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TREM1 PROMOTES GASTRIC CANCER METASTASIS AND LEADS TO POOR PROGNOSIS IN PATIENTS THROUGH THE ERK/IL-6 SIGNALING PATHWAY

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Abstract: Objective: To analyze the expression of TREM1 in gastric cancer tissues and serum of gastric cancer patients, and to explore the link between TREM1 expression levels and the clinical features of patients. Furthermore, to explore the specific mechanism by which TREM1 promotes the metastatic ability of MKN45 cells. Method: Gene expression analysis, differential genes, KEGG enrichment analysis, and immune correlation analysis were performed using TCGA and GEO databases; Compare the concentration of TREM1 in the serum of gastric cancer patients using clinical serum sample standards and group them to analyze the relationship between TREM1 and clinical characteristics and prognosis of patients; Select MKN45 cells, silence the expression of TREM1 and IL-6 using small interfering RNA, overexpress TREM1 using plasmid, and perform corresponding treatments. Compare the changes in cell proliferation and migration ability in different groups using scratch assay and transwell assay; Western blotting was used to detect changes in the ERK signaling pathway, IL-6 expression, and EMT related proteins among different groups; Real time fluorescence quantitative PCR comparison of changes in downstream immune factors of TREM1. Result: TREM1 concentration increased in the serum of gastric cancer patients and was associated with their recurrence, metastasis, and overall survival time; TREM1 promotes invasion and metastasis of MKN45 cells; TREM1 upregulates IL-6 by activating the ERK signaling pathway; TREM1 promotes gastric cancer EMT by upregulating IL-6, leading to enhanced metastatic ability of gastric cancer cells. Conclusion: TREM1 is highly expressed in the serum of gastric cancer patients and upregulates IL-6 by activating the ERK signaling pathway, thereby promoting EMT and invasion and metastasis of gastric cancer, leading to poor prognosis for patients.
Keywords: TREM1; IL-6; Gastric cancer; EMT; ERK signaling pathway; Recurrence and metastasis

1 INTRODUCTION

Gastric cancer is a malignant tumor originating primarily from the gastric mucosal epithelial cells, and its diagnosis relies on gastroscopic pathology [1-2]. Although the widespread promotion of early screening has led to a slight decline in its incidence, according to the report on the burden of malignant tumors in China released by the National Cancer Center of China in 2024, the annual number of new gastric cancer cases in China is 358,700, still ranking fifth among malignant tumors, with annual deaths reaching 260,400, ranking third among malignant tumors [3]. The high incidence and mortality of gastric cancer continue to pose a serious threat to the health of Chinese residents and place a substantial burden on healthcare resources. Currently, the best treatment for gastric cancer remains radical surgical resection. However, due to its insidious onset and high tendency for distant metastasis, patients are often diagnosed at an advanced stage, losing the opportunity for radical surgery[4-5]. Even among patients who undergo radical resection, about 40% experience recurrence and metastasis, significantly affecting survival time and quality of life [6]. Therefore, exploring the potential molecular mechanisms of distant metastasis in gastric cancer and identifying suitable therapeutic targets are critical issues that urgently need to be addressed in clinical practice.

The growth and metastasis of gastric cancer involve a multifactorial, multi-step process, typically including gene mutations, evasion of immune surveillance, malignant transformation and proliferation of cells, angiogenesis, and epithelial-mesenchymal transition (EMT) [7-10]. Among these, immune escape and EMT are central steps in the eventual metastasis of gastric cancer. Multiple studies have shown that various immune factors in the tumor microenvironment, such as IL-1β, IL-6, and IL-8, are involved in the EMT process of gastric cancer[11-13]. However, whether triggering receptor expressed on myeloid cells-1 (TREM1), an upstream regulator of immune factors such as IL-1β, IL-6, and IL-8, participates in the malignant progression of gastric cancer and its underlying mechanisms remain insufficiently explored. TREM-1 belongs to the immunoglobulin superfamily and primarily exerts immunomodulatory effects in its soluble form. It can be synthesized not only by immune cells (e.g., monocytes) but also by some tumor cells [14-15]. his protein has been implicated in the development and progression of several human cancers, including melanoma[16], thyroid cancer[17], and lung adenocarcinoma[18]. This study is the first to investigate the expression of TREM1 in gastric cancer and its relationship with cancer progression, providing preliminary evidence for TREM1 as a potential predictor of metastasis and a novel therapeutic target in gastric cancer patients.

2 MATERIALS AND METHODS

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2.1 Bioinformatics Databases and Analysis Methods

Gene microarray data for gastric cancer were downloaded from The Cancer Genome Atlas (TCGA) database. Differential gene expression analysis and enrichment analysis were performed using data from the GEO database (http://www.ncbi.nlm.nih.gov/geo/(http://www.ncbi.nlm.nih.gov/geo/)). The analysis process was implemented using R packages.

2.2 Clinical Patient Sample Collection and Detection

Blood samples from healthy individuals (n=15) and gastric cancer patients (n=60) were collected at the Affiliated Hospital of Jiangsu University from March 2021 to August 2024. Serum was collected after centrifugation and stored at -80°C (aliquoted to avoid repeated freeze-thaw cycles). This study was approved by the Medical Ethics Committee of the Affiliated Hospital of Jiangsu University (Approval No.: KY2023K0701). Reagents and antibodies required for detection were prepared according to the instructions of the TREM1 ELISA kit. After the test samples were restored to room temperature, operations were performed step-by-step as per the manual. The OD450 value was read after stopping the reaction. A standard curve was plotted to calculate the concentration of TREM1 in the serum. Based on the detection results, gastric cancer patients were divided into a low-TREM1 group and a high-TREM1 group (referencing previous related studies [19], with the cutoff value set at the median of 391 pg/mL).

2.3 Cell Culture and Transfection

Complete medium was prepared: 20% fetal bovine serum + RPMI-1640 basal medium + 1% penicillin/streptomycin. MKN45 cells were cultured in the prepared medium in an incubator set at 37°C with 5% CO_2. MKN45 cells were seeded into 6-well plates. Transfection was performed using Lipofectamine 2000 as the carrier when the cells were in the logarithmic growth phase. Small interfering RNA (siRNA) and plasmids were designed and synthesized by GenePharma (Suzhou). The medium was replaced with complete medium 6-8 hours after transfection.

2.4 Quantitative Real-Time PCR (qRT-PCR)

RNA was extracted and reverse-transcribed into cDNA. Using 2× ChamQ SYBR and 50× ROX Reference Dye I as fluorescent dyes, Ct values were read after amplification. According to the manual, the PCR reaction system (20 μL/well) was set up as follows: 10 μL of SYBR® Premix Ex Taq™; 0.4 μL of 50× ROX Reference Dye I; 0.4 μL each of forward and reverse primers (both at 10 μmol/L); 1 μL of cDNA template; 7.8 μL of sterile, nuclease-free water. The reaction conditions were set as: Pre-denaturation: 95°C for 2 min; Denaturation: 95°C for 15 s, Annealing/Extension: 60°C for 30 s, for a total of 40 cycles. Based on the Ct values, using β-actin as the internal reference gene, the relative transcription levels of TREM1, IL-1β, IL-6, and IL-8 were calculated using the 2^(-ΔΔCt) method.

2.5 Western Blotting Assay

Protein samples were loaded and separated by 12% SDS-PAGE. After electrophoresis, proteins were transferred to a membrane, blocked, and then incubated with primary antibodies and corresponding species-specific secondary antibodies. Following incubation with ECL substrate, images were captured using a chemiluminescence imaging system. α-Tubulin was used as the internal reference protein. Grayscale value quantification of target protein bands was performed using ImageJ software. The antibodies used included: TREM1 antibody (1:1000) and α-tubulin antibody (1:1000) from Beyotime Biotechnology (Shanghai, China); IL-6 antibody (1:1000) from Beyotime Biotechnology; p-ERK antibody (1:1000) from Santa Cruz Biotechnology (USA); ERK antibody (1:1000), E-cadherin antibody (1:1000), and Vimentin antibody (1:1000) from Cell Signaling Technology (USA).

2.6 Transwell Assay

MKN45 cells subjected to different treatments were resuspended in RPMI-1640 basal medium and seeded into the upper chamber of a Transwell insert, with 70,000 cells per well. The lower chamber of the 24-well plate was filled with 500 μL of medium containing 40% fetal bovine serum. After 24-36 hours, the inserts were removed. Cells were fixed with paraformaldehyde, stained with crystal violet, and photographed under an inverted optical microscope. Cells were counted using ImageJ software to determine the average number.

2.7 Wound Healing Assay

MKN45 cells subjected to various treatments were seeded in 6-well plates and monitored for density. Once reaching 95%-100% confluency, the medium was replaced with serum-free medium, and a scratch/wound was created using a 200 μL pipette tip. Images were taken at 0 hours and 24 hours. Cell migration rate was calculated using ImageJ software.

2.8 Statistical Analysis

All data were processed and analyzed using GraphPad Prism 9.02 software. Results are expressed as mean ± standard deviation ($\bar{x} \pm s$). Statistical significance between groups was determined using t-tests or chi-square tests, with $p < 0.05$ considered statistically significant. The symbols *,**,*** indicate $P < 0.05$, $P < 0.01$, and $P < 0.001$, respectively, and "ns" indicates no statistical significance.

3 RESULTS

3.1 Elevated TREM1 Expression in Gastric Cancer and Its Impact on Patient Survival

Bioinformatics results indicated that TREM1 is highly expressed in gastric cancer tissues, and patients with high TREM1 expression had shorter overall survival (Figures 1A-B). Clinical sample results confirmed that the serum concentration of TREM1 was significantly higher in gastric c ancer patients compared to healthy individuals ($P < 0.001$) (Figure 1C).

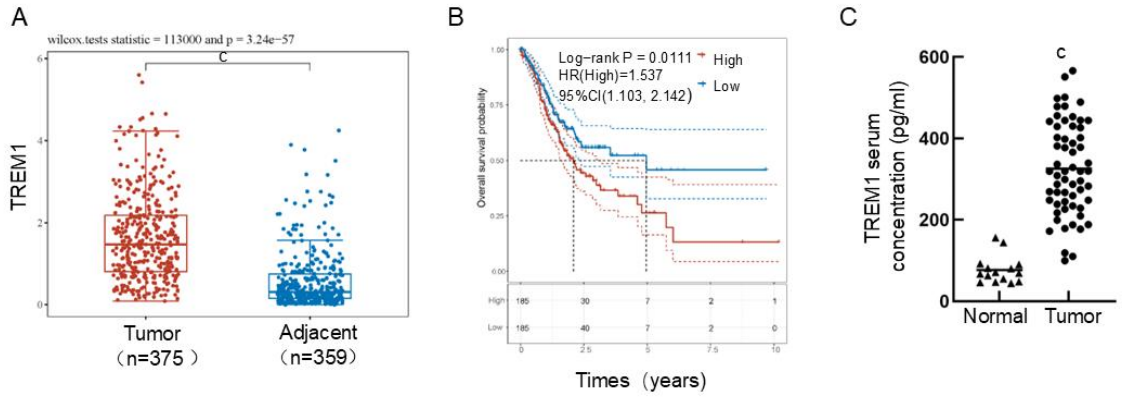

Figure 1 Expression of TREM1 in Gastric Cancer Patients and Its Impact on Patient Survival

A: Box plot comparing TREM1 expression levels in gastric cancer tissues and adjacent normal tissues from a public database; B: Kaplan-Meier curves for the high-TREM1 expression subgroup and the low-TREM1 expression subgroup; C: TREM1 concentration in serum from 15 healthy individuals and 60 gastric cancer patients detected by ELISA.

3.2 Comparison of Serum TREM1 Concentration with Patient Clinical Characteristics

Comparison of clinical characteristics between the two subgroups revealed that TREM1 expression levels were not associated with patient age, sex, histological differentiation grade, pathological stage, or lymph node metastasis. However, a significant association was found with distant metastasis and recurrence (Table 1). Patients in the high-TREM1 expression subgroup were more prone to metastasis and recurrence.

Table 1 Comparison of Clinical Information among Different Subgroups of Gastric Cancer Patients

Variable	Total n = 60	TREM1 expression Low n=29	High n=31	χ^2/t	p
Age	67.53±7.56	67.66±7.69	67.42±7.56	0.12	0.905
Gender				0.010	0.927
Male	40	20	20		
Female	20	9	11		
Differentiation				0.600	0.741
High	24	11	13		
Median	12	7	5		
Low	24	11	13		
Stages				0.650	0.421
I/II	33	18	15		
III/IV	27	11	16		
Lymph node metastasis				1.000	0.317
NO	38	16	22		
Yes	22	13	9		
Distant metastasis				5.770	0.016
NO	35	22	13		

Variable	Total n = 60	TREM1 expression Low n=29	High n=31	χ^2/t	p
Yes	25	7	18		
Recurrence				4.670	.031
NO	36 (60.00%)	22	14		
Yes	24 (40.00%)	7	17		

3.3 Knockdown of TREM1 Inhibits the Invasion and Migration Capabilities of MKN45 Cells

Based on the above findings, in vitro experiments were conducted to further validate the effect of TREM1 on the invasion and migration abilities of gastric cancer cells (Figure 2).

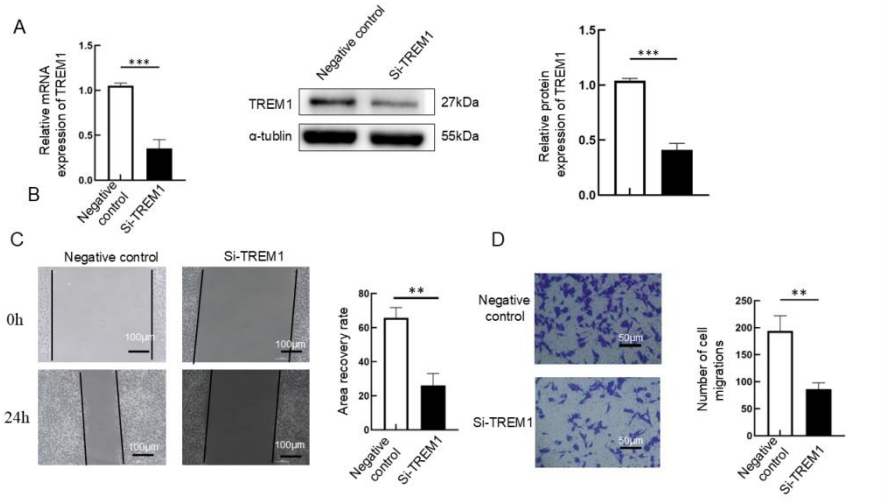

Figure 2 Effect of Interfering with TREM1 on the Migration Ability of MKN45 cells

A-B: Transcriptional (A) and protein (B) expression levels of TREM1 in MKN45 cells after Si-RNA-TREM1 treatment; C-D: Changes in migration ability of MKN45 cells after TREM1 knockdown, as assessed by wound healing assay (C) and Transwell assay (D).

3.4 TREM1 Regulates IL-6 Expression via the ERK Signaling Pathway

To investigate the potential mechanism by which TREM1 promotes gastric cancer metastasis, downstream genes associated with TREM1 were analyzed. Results showed a significant positive correlation between TREM1 and three immune factors: IL-1β, IL-6, and IL-8 (Figure 3A). qRT-PCR results indicated that after TREM1 knockdown, the downregulation trend of IL-6 was the most pronounced (Figure 3B). Subsequent KEGG enrichment analysis revealed that TREM1 is closely associated with the activation of the ERK signaling pathway (Figure 3C), which has been proven to regulate IL-6 expression. Western blot analysis demonstrated that TREM1 knockdown inhibited the activity of the ERK signaling pathway and decreased IL-6 expression (Figure 3D). Conversely, TREM1 overexpression upregulated IL-6 expression, but this upregulation was suppressed when ERK signaling activity was inhibited (Figure 3E).

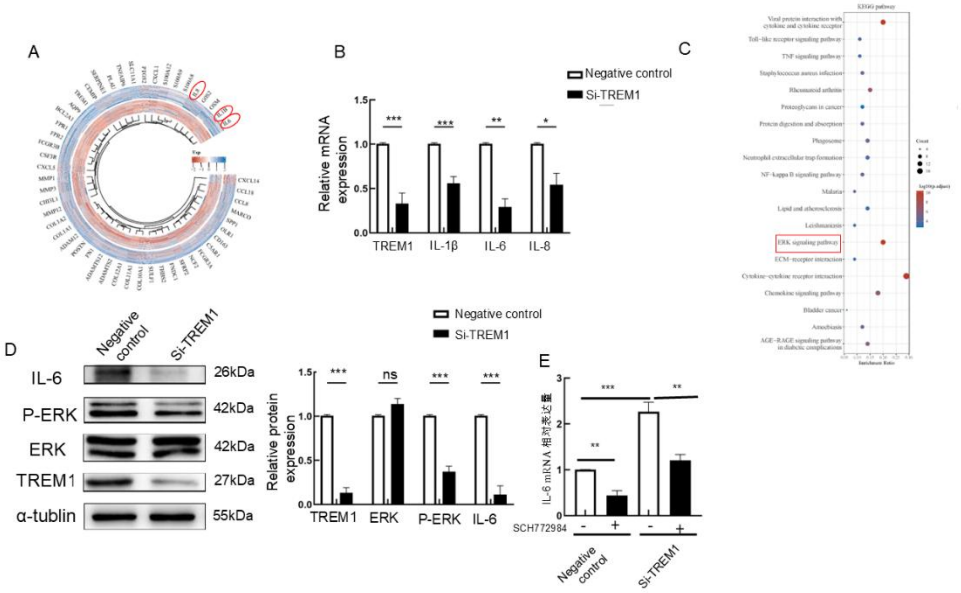

Figure 3 Differential Genes Downstream of TREM1 and Related Pathways Regulating IL-6

A: Differentially expressed genes (DEGs) between high-TREM1 and low-TREM1 expression groups, B: Transcript levels of IL-1β, IL-6, and IL-8 after TREM1 knockdown, compared by qRT-PCR, C: KEGG enrichment analysis of the impact of TREM1 on related pathways, D: Changes in the ERK signaling pathway and IL-6 expression after TREM1 knockdown, detected by Western blot, E: Changes in IL-6 transcript levels after TREM1 overexpression and subsequent inhibition of the ERK signaling pathway.

3.5 TREM1 Promotes Invasion and Metastasis of Gastric Cancer by upregulating IL-6

Existing research has shown that IL-6 can promote EMT and metastasis of gastric cancer, while TREM1 can positively regulate the expression level of IL-6. Based on this, it is speculated that TREM1 may primarily promote the invasion and metastasis of gastric cancer by upregulating the expression of IL-6. To verify this hypothesis, the expression of IL-6 was silenced while TREM1 was overexpressed, and the migration ability of MKN45 cells was observed. The results showed that after TREM1 overexpression, the migration ability of MKN45 was enhanced, and the enhancement effect of TREM1 on MKN45 migration ability could be partially reversed by silencing the expression of IL-6 (Figure 4A-B).

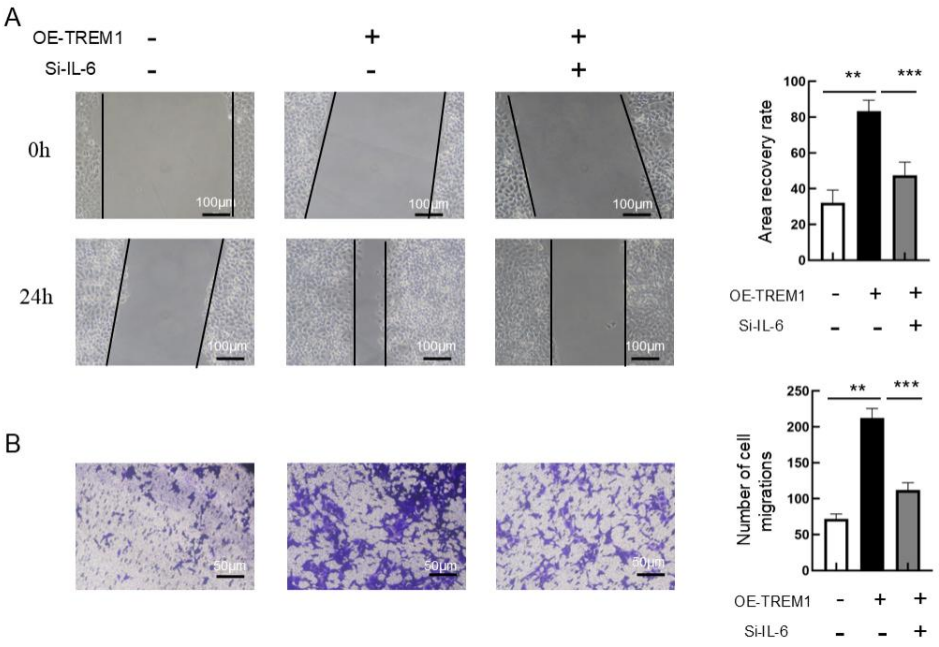

Figure 4 Changes in the Migration Ability of MKN45 Cells after Overexpressing TREM1 and Interfering with IL-6 (A: Migration ability of MKN45 cells after TREM1 overexpression and IL-6 knockdown, compared by wound healing assay. B: Migration ability of MKN45 cells after TREM1 overexpression and IL-6 knockdown, compared by Transwell

assay)

3.6 TREM1 Influences MKN45 Cell Metastasis by Regulating IL-6 to Affect Epithelial-Mesenchymal Transition (EMT) in Gastric Cancer

Key steps in gastric cancer metastasis are immune escape and EMT. Bioinformatics analysis was used to predict whether TREM1 is involved in these processes in gastric cancer. Results indicated that TREM1 can suppress the activation of B cells and CD4+ T cells, creating an inhibitory immune microenvironment in gastric cancer (Figure 5A). Furthermore, TREM1 promotes immune evasion by increasing the expression of immune checkpoint-related proteins such as CD274 (PD-L1) and CTLA4 (Figure 5B). TREM1 also showed a significant positive correlation with marker genes associated with EMT (Figure 5C). TREM1 overexpression led to downregulation of E-cadherin and upregulation of Vimentin, which are hallmark protein changes in EMT. These changes were partially reversed when IL-6 expression was simultaneously knocked down (Figure 5D).

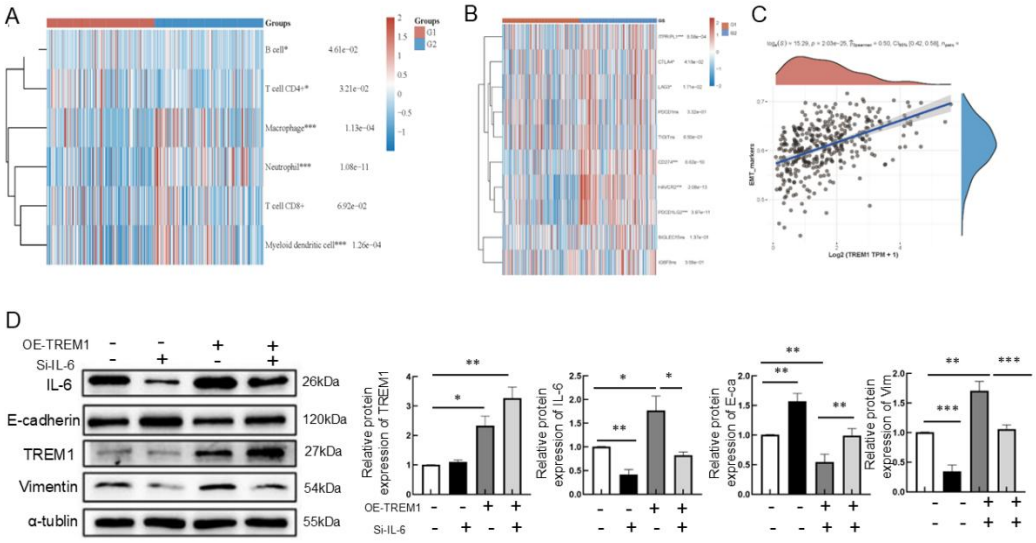

Figure 5 Correlation between TREM1, Immune Microenvironment, and EMT in Gastric Cancer

A: Bioinformatics analysis of immune cell infiltration in the high-TREM1 expression subgroup, B: Bioinformatics analysis of immune checkpoint-related protein expression in the high-TREM1 expression subgroup, C: Bioinformatics analysis of the correlation between TREM1 and EMT marker genes, D: Changes in EMT-related proteins after TREM1 overexpression and concurrent IL-6 knockdown, detected by Western blot. G1: Low TREM1 expression group; G2: High TREM1 expression group.

4 DISCUSSION AND CONCLUSION

TREM1, a secreted immune factor primarily produced by immune cells though also synthesized by tumor cells, is garnering increasing attention for its distinct role in malignancies. However, its expression profile and mechanistic function in gastric cancer remain unclear. This study first identified that TREM1 is highly expressed in gastric cancer tissues and associated with patient long-term survival. Subsequent analysis further revealed a close correlation between TREM1 and distant metastasis as well as recurrence in gastric cancer. This finding suggests the potential of TREM1 as an independent biomarker for predicting metastasis and recurrence in gastric cancer patients.

Recurrence and metastasis play crucial roles in determining patient prognosis and quality of life[20]. Factors contributing to gastric cancer recurrence and metastasis include the formation of an inhibitory tumor immune microenvironment, loss of immune surveillance, and epithelial-mesenchymal transition (EMT) [21-22]. MT refers to the process where tumor cells of epithelial origin lose polarity and acquire mesenchymal cell characteristics, retaining the migratory capacity of mesenchymal cells to promote tumor metastasis and drug resistance [23]. During EMT, characteristic molecular markers undergo changes, such as the downregulation of the epithelial marker E-cadherin and the upregulation of mesenchymal markers like N-cadherin, Vimentin, Snail, and fibronectin[24]. The EMT process is regulated by various factors, including transcription factors, the tumor immune microenvironment, and cytokines [25]. This study provides initial evidence that TREM1 promotes the invasion and metastasis of gastric cancer cells and is closely associated with the formation of an inhibitory immune microenvironment and immune evasion in gastric cancer. TREM1 can suppress effector T cell activity and enhance the expression of immune checkpoint-related proteins, thereby enabling gastric cancer cells to evade immune surveillance, proliferate, and metastasize. Furthermore, TREM1 expression showed a significant positive correlation with EMT marker genes (Pearson correlation coefficient = 0.50).

IL-6 is involved in EMT in gastric cancer. While TREM1's role as an upstream regulator of IL-6 has been established, its function in gastric cancer remains undefined. This study found that TREM1 similarly upregulates IL-6 in gastric cancer, primarily by modulating IL-6 expression through influencing the stability of the ERK signaling pathway. Based on this, we hypothesized that TREM1 might drive EMT in gastric cancer via elevated IL-6. Subsequent in vitro experiments confirmed that silencing IL-6 partially reversed the TREM1-induced EMT phenotypic changes and inhibited gastric cancer metastasis.

In summary, this study demonstrates for the first time that TREM1 is highly expressed in the serum of gastric cancer patients and can serve as an independent factor for monitoring distant metastasis and recurrence, as well as a prognostic marker. By activating the ERK signaling pathway, TREM1 increases IL-6 levels, thereby triggering EMT in gastric cancer cells to promote metastasis. Consequently, TREM1 represents a promising novel therapeutic target for gastric cancer.

COMPETING INTERESTS

The authors have no relevant financial or non-financial interests to disclose.

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AUTHORS' CONTRIBUTION

Z.Z. and X.T. conceived and planned the study. Y.F., J.G., W.S., designed the experiments. Z.Z. conducted the experiments and scrutinize the data. X.T. wrote, edited and revised the manuscript. The final manuscript has been read and approved by all the authors.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The retrospective study was reviewed and approved by the Ethics Committee of the Affiliated Hospital of Jiangsu University (approval number: KY201901).

CONSENT FOR PUBLICATION

All authors have given consent for publication.

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