Effect of Sodium Butyrate and Epigallocatechin-3-Gallate on the Genes Expression of Intrinsic Apoptotic Pathway on PA-TU-8902, CFPAC-1, and CAPAN-1 Human Pancreatic Cancer Cell Lines: Epi-drugs and Intrinsic Apoptotic Pathway in Pancreatic Cancer

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Abstract

Background: Histone deacetylase inhibitors (HDACIs) are novel anticancer agents that induce cell death and cycle arrest. Several studies reported that HDACIs induce apoptosis via two well-defined intrinsic/mitochondrial and death receptor pathways. In addition to HDACIs, DNA methyltransferase inhibitors effectively revert the promoter hypermethylation of tumor suppressor genes and apoptosis induction. The current study aimed to investigate the effect of sodium butyrate and epigallocatechin-3-gallate (EGCG) on the genes expression of the intrinsic pathway (BAX, BAK, APAF1, Bcl-2, and Bcl-xL), p21, and p53 on PA-TU-8902, CFPAC-1, and CAPAN-1 human pancreatic cancer cell lines.

Materials and Methods: The PA-TU-8902, CFPAC-1, and CAPAN-1 cells were treated with sodium butyrate and EGCG. To determine cell viability, cell apoptosis, and the relative gene expression level, the 3-(4,4-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) assay, flow cytometry, and real-time quantitative reverse transcription polymerase chain reaction were done, respectively.

Results: Both compounds changed the expression levels of the mentioned genes in a p53-dependent and -independent manner, which induced cell apoptosis and inhibited cell growth in all three cell lines.

Conclusion: We indicated that sodium butyrate and EGCG could induce apoptosis in human pancreatic cancer cell lines.

Key Words: Sodium Butyrate, Epigallocatechin-3-Gallate, Gene Expression Regulation, Pancreatic Cancer