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Impact of the Operation Ciglitazone on PPAR Activation and Apoptosis Process in Models of Gastrointestinal Cancer in vitro

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Abstract

Background: *Peroxisome proliferator-activated receptors (PPAR-γ) are nuclear transcription factors which affect the stimulation of glucose and lipid metabolism, modulation of inflammation, tissue sensitivity to insulin, immune response, cell proliferation and differentiation. Current research on PPAR-γ receptors is contradictory. A significant part of the research suggests that these receptors may be targeted for anti-cancer therapy and have anti-inflammatory properties. Other analyzes speculate on the role of PPAR-γ in promoting cancer. It is therefore important that further studies help to better understand the role of PPAR-γ receptors, which may be relevant in the context of public health and cancer therapy.*

Methods: *The purpose of the study was to determine the effect of ciglitazone (10 μM) on expression of PPAR-γ receptors. In addition, it was investigated whether action on PPAR-γ nuclear receptors with a specific ligand concentration (10 μM of ciglitazone) could lead to increased expression of apoptotic protein (Bcl-2, PKB/Akt) in gastric cancer cells (PANC-1, HT-29) in models In vitro. The effects of ciglitazone were tested in HT-29 and PANC-1 cell lines by MTT growth test (tetrazolium growth assay) for 48 hours post-treatment. Investigation of the relationship between ciglitazone and PPARγ in the context of apoptosis was investigated at the protein level by Western Blot analysis.*

Results: *The results obtained reflect the trend in the publication. In the conducted studies, it was observed that at 10 and 20 μM concentration of ciglitazone affects the growth of the investigated cell lines (PANC-1, HT-29). In addition, studies have shown that this drug increases the activity of PPAR-γ receptors and may affect the kinase gene Akt and Bcl-2 through the receptors themselves.*

Conclusion: *The studies show that treatment of cancer cells 10 μM ciglitazone for a certain time affects the upregulation of anti-apoptotic proteins. What may suggest that certain types of ligand does not result in inhibition of the process of carcinogenesis. Therefore, studies on the effect of PPAR-γ receptors and their ligands on intestinal tumors should be conducted.*

Author Keywords

peroxisome proliferator-activated receptor γ (PPAR-γ), ciglitazone, apoptosis, gastrointestinal cancer

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