Borax-induced apoptosis in HepG2 cells involves p53, Bcl-2, and Bax

Y. Wei¹, F.J. Yuan¹, W.B. Zhou², L. Wu², L. Chen¹, J.J. Wang¹ and Y.S. Zhang²

¹Experiment Center of Medicine, DongFeng Hospital, Hubei University of Medicine, Shiyan, Hubei, China
²Institute of Liver Surgery, DongFeng Hospital, Hubei University of Medicine, Shiyan, Hubei, China

Corresponding: Y.S. Zhang
E-mail: 57729968@qq.com

Received December 17, 2015
Accepted February 11, 2016
Published June 21, 2016
DOI http://dx.doi.org/10.4238/gmr.15028300

ABSTRACT. Borax, a boron compound and a salt of boric acid, is known to inhibit the growth of tumor cells. HepG2 cells have been shown to be clearly susceptible to the anti-proliferative effects of borax. However, the specific mechanisms regulating this effect are poorly understood. This study aimed to investigate the pathways underlying the growth inhibition induced by borax in HepG2 cells. The effects of borax on HepG2 cell viability were characterized using MTT. Apoptosis was also verified by annexin V/propidium iodide staining. JC-1 dye and western blotting techniques were used to measure mitochondrial membrane potential and p53, Bax, and Bcl-2 protein expression, respectively. Relevant mRNA levels were measured by qRT-PCR. Borax inhibited the proliferation of HepG2 cells in a time- and dose-dependent manner in vitro. The apoptotic process triggered by borax involved the upregulation of p53 and Bax and the downregulation of Bcl-2, which was confirmed by a change in the mitochondrial membrane potential. These results elucidate a borax-induced apoptotic
pathway in HepG2 cells that involves the upregulation of p53 and Bax and the downregulation of Bcl-2.

**Key words:** Borax; HepG2 cells; Apoptosis; p53; Bcl-2; Mitochondrial membrane potential