of silybin (MannpSil, GalpSil, GlcpSil, LactpSil) on MKN28 and HepG2 cell viability and cell death, in vitro, after induction of oxidative stress. *Materials and Methods:* Oxidative stress was induced by incubating HepG2 and MKN28 cells with xanthine oxidase in the presence of its substrate xanthine. Cell viability was determined by the 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyl tetrazoliumbromide assay. Determination of Malondialdehyde (MDA) in MKN28 cells was performed by high-performance liquid chromatography. Quantitative analysis of apoptotic cells was carried-out using annexin. *Results:* SilPho and new silybin glycoconjugates did not affect cell viability, while silybin induced about 50% cell death in both MKN28 and in HepG2 cells. Pre-treatment of cells with silybin and new silybin glycoconjugates (before oxidative stress induction) did not affect cell viability, while SilPho had a protective effect. Exposure of MKN28 cells to oxidative stress caused a two-fold increase in cellular MDA concentration compared to untreated cells. Moreover, pre-treatment with SilPho, but not with silybin, significantly prevented oxidative stress-induced increase in cellular Malondialdehyde. Moreover, silybin induced apoptosis potentiated by oxidative stress, while SilPho did not induce any effect. Oxidative stress caused cell death primarily by necrosis, antagonized by SilPho. *Conclusion:* The protective effect of SilPho is partially due to inhibition of radical oxidative species.

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