عنوان مقاله:

β-sitosterol induces reactive oxygen species-mediated apoptosis in human hepatocellular carcinoma cell line

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خلاصه مقاله:

Objective: It is of interest to investigate the anti-proliferative effect of β-sitosterol (BS) on human hepatocellular carcinoma (HepG۲) cell line.Materials and Methods: β-sitosterol treatments (۰.۶ and ۱.۲ mM/ml) were done in HepG۲ and after ۲۴ hr, cell viability was evaluated by MTT assay. Reactive oxygen species (ROS) accumulating potential of BS was assessed by dichloro-dihydro-fluorescein diacetate staining. Morphology related to apoptosis was investigated by acridine orange and ethidium bromide dual staining. Cytochrome c and caspase ۳ expressions were evaluated by immunofluorescence and western blot analyses.Results: β-sitosterol induced cytotoxicity ($p<۰.۰۰۱$) and intracellular ROS in HepG۲ cells in a dose-dependent manner.BS treatments accumulated induced intracellular ROS accumulation which led to membrane damage and mitochondrial toxicity. At the molecular level, BS treatments induced cytochrome c release from mitochondria and enhanced the protein expressions ($p<۰.۰۵$ vs ۰.۶ mM/ml and $p<۰.۰۰۱$ vs ۱.۲ mM/ml) of both caspase ۳ and cleaved caspase ۳.Conclusion: β-sitosterol induced ROS accumulation which plays a critical role in apoptosis via the intrinsic pathway in HepG۲ cells. The present investigation paves the way for further in vivo studies.

کلمات کلیدی:

Liver cancer, β-sitosterol, Reactive Oxygen Species, Apoptosis, Caspase

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