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Original Article

Pro-inflammatory Cytokine TNF-α Reduces Matrix Calcification in Papillary Thyroid Carcinoma

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ABSTRACT

Aim/Background: Tumor Necrosis Factor-α (TNF-α) is a key pro-inflammatory cytokine involved in immune regulation, chronic inflammation, and tumor progression. However, its role in the calcification process of thyroid malignancies, particularly Papillary Thyroid Carcinoma (PTC), remains unclear. To investigate the effect of TNF-α on matrix calcification and the invasive behavior of papillary thyroid carcinoma cell lines. **Materials and Methods:** Two PTC cell lines, TPC-1 and BCPAP, were treated with varying concentrations of TNF-α. The expression of the calcification marker Osteopontin (OPN) was measured. Alizarin Red and Alkaline Phosphatase staining were used to assess matrix calcification, while cell migration and invasion assays evaluated the malignant potential of the cells. **Results:** TNF-α treatment significantly reduced OPN expression in both TPC-1 and BCPAP cells ($p < 0.05$). Cells treated with 100 ng/mL TNF-α showed decreased calcification and increased invasive and migratory abilities compared to controls and the 1 ng/mL TNF-α group ($p < 0.05$). **Conclusion**: TNF-α suppresses matrix calcification and enhances the invasive potential of PTC cells, suggesting a potential role in the progression and pathogenesis of calcification in thyroid cancer.

Keywords: Thyroid papillary carcinoma, Calcification, Inflammatory cytokines.

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INTRODUCTION

Thyroid cancer is recognized as the most prevalent endocrine malignancy in women, and Papillary Thyroid Carcinoma (PTC) is the most common subtype.[1] Metastasis of PTC predominantly occurs in cervical lymph nodes, with pulmonary metastases being less frequent, and the overall prognosis remains favorable. However, given the substantial global prevalence of this disease and its large patient population, its socioeconomic impact remains profound.[2] Consequently, elucidating the pathogenesis of thyroid cancer is imperative to develop novel clinical therapeutic strategies.

Clinically, calcified foci are observed in 40% of the PTC cases.[3] Although calcification is not exclusive to malignancy and may occasionally appear in benign thyroid lesions, it remains a valuable diagnostic marker for PTC screening. Extensive research has been conducted to elucidate the mechanisms underlying calcification in thyroid cancer.

Previous studies have identified several key proteins involved in this process,[4] including Osteopontin (OPN), Alkaline Phosphatase (ALP), Runx-related transcription factor 2 (Runx-2) and Bone Sialoprotein (BSP). Among these, OPN functions as an extracellular signaling regulatory protein that plays a pivotal role in intercellular communication. It participates in modulating cell growth, differentiation, and apoptosis, and may accelerate carcinogenesis via the NF-κB pathway.[5]

However, the role of inflammatory cytokines in PTC remains poorly characterized. To investigate whether inflammatory factors contribute to calcification in PTC and to explore their molecular mechanisms, we treated PTC cell lines with varying concentrations of Tumor Necrosis Factor-α (TNF-α) to assess their effects on matrix calcification, transcriptional regulation, and matrix calcification dynamics.

MATERIALS AND METHODS

Cell Culture

The cell lines utilized in this study were selected based on references to the Human Protein Atlas (https://www.proteinatlas.org/) and the work of Ferreira.[6] Among these, the human thyroid carcinoma cell line TPC-1, characterized by relatively high OPN expression, served as a representative model, whereas the BCPAP cell line exhibited significantly lower OPN levels, with marked

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differences observed between the two. The cells used in the experiments were purchased from Cell Bank and authenticated by STR profiling.

TPC-1 cells were cultured in Roswell Park Memorial Institute (RPMI) 1640 basal medium (GIBCO, USA), whereas BCPAP cells were maintained in Dulbecco's modified eagle medium (DMEM, GIBCO, USA). Both media were supplemented with 10% fetal bovine serum (FBS, Servicebio, Wuhan, China) and 100 U/mL streptomycin to prepare the complete growth medium. Calcification-inducing medium (PromoCell, UK) was used to induce calcification for up to 21 days, with medium replacement conducted every 48 hr in both cell lines.

RNA extraction, reverse transcription and real time PCR

Total RNA was extracted using TRIzol reagent (Thermo Scientific, Waltham, MA, USA), and mRNA was reverse-transcribed into cDNA using the RevertAid first-strand cDNA synthesis kit (Thermo Scientific, Waltham, MA, USA). Quantitative real-time PCR (qPCR) was performed on the resulting cDNA using the SYBR Green Master Mix (Thermo Scientific, Waltham, MA, USA). The oligonucleotide primers used were based on the research of Liu *et al.*[7] The β-actin gene served as the constitutive control, and the relative expression levels of the target genes were calculated using the Delta-Delta CT method.

OPN AND TNF-α protein expression

The cells were lysed using TRIzol reagent, and the supernatants were collected. Protein samples were separated by electrophoresis on a pre-cast SDS-PAGE gel (Servicebio, Wuhan, China). Following separation, proteins were wet-transferred onto nitrocellulose membranes. The membrane was then incubated in blocking buffer followed by incubation with either mouse anti-human β-actin (Servicebio, GB12001, 1:2,000 dilution, Wuhan, China), rabbit anti-human OPN (Servicebio, GB112328, 1:1,000 dilution, Wuhan, China), or rabbit anti-human TNF-α (Servicebio, GB11188, 1:1,000 dilution, Wuhan, China) overnight at 4°C. Subsequently, the membrane was washed three times and incubated with horseradish peroxidase (HRP)-conjugated anti-rabbit secondary antibody (Servicebio, GB23303, 1:5,000 dilution, Wuhan, China) or anti-mouse secondary antibody (Servicebio, GB 23301, 1:5,000 dilution, Wuhan, China) at room temperature for 1 hr.

For signal detection, a chemiluminescent substrate (ECL Reagent A and B, Abcam, Cambridge, MA, USA) was mixed at a 1:1 ratio and applied to the membrane. The excess liquid was removed using absorbent paper. After developing and fixing, the images were captured and analyzed using the ImageJ software.

Scratch Assay

Thyroid cancer cells were seeded into 6-well plates and resuspended in 2 mL of RPMI 1640 or DMEM complete medium. After 24 hr of incubation at 37°C, a scratch was created perpendicular to the reference lines using a pipette tip. The cells were washed with PBS to remove debris, and serum-free medium was added. The plates were then incubated at 37°C in a 5% CO_2 humidified incubator and observed under a microscope. Images of migrating cells were analyzed using ImageJ software. Six to eight horizontal lines were randomly drawn across the scratch area, and the mean intercellular distance was calculated to quantify the migratory capacity.

Transwell Assay

Thyroid cancer cells in the logarithmic growth phase were counted and resuspended. For the invasion assay, 60 µL of Matrigel (diluted 1:8 in serum-free medium) was uniformly coated onto the basement membrane surface of the Transwell insert and incubated at 37°C for 1 hr to form a gel layer. The migration assay group did not require a Matrigel coating. Subsequently, 200 µL of the cell suspension was added to the upper chamber of the Transwell insert, while 800 µL of complete medium containing 10% FBS was added to the lower chamber as a chemoattractant. After 24 hr of incubation at 37°C in a 5% CO_2 humidified incubator, cells were fixed with 4% paraformaldehyde for 30 min and stained with 0.1% crystal violet (Beyotime, Shanghai, China) for 30 min. The non-migrating cells on the upper chamber were gently removed using a cotton swab. The migrated cells on the lower membrane surface were imaged and quantified using an inverted optical microscope.

Alkaline Phosphatase (ALP) Staining and Alizarin Red Staining

After removing the old culture medium, the cells were washed with PBS to eliminate residual medium and fixed with 4% paraformaldehyde (Servicebio, Wuhan, China) for 10 min. Following fixation, the paraformaldehyde was discarded, and the cells were rinsed three times with PBS. After removing the washing solution, ALP staining (Solarbio, Beijing, China) was performed by incubating the cells with the staining working solution at room temperature in the dark for 30 min. For Alizarin Red staining, 1 mL of 2% Alizarin Red solution (Solarbio, Beijing, China) was added to each well of a 6-well plate and incubated at room temperature for 30 min. The staining reaction was terminated by rinsing with double-distilled water once the desired color intensity was achieved. Finally, the cells were covered with PBS and examined under a light microscope.

Ethical Approval

This study was approved by the Institutional Ethics Committee of the First Affiliated Hospital of Shihezi University (No: KJX-2021-072-01; Date: May 1, 2022).

Statistical Analysis

Data were statistically analyzed using the Statistic Package for Social Science (SPSS) software (version 20.0, IBM, Armonk, NY, USA). Continuous variables conforming to a normal distribution were expressed as Mean±Standard deviation. Group comparisons for normally distributed data were performed using Student's *t*-test or one-way ANOVA. A two-tailed $p < 0.05$ was considered statistically significant for all analyses.

RESULTS

TNF-alpha increases invasion and migration of papillary thyroid cancer cell lines

Based on the data obtained in TPC-1 cells with high OPN expression, we evaluated the expression of OPN.[6] Papillary thyroid carcinoma cells TPC-1 were exposed to different concentrations of TNF-α (0, 0.01 ng/mL, 0.1 ng/mL, 1 ng/mL, 10 and 100 ng/mL) for 48 hr. However, the downregulation of OPN expression induced by TNF-α was not statistically significant, with reduced expression observed only at 100 ng/mL ($p < 0.05$). Although the decline in OPN mRNA levels with 1 ng/mL TNF-α treatment was limited ($p > 0.05$), a stepwise decreasing trend in expression was evident. Consequently, both 1 ng/mL and 100 ng/mL TNF-α concentrations were included in subsequent studies.

Furthermore, Western blotting results demonstrated reduced levels of the calcification marker OPN in TPC-1 cells, with similar observations in the BCPAP cell line (Figure 1). These findings indicate that TNF-α effectively suppressed matrix calcification in papillary thyroid carcinoma cells, with the most pronounced changes in protein levels occurring at a concentration of 100 ng/mL.

TNF-α Enhances the Invasive and Migratory of Papillary Thyroid Carcinoma Cell Lines

Following 24-hr treatment with 100 ng/mL TNF-α, the invasive potential of TPC-1 and BCPAP cells was evaluated using wound healing assay. The results demonstrated a significant increase in the migratory ability of both cell lines after 24 hr of co-culture ($p < 0.001$), with findings representative of at least three independent experiments (Figure 2).

Similarly, in the transwell assays, TNF-α significantly enhanced the cells invasion ability. Under the influence of 100 ng/mL TNF-α, both cell lines exhibited enhanced invasive and migratory capabilities compared to the untreated controls (Figure 3).

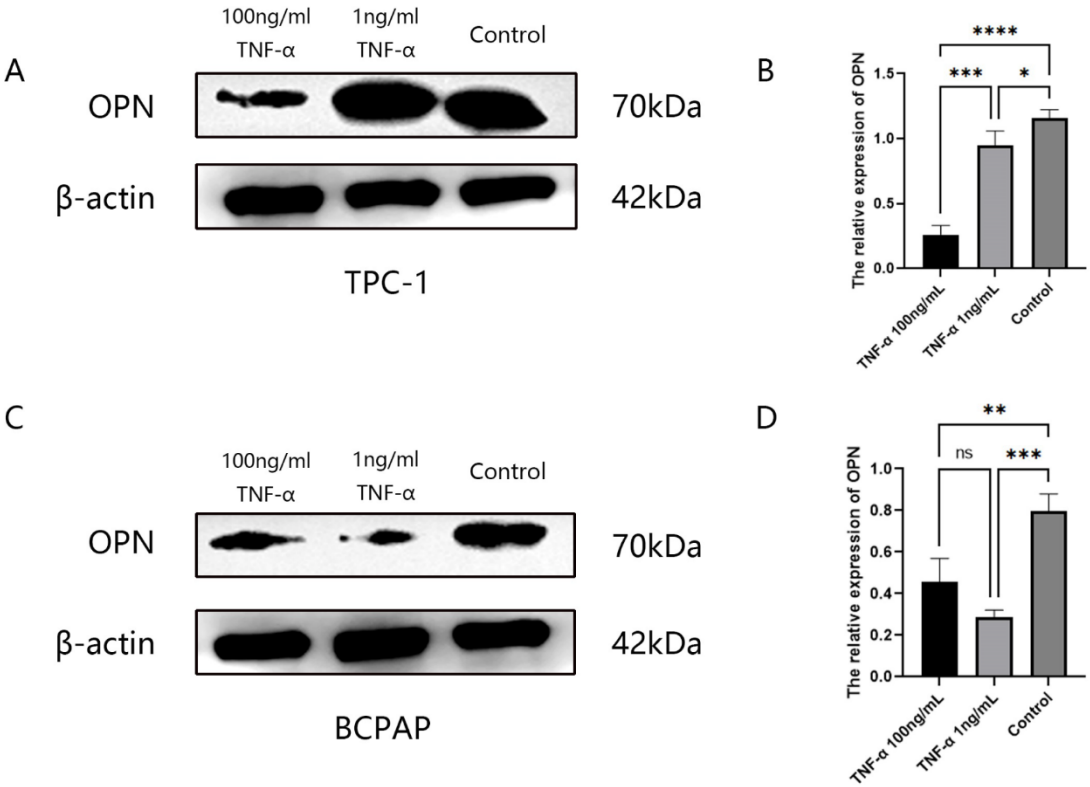

Figure 1: TNF-α significantly suppressed the calcification capacity of PTC cell lines (TPC-1, BCPAP). (A) The mRNA expression of OPN gene after the addition of different concentrations of TNF-α. (B) OPN expression was analyzed via Western blot with β-actin serving as the loading control for normalization. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; ns: no significance.

TNF-α decreases Matrix Calcification in papillary thyroid carcinoma cells

Both cells were cultured in calcification-inducing medium for 7 days and then stained with ALP to detect intracellular alkaline phosphatase. The ability of the two thyroid carcinoma cells to produce calcified Extracellular Matrix (ECM) was assessed by alizarin red staining after 14 days of cell culture.

We found that alkaline phosphatase staining intensity was lower in the TNF-α 100 ng/mL group exhibited significantly lower staining intensity compared to the TNF-α 1 ng/mL group and the control group, especially in TPC-1, whereas in alizarin red staining with longer incubation time, the TNF-α 1 ng/mL group exhibited lower alizarin red staining intensity compared with the control group, while the TNF-α 100 ng/mL group was further lower than the TNF-α 1 ng/mL group (Figure 4).

DISCUSSION

In this study, we investigated the effects of the pro-inflammatory cytokine TNF-α on calcification of PTC cells. Our results demonstrated that TNF-α exposure downregulated OPN expression in PTC cells. Both TPC-1 and BCPAP cell lines exhibited similar responses to TNF-α, and Western blot analyses further corroborated the inhibitory role of TNF-α on OPN, a critical marker of matrix calcification.

TNF-α, widely recognized as a cytokine released during chronic inflammation and immune responses, plays multifaceted roles in inflammatory activation, immune cell modulation, cellular homeostasis, and tumor progression.[8] Initially identified for its anti-tumor properties, accumulating evidence now highlights TNF-α as a key mediator of inflammation-associated carcinogenesis, acting as a pro-tumorigenic factor under specific conditions.[8] Recent studies suggest that dual role of TNF-α in PTC-either promoting or suppressing carcinogenesis-depends on the balance between pro-tumor and anti-tumor cytokine concentrations, as well as receptor expression profiles.[9] In this study, we selected two representative PTC cell lines characterized by predominant RET and BRAF mutations. TNF-α treatment enhanced the invasive and migratory capacities of these cell lines, suggesting its potential to promote PTC progression. This aligns with the findings of Wieczorek *et al.*, where SNAIL protein overexpression induced Epithelial-Mesenchymal Transition (EMT) and promoted thyroid cancer cell invasiveness.[10] Additionally, TNF-α has been shown to upregulate Twist1

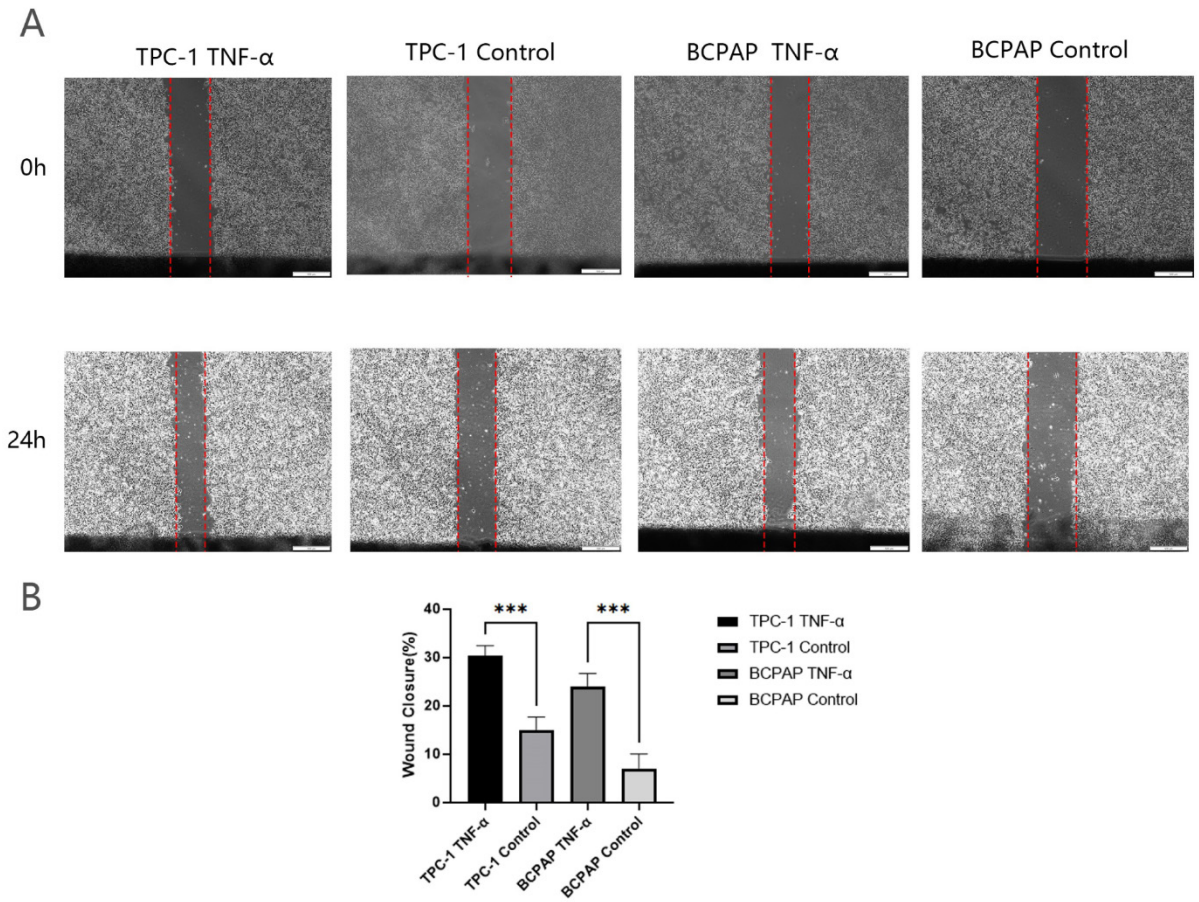

Figure 2: TPC-1 and BCPAP was stimulated with TNF-α (100 ng/mL) for 24 hr and observed using an inverted microscope. *** $p < 0.001$.

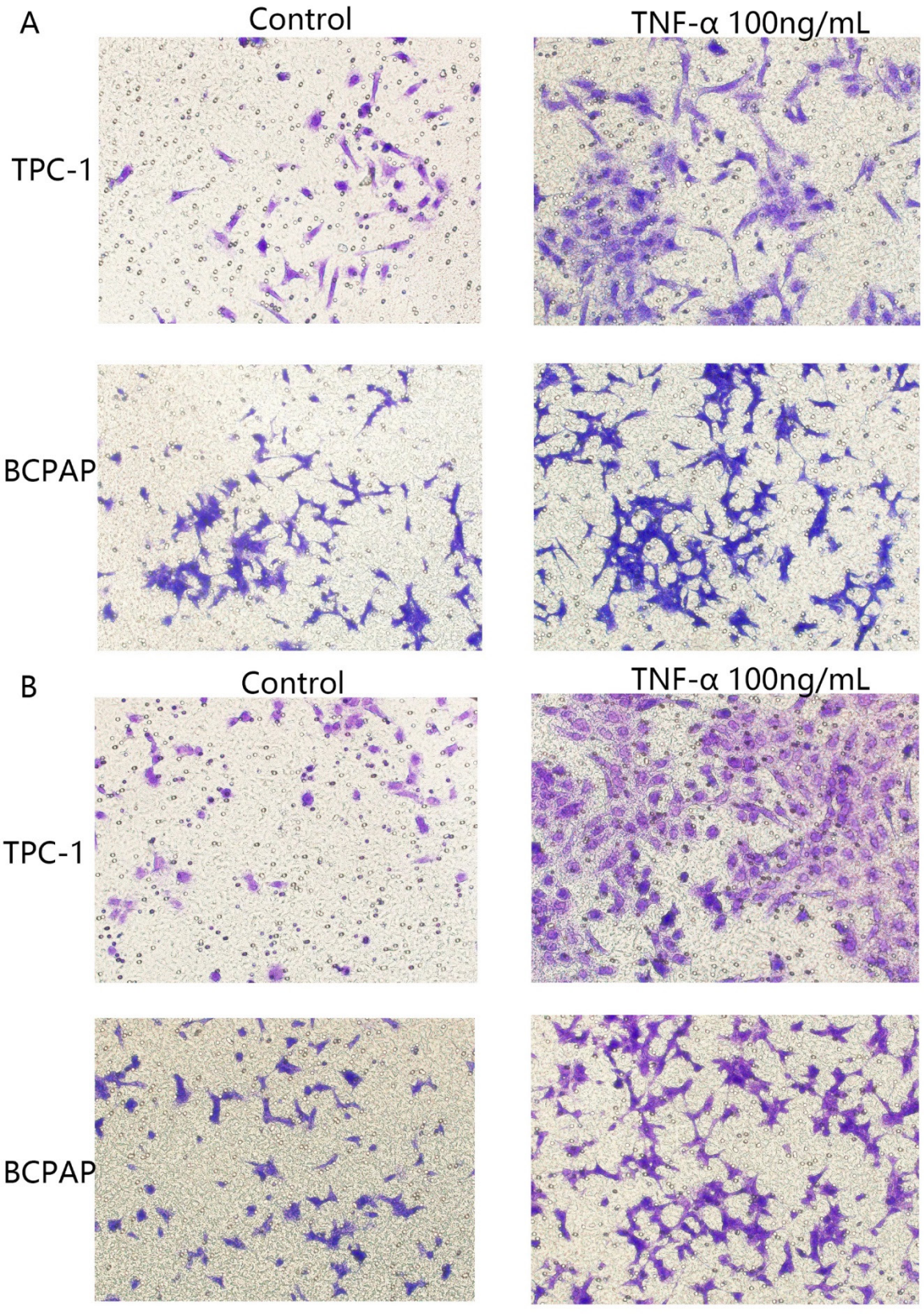

Figure 3: Changes in cell invasion and migration of TPC-1 cell line and BCPAP cell line after TNF-α 100 ng/mL were added. (A) Transwell invasion assays demonstrated a significant increase in invasive capacity in TPC-1 and BCPAP cells treated with 100 ng/mL TNF-α; (B) Transwell migration assays revealed enhanced migratory capacity in both cell lines after exposure to 100 ng/mL TNF-α. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; **** $p < 0.0001$.

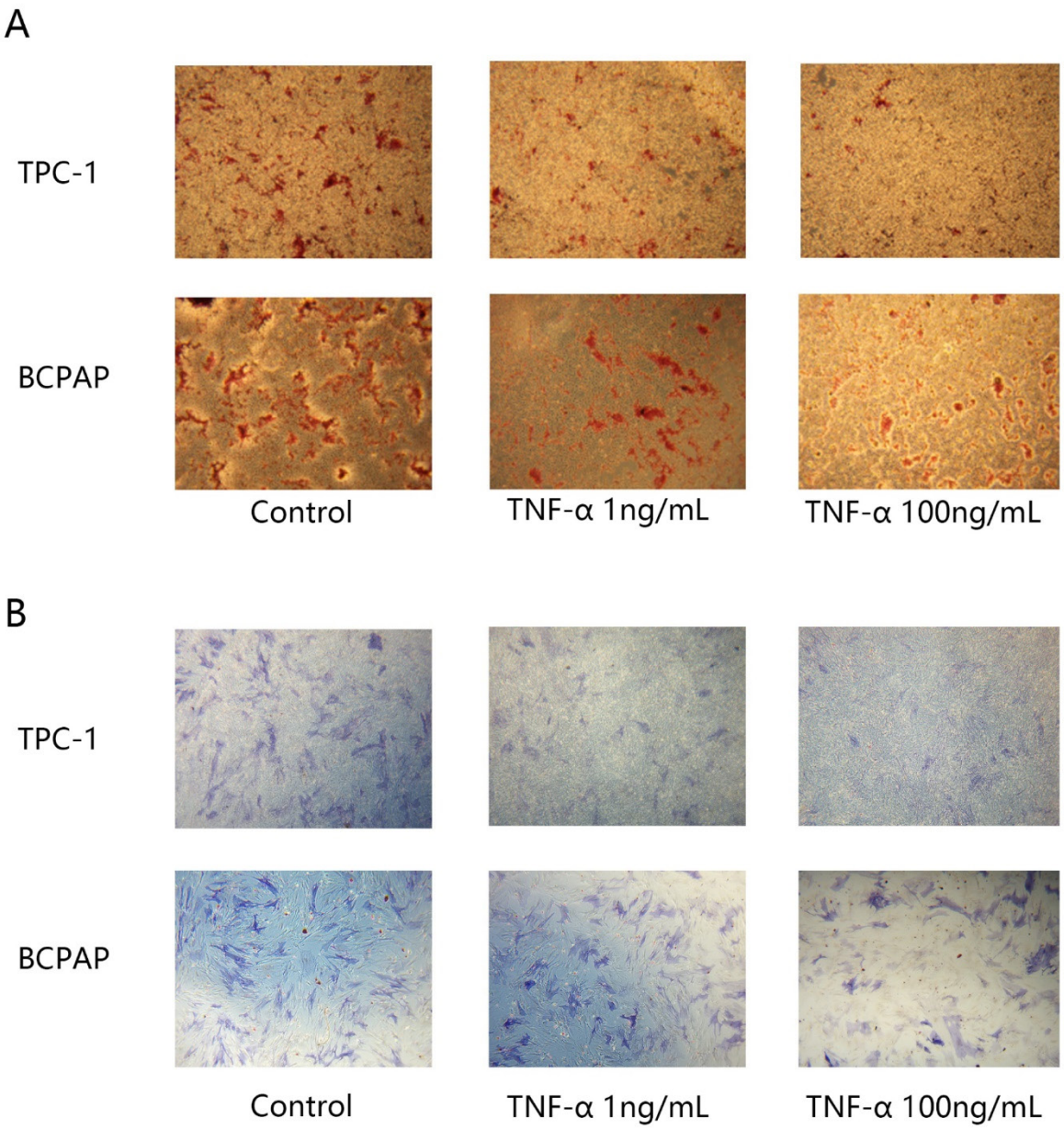

Figure 4: Staining in TPC-1 cell lines and BCPAP cell lines. (A) Alizarin red staining: where the darker orange areas represent areas of the Extracellular Matrix (ECM) rich in calcium deposits. (B) Alkaline phosphatase staining: where blue is the precipitation formed by alkaline phosphatase and chromogenic agent.

expression at both mRNA and protein levels.[11] Although TNF-α and its receptors activate diverse signaling pathways, the central role of NF-κB is well-established. As an upstream regulator of Twist1, NF-κB-mediated modulation of Twist1 has been extensively documented. These findings underscore the complexity of cancer cell invasion and metastasis, implicating multiple interconnected mechanisms.

To further elucidate the involvement of TNF-α in PTC matrix calcification, we observed that TNF-α modulates extracellular matrix calcification levels in TPC-1 and BCPAP cells. Although TNF-α has traditionally been regarded as a primary activator of matrix calcification,[12] our data position it as a calcification inhibitor. This paradox parallels the findings of Hase *et al.*, in which TNF-α exhibited dual roles in promoting calcification while simultaneously stimulating osteoclastogenesis and bone resorption.[13] Such cytokines are elevated in osteoporosis and are implicated in bone loss. Notably, TNF-α activates T cells to dysregulate bone metabolism, increasing osteoporosis risk.[14] Furthermore, hyperthyroidism-associated osteoporosis may correlate with elevated TNF-α levels.[15]

OPN, a multifunctional phosphoglycoprotein involved in bone remodeling and calcification, serves as a key biomarker for assessing calcification.[16] In non-neoplastic bone tissue, OPN is predominantly expressed by osteoclasts and osteoblasts, which

orchestrate bone remodeling. This functional association arises from the calcium-binding properties and hydroxyapatite affinity of OPN. Bailey *et al*. emphasized that phosphate groups of OPN are essential for hydroxyapatite crystal formation during bone matrix calcification.[17] Intriguingly, OPN splice variants have been reported to exert pro-tumorigenic effects in thyroid cancer,[18] consistent with our conclusions.

CONCLUSION

In summary, our findings indicate that OPN is upregulated in PTC, and underscores the correlation between calcification and inflammatory cytokines. Although TNF-α reduces both OPN expression and matrix calcification levels, its overall association with elevated OPN levels in PTC suggests either a weak inhibitory effect or the presence of a more complex regulatory network. Further investigation into the TNF-α/OPN axis may identify potential therapeutic targets for PTC. Our work provides mechanistic insights into calcification in PTC, linking inflammatory cytokines to this process-a relationship warranting close scrutiny in future research.

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CONFLICT OF INTEREST

The authors declared no conflict of interest.

ABBREVIATIONS

TNF-α: Tumor necrosis factor-α; **PTC:** Papillary thyroid carcinoma; **OPN:** Osteopontin; **ALP:** Alkaline phosphatase; **RUNX-2:** Runt-related transcription factor 2; **BSP:** Bone sialoprotein; **NF-κB:** Nuclear factor kappa-light-chain-enhancer of activated B cells; **RPMI:** Roswell Park Memorial Institute; **DMEM:** Dulbecco's Modified Eagle Medium; **FBS:** Fetal bovine serum; **qPCR:** Quantitative polymerase chain reaction; **HRP:** Horseradish peroxidase; **ECL:** Enhanced chemiluminescence; **PBS:** Phosphate-buffered saline; **ECM:** Extracellular matrix; **EMT:** Epithelial-mesenchymal transition; **SPSS:** Statistical Package for the Social Sciences; **mRNA:** Messenger ribonucleic acid; **cDNA:** Complementary deoxyribonucleic acid; **STR:** Short tandem repeat.

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DECLARATION OF GENERATIVE AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

The authors employed an AI-based language model during the manuscript preparation process to assist with linguistic refinement. Final content validation, editorial oversight, and scholarly accountability remain solely with the authors.

SUMMARY

This study investigated the role of Tumor Necrosis Factor-α (TNF-α) in regulating matrix calcification and invasive behavior in Papillary Thyroid Carcinoma (PTC) cell lines TPC-1 and BCPAP. Cells were treated with varying concentrations of TNF-α, and calcification was assessed by Osteopontin (OPN) expression, Alizarin Red staining, and alkaline phosphatase staining. Migration and invasion were evaluated using wound healing and transwell assays. TNF-α at 100 ng/mL significantly reduced OPN expression and matrix calcification while enhancing cell migration and invasion compared with controls and lower-concentration groups. These findings indicate that TNF-α exerts a dual role in PTC, acting as a calcification inhibitor but a promoter of invasive potential. The results suggest that the TNF-α/OPN axis may contribute to PTC progression and could represent a potential target for therapeutic intervention. This study provides novel mechanistic insights into the interplay between inflammation, calcification, and tumor invasiveness in thyroid cancer.

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