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Chemerin-induced oxidative stress triggers apoptosis in HT-29 colon adenocarcinoma cells

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Colorectal cancer (CRC) remains one of the most common cancers and is the third leading cause of cancer-related deaths despite conventional treatments. Chemerin is an endogenous adipocytokine that plays a role in cancer, apoptosis, oxidative stress, and inflammation processes. The aim of this study is to investigate intracellular chemerin signalling in apoptosis, oxidative stress, and inflammation in HT-29 cells treated with exogenous chemerin. The study aims to reveal how chemerin affects these processes and potentially triggers apoptosis in colon cancer cells. Cell viability was determined using the XTT cell proliferation assay after HT-29 cells were treated with 5, 20, and 40 nM chemerin for 48 hours. Oxidative stress (TOS, TAS) and inflammation markers (TNFα and IL1β) were measured using ELISA, while NF-kB mRNA expression was assessed with qRT-PCR. Additionally, CMKLR1, cytochrome c, and caspase-3 gene expressions were quantified using qRT-PCR. PTEN and Bax/Bcl-2 protein expressions were evaluated through Western blotting. Chemerin dose-dependently reduced CMKLR1 levels. Notably, low-dose chemerin increased TOS, OSI, TNFα, IL1β, and NF-kB levels, and low doses were more effective in reducing TAS compared to other doses. Administration of chemerin in human HT-29 cells also resulted in the up-regulation of PTEN, Bax, cytochrome c, and caspase-3, and the down-regulation of Bcl-2. Overall, the study data demonstrated that chemerin induces apoptosis in colon cancer cells by increasing oxidative stress and inflammation through intracellular PTEN, Bax/Bcl-2, cytochrome c, and caspase-3 signalling pathways.

Keywords: Colorectal cancer, PTEN, Bax/Bcl-2, Cytochrome c, Caspase-3