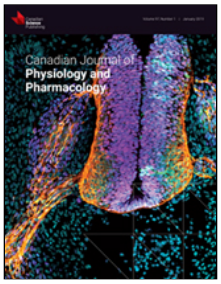

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3-Bromopyruvate potentiates TRAIL-induced apoptosis in human colon cancer cells through a reactive oxygen species- and caspase-dependent mitochondrial pathway

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ABSTRACT

Tumor necrosis factor (TNF)-related apoptosis-inducing ligand (TRAIL) is a promising anticancer cytokine with minimal toxicity towards normal cells. Nevertheless, most primary cancers are often intrinsically TRAIL-resistant or can acquire resistance after TRAIL therapy. This study aimed to investigate the inhibitory effect of co-treatment of 3-bromopyruvate (3-BP) as a potent anticancer agent with TRAIL on colon cancer cells (HT-29). The results of present study indicated that combined treatment with 3-BP and TRAIL inhibited the proliferation of HT-29 cells to a greater extent (88.4%) compared with 3-BP (54%) or TRAIL (11%) treatment alone. In contrast, the combination of 3-BP and TRAIL had no significant inhibitory effect on the proliferation of normal cells (HEK-293) (8.4%). At a cellular mechanistic level, the present study showed that 3-BP sensitized human colon cancer cells to TRAIL-induced apoptosis via reactive oxygen species generation, upregulation of Bax, downregulation of Bcl-2 and survivin, release of cytochrome c into the cytosol, and activation of caspase-3. In normal cells, 3-BP, TRAIL, or combination of both had no significant effect on the reactive oxygen species levels, release of cytochrome c, and caspase-3 activity. Therefore, the combination of 3-BP and TRAIL can be a promising therapeutic strategy for treatment of colon cancer.

Keywords: 3-BP, TRAIL, colorectal cancer, TRAIL resistance, apoptosis

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