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Original Article

Tirzepatide alters oncogenic signaling pathways in colorectal cancer cells *in vitro*

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Abstract

Obesity prevalence is rapidly increasing worldwide, necessitating diverse treatment approaches ranging from pharmacotherapy to surgical interventions. Tirzepatide, a recently approved dual GIP/GLP-1 receptor agonist, has shown therapeutic promise, but its impact on cancer-related pathways remains unclear. This in vitro study investigated the molecular effects of tirzepatide on colorectal cancer SW48 cells by assessing the expression of key regulatory genes, including NF-kB, p53, c-Myc, and CASP8, after treatment with varying tirzepatide concentrations compared to untreated controls. Results demonstrated significant upregulation of the tumor suppressor gene p53 and the pro-apoptotic gene CASP8 (notably a 68.37-fold increase in one treatment group, P = 0.0002), alongside increased c-Myc expression in higher dose groups. These findings suggest that tirzepatide exerts anti-cancer effects in colorectal cancer cells by suppressing NF-kB–mediated inflammation, activating p53-dependent tumor suppression, and promoting CASP8-mediated apoptosis. The concurrent upregulation of c-Myc with p53 and CASP8 highlights potential context-dependent regulatory mechanisms. Overall, this study provides mechanistic insights into tirzepatide's modulation of oncogenic signaling pathways, supporting its potential role in colon cancer therapeutics.

Keywords: Trizepatide, Cancer, Oncogenic pathways, Obesity, p53.

1. Introduction

Nearly 13% of the global population was classified as obese in 2022[1]**.** Treating obesity varied from dietary intervention, medicines such as orlistat and liraglutide, Glucagon-like peptide-1 (GLP-1), to intragastric balloons and bariatric surgery[2]. LP-1 downregulates glucagon levels and enhances insulin release in normal physiological conditions[3]. Tirzepatide, a first-in-class "Twincretin" that is a dual stimulator of glucose-dependent insulinotropic polypeptide (GIP) and GLP-1R and improves blood sugar control in type 2 diabetics (T2D) and it was approved by the U.S. Food and Drug Administration (FDA) on May 13th of 2022. The pharmaceutical company Eli Lilly and Co.® developed the medication, which sold under the trade name "Mounjaro™."[4].

The majority of side effects are gastrointestinal (diarrhea, nausea, and vomiting)[5]. More but less frequent serious adverse effects, including gallbladder problems, including cholelithiasis, cholecystitis, and acute pancreatitis, have also been linked to the use of GLP-1 receptor agonists[6]. A possible carcinogenic action of TRZD is mostly triggered by GLP-1R and GIPR agonists as well as DPP4 (dipeptidyl peptidase 4) inhibitors, which primarily affect the thyroid and pancreas[7].

Kinases for proteins Insulin, insulin-like growth factors 1 and 2 (IGF 1 and 2), and insulin-like growth factor binding protein 3 (IGF-BP3) are all expressed as a result of activation, which also starts the transcription of the gene that codes for insulin. These compounds then use the insulin receptor or insulin-like growth factor receptors to activate key pathways in target cells, such as PI3K/Akt/mTOR, Ras-Raf-ERK, MAPK, and the JAK-STAT pathway increase[8]. If these signaling pathways are abnormally activated, people who have had pancreatitis in the past may be much more likely to develop pancreatic cancer[9]. The breast is one of the other target tissues where these carcinogenic pathways are activated [10], liver[11], and colon, by insulin and insulin-like growth factors. consequently, promoting cellular growing and proliferation, abnormal cancer metabolism, and the prevention of cell death, invasion, and metastasis in these tissues, all of which contribute to the advancement of cancers[12].

Another target for GLP-1 agonists is the parafollicular thyroid C-cells that release calcitonin, also express GLP-1R and GIPR. GLP-1 agonist intervention may increase the incidence of thyroid C-cell cancers in animals, accor-

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ding to experimental data [13]. Liraglutide and exenatide are anti-hyperglycemic incretin mimetics/GLP-1 analogs/agonists that significantly raise levels of cAMP and protein kinase in a dose-dependent manner [14]. Calcitonin is released as a result of activation of thyroid C-cells that activate the expression of the calcitonin gene. C-cell hyperplasia, conversion to C-cell adenomas, and medullary thyroid carcinoma occur in parallel with the prolonged activation of these receptors and calcitonin emission [15].

This study aims to analyze the mechanism of action of tirzepatide in vitro by assessing the gene expression levels of P53, C-MYC, and CASP8 in treated cells compared to control-treated samples.

2. Materials and Methods

2.1. Cell lines and culture conditions

Cell lines used in this study were obtained from Rawafid Aleloom (Hilla, Iraq). SW48 colorectal cancer cells were refreshed with 10% FCS-supplemented DMEM and incubated until confluent. The confluent monolayer was sub-cultured by trypsinization. Sub-confluent cells were plated into 24-well plates at 1×10^5 cells/mL and left to grow for 24 hours.

2.2. Tirzepatide treatment

Cells were treated with tirzepatide (Mounjaro™) at concentrations of 0.05, 0.5, and 5 µg/µL in triplicate for each group. Control cells were treated with serum-free DMEM. Following 24 hours of treatment, cells were harvested for analysis.

Table 1. Composition of reagents for the real-time quantitative PCR reaction.

Material	Volume/Amount
SolGent 2× Master Mix	10 µl
Forward of primers	100 pmol
Reverse of primers	100 pmol
Nuclease-free water	6 µl
Template cDNA	15 ng
Final volume	20 µl

2.3. Cell harvest and morphological examination

Cells were examined under an inverted microscope before and after treatment. Harvesting was performed mechanically using sterile tips; samples were transferred to sterile Eppendorf tubes and stored at -20°C for downstream analysis.

2.4. RNA extraction and cDNA synthesis

RNA isolation from treated and control samples followed the Solarbio protocol (Cat No: R1200). RNA (50 µL, 7 ng/µL) was mixed with 2 µL oligo(dT) and 14.5 µL RNase-free ddH_2O. The mixture was incubated at 70°C for 5 minutes, immediately cooled on ice for 2 minutes, then reverse transcribed at 42°C for 60 minutes. The reaction was stopped at 95°C for 5 minutes and stored at -20°C.

2.5. Gene expression analysis by Real-Time PCR

2.5.1. qPCR reaction setup

Real-Time PCR was performed using a kit from SolGent Co., Ltd. (catalog number 34014, Korea). Reaction components were mixed as follows (Table 1).

2.5.2. qPCR thermal cycling conditions

The cycling protocol comprised (Table 2):

1. Initial denaturation: 94°C for 3 min (1 cycle)
2. Denaturation: 94°C for 30 sec (40 cycles)
3. Annealing: 60°C for 30 sec (40 cycles)
4. Extension: 72°C for 1 min (40 cycles)
5. Final extension: 72°C for 5 min (1 cycle)

2.5.3. Primers used

Specific primers for CASP8, p53, c-Myc, and β-actin (housekeeping gene) were sourced from Macrogen, Korea (Table 3).

2.6. Statistical analysis

Unpaired t-tests using Excel software assessed differences between experimental groups. A p-value < 0.05 was considered statistically significant.

Table 2. Thermal cycling parameters for real-time PCR

No.	Steps	TM	Time	No. of cycles
I	Denaturation I	94°C	3 min	1
II	Denaturation II	94°C	30 sec	
III	Annealing	60°C	30 sec	40
IV	Extension I	72°C	1 min	
V	Extension II	72°C	5 min	1

Table 3. Primer sequences used for quantitative real-time PCR analysis.

Gene		Primers	Company	Origin	References
CASP8	F	5'– AGAAGAGGGTCATCCTGGGAGA – 3'	Macrogen	Korea	[16]
	R	5' TCAGGACTTCCTTCAAGGCTGC- 3'			
P53	F	5' – CCTCAGCATCTTATCCGAGTGG – 3'	Macrogen	Korea	[17]
	R	5' – TGGATGGTGGTACAGTCAGAGC – 3'			
C-MYC	F	5' – TGAGGAGACACCGCCCAC – 3'	Macrogen	Korea	[18]
	R	5' – CAACATCGATTTCTTCCTCATCTTC – 3'			
BA	F	5'- ACTCCTATGTGGGCAACGAG – 3'	Macrogen	Korea	[19]
	R	5' – AGGTGGTGCCAGATCTTC – 3'			

3. Results

3.1. Gene expression analysis

The RNA concentrations and quality across all samples were consistent, ensuring uniform input for analysis. Melting curve analysis confirmed the specificity of primers, with SYBR green producing single, distinct amplification products for each target gene. Dissociation curve analysis using Rotor Gene-Q software verified the presence of specific PCR amplicons with characteristic melting peaks for c-Myc, p53, and CASP8. No amplification was observed in the no-template control (NTC), confirming assay specificity. Fluorescence amplification curves exhibited expected profiles for all genes, including β-actin used as the housekeeping control (Table 3), supporting the reliability of the qPCR data.

3.2. Relative gene expression analysis

3.2.1. P53 gene expression

P53 regulates an essential growth checkpoint that guards against cellular alterations caused by oncogene activation or the depletion of tumor suppressor pathways (Zhang et al., 2020). To quantify gene expression using a qPCR relative assay with SYBR green master mix. In contrast to the control samples, the fluorescence signal was higher in all treated samples Figure 1. After normalizing the control samples to 1, the data were evaluated to determine the fold difference in expression. The gene expression levels in the treated samples were higher compared to the control, with fold changes of 2.7, 0.35, and 0.32, as shown in Figure 2.

3.2.2. c-Myc gene expression

A crucial element in the ontogenesis and development of colorectal cancer (CRC). The gene expression of extracted RNA samples that were reverse transcribed into cDNA template was assessed by combining them with c-Myc-specific primers using a qPCR relative assay with SYBR green master mix. After determining the signal of the house keeping gene, c-Myc expression was lower in group 1 samples but the expression upregulated in group 2 & 3 figure 3. After normalizing the control sample to 1. In comparison to its value in the negative control, it was lower by 0.51 in group 1 samples while higher by 4.79 and 5.79 in group 2 & 3 samples figure 4.

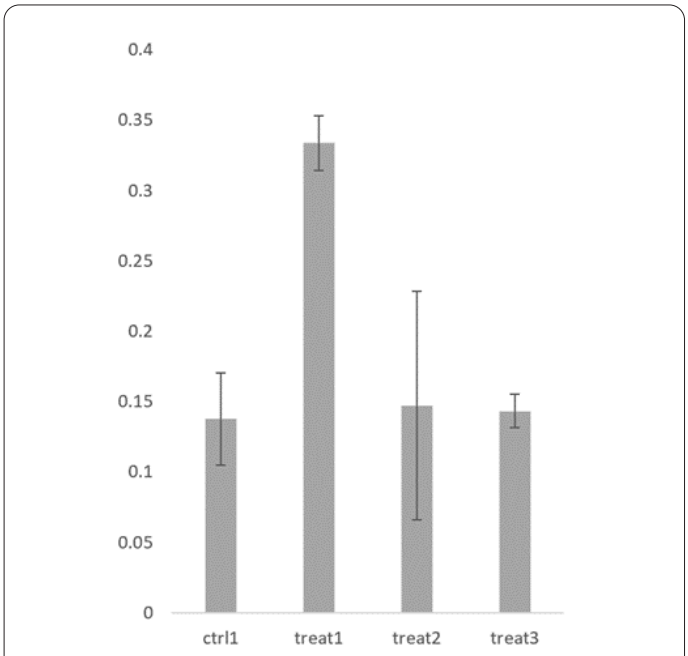

Fig. 1. Relative gene expression of P53 in both positive and negative specimens. To create the cDNA template for the qPCR relative expression test, the extracted RNA was reverse transcribed using a SYBR green master mix. The housekeeping gene (β-actin) was applied to the data, and ΔΔ CTs were used for analysis (A t-test was conducted, yielding a p-value of 0.59).

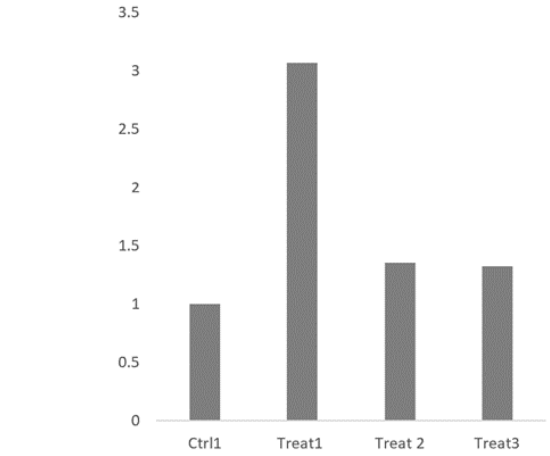

Fig. 2. Fold change analysis of P53 gene expression. Control sample values were normalized to 1, and fold changes in all treated samples were calculated relative to this baseline.

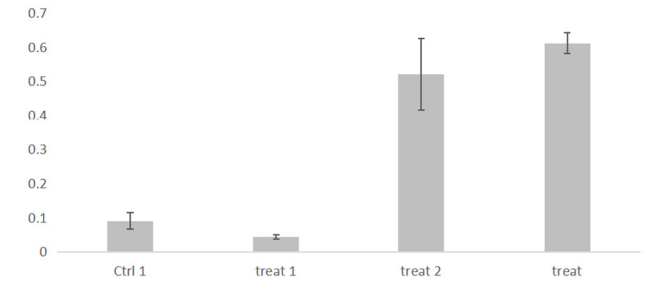

Fig. 3. levels of c-Myc expression in both positive and negative samples. The extracted RNA was reverse transcribed using a SYBR green master mix. The housekeeping gene (β-actin) was applied to the data, and ΔΔ CTs were used for analysis (t-test was conducted at P=0.42).

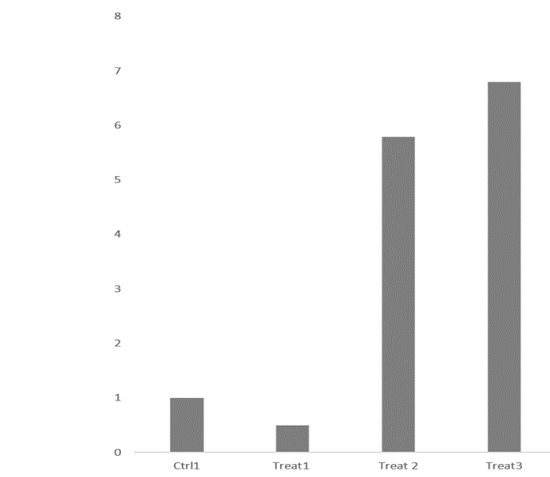

Fig. 4. Fold change gene expression analysis. The value of the control samples was adjusted to 1 and all the treated samples were compared to that 1 to extract the fold change values.

3.2.3. CASP8 gene expression

CASP is essential for both cell death and inflammation. Initially, it was believed to be the cause of extrinsic apoptosis. It has since been found to play a role in several other processes of cell death, including the inhibition of necroptosis, pyroptosis, and intrinsic apoptosis (Orning and Lien, 2021). Hence, CASP8 gene expression was analysed in this study and cDNA templates and SYBR green master mix. SYBR green intercalating dye, particular primers, and cDNA were used to prepare the reaction. The results showed that both group 1 & 3 samples had lower levels of CASP expression than the control sample. Figure 5. Additionally, CASP8 levels in control samples were normalized to 1, and their values in group 1 & 3 samples were 0.07 and 4.46, while in group 2 samples were 68.37, in order to accurately calculate the fold change Figure 6.

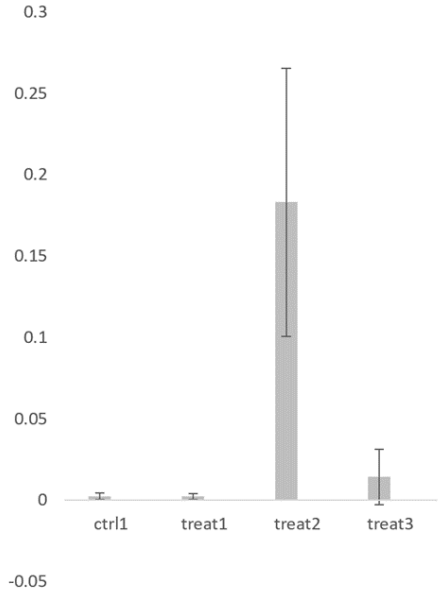

Fig. 5. The degree of CASP8 expression in both positive and negative samples. To create the cDNA template for the qPCR relative expression test, the extracted RNA was reverse transcribed using a SYBR green master mix. The housekeeping gene (β-actin) was applied to the data, and ΔΔ CTs were used for analysis. (t-test was conducted at P=0.0002).

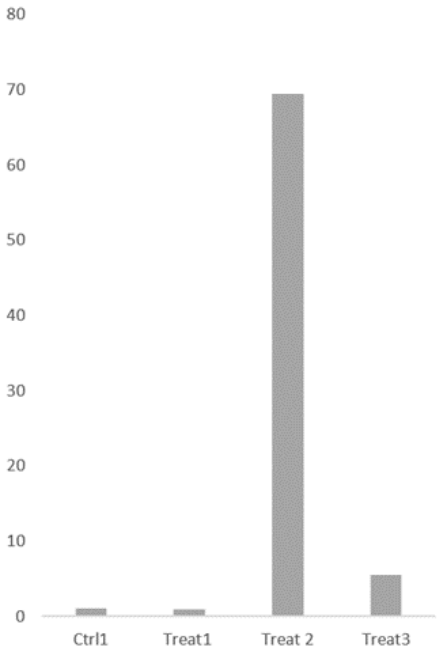

Fig. 6. Fold change gene expression analysis. The value of the control samples was adjusted to 1 and all the treated samples were compared to that 1 to extract the fold change values.

4. Discussion

The current study attempted to investigate the underlying molecular mechanisms behind the cellular effect of tirzepatide through studying its impact on gene expression of various transcription factors that are believed to be involved in the regulation of cell proliferation or induction of apoptosis. While there are many concerns regarding the carcinogenic effects of GLP-1 receptor agonists and their association with thyroid cancers in rodents, clinical studies remain uncertain. Various hypotheses are present that GLP-1 agonists can carry a protective or anti-cancer effect, depending on the cancer type. Studies linking GLP-1RAs with cancers have found that GLP-1 RAs are associated with increased risks of pancreatic and thyroid cancers, while they can decrease the risks of breast, prostate, endometrial, colon, ovarian cancers and various other cancers[20].

The present study revealed an elevated expression of the tumor suppressor gene p53 in the treated colonic cancer cells. p53 is a transcription factor that is distributed in the nucleus and cytoplasm, binds specifically to DNA, and regulates a diversity of genes. Under normal conditions, cellular p53 protein levels are very low owing to strict control by its negative regulators. When cells are exposed to internal and external stresses, including DNA damage, hypoxia, nutrient deprivation, and cancer cell risk, p53 ubiquitination is inhibited, triggering a rapid increase in intracellular p53 protein levels[21]. In response to cellular stress, p53 prevents the differentiation of cells with mutated or damaged DNA and terminates cellular processes by transcriptionally activating various genes involved in apoptosis and cell cycle, which significantly contributes to its tumor suppressor function[21].

In a study conducted by Fidan-Yaylalı in 2016, exenatide was found to activate apoptotic pathway and provide protection in breast cancer cells, where p53 was one of the cellular protective factors that increased upon treatment[22]. On examining the impact of tirzepatide on gene expression of caspase 8, it was found that the level of this pro-apoptotic enzyme was increased significantly in two treatment groups, while it remained almost unchanged in the lowest concentration group. Relative studies found that exenatide binds with GLP-1receptors on ovarian cancer cells and encourages apoptosis by activating caspase 3/7 and inhibits proliferation via inhibiting the PI3K/AKT pathway[23, 24].

A similar mechanism was postulated by Iqbal et al. (2018) [20] while reviewing the preventive effect of GLP-1 receptor agonists on cancer cells. Based on the results of the current study, tirzepatide is suggested to exert a protective effect against colon cancer by activating the tumor suppressor p53 and upregulating the expression of the pro-apoptotic enzyme caspase 8.

The study also showed that treating colorectal cells with tirzepatide upregulated gene expression of c-Myc. Although c-Myc, recognized as an oncogene and exhibits abnormal levels in various types of tumors[25] but in the

context of our study, where an increase in c-Myc is associated with the presence of high expression of p53 and caspase 8, it could be interpreted as an anti-oncogenic effect of tirzepatide[25].

C-Myc is pivotal in driving cellular transformation through two coordinated mechanisms: metabolic reprogramming by inducing changes to meet increased demands for nucleic acids, proteins, and lipids required for rapid proliferation or genetic Coordination by synchronizes expression of gene families that directly accelerate proliferation. Such a dual role establishes c-Myc as a core regulator of the transformed phenotype. C-Myc activation occurs either primarily (via amplification/translocation) or secondarily (as a downstream effector of other oncogenes). In both scenarios, c-Myc sustains the transformational changes underlying malignant progression[26].

The present study demonstrates that tirzepatide exerts anti-cancer effects in colorectal cells through different mechanisms. Firstly, suppression of activation of p53 – enhancing the tumor suppressor's role in stress response, cell cycle arrest, and apoptosis. Secondly, upregulation of Caspase 8 – promoting apoptosis in higher-dose treatment groups and modulation of c-Myc – despite its oncogenic role, its upregulation alongside p53 and Caspase 8 suggests a context-dependent, potentially protective mechanism in this setting.

Limitations

The study is limited by its in vitro model using colonic cancer cell lines, which may not fully capture the complexity of human tumors, including the tumor microenvironment and immune interactions. Additionally, the concurrent increase of the oncogenic c-Myc alongside tumor suppressors warrants further investigation to clarify its role. Furthermore, a dose-response ambiguity was observed, as upregulation of caspase 8 was absent at the lowest tirzepatide dose, suggesting that threshold effects require more comprehensive exploration.

Declaration

Ethics approval and consent to participate

This study was conducted in accordance with ethical standards. All experimental protocols involving cell lines were approved by the relevant institutional review board or ethics committee of the University of Basrah (Approval number if applicable). As an in vitro study, no human or animal subjects were directly involved.

Consent for publication

Not applicable. This manuscript does not contain any individual person's data.

Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests in relation to this work.

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Authors' contributions

N. M. Fadhil and J. H. Kadhim conceived and designed the study. H. W. Atwan and H. A. Alwan performed the experiments and data collection. Z. W. Atwan contributed to data analysis and interpretation. All authors contributed to drafting and revising the manuscript and approved the final version for publication.

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Use of artificial intelligence tools

Some sentences in this manuscript were revised using artificial intelligence language models to enhance clarity and readability. All final content decisions and intellectual contributions remain solely the responsibility of the authors.

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