Electronic Journal of Biotechnology 81 (2026) 100713

Contents lists available at ScienceDirect

Electronic Journal of Biotechnology

journal homepage: www.elsevier.com/locate/ejbt

Research article

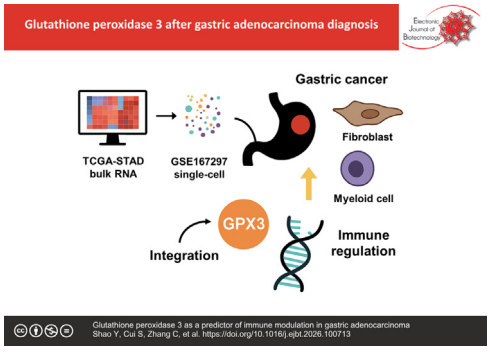
Glutathione peroxidase 3 as a predictor of immune modulation in gastric adenocarcinoma ☆

Ying Shao [a], Shanpeng Cui [b], Chunfeng Zhang [c], Li Li [d], Lijuan Ma [e,*]

[a] Department of Vascular Interventional Surgery, The Fourth Affiliated Hospital of Harbin Medical University, Harbin, China
[b] Department of Critical Care Medicine, The Second Affiliated Hospital of Harbin Medical University, Harbin, China
[c] Department of Endocrinology, The First Affiliated Hospital of Harbin Medical University, Harbin, China
[d] Department of Nephrology, The First Affiliated Hospital of Harbin Medical University, Harbin, China
[e] Nursing Department, The Fourth Affiliated Hospital of Harbin Medical University, Harbin, China

GRAPHICAL ABSTRACT

Glutathione peroxidase 3 as a predictor of immune modulation in gastric adenocarcinoma.



ARTICLE INFO

Article history:
Received 23 September 2025
Accepted 8 January 2026
Available online 16 April 2026

Keywords:
Fibroblasts
Gastric adenocarcinoma
Glutathione peroxidase
Immune cell infiltration
Immune checkpoint inhibitors
Immune modulation
Myeloid cells

ABSTRACT

Background: Gastric cancer is a highly prevalent and lethal malignancy worldwide, with its immune microenvironment playing a crucial role in tumor initiation and progression. Among selenoproteins, glutathione peroxidase 3 (GPX3) is an important antioxidant enzyme that has recently attracted attention for its roles in various cancers. However, the function of GPX3 and its impact on the immune microenvironment in stomach adenocarcinoma (STAD) remain insufficiently explored.
Results: Deep STAD tissues were more enriched in immune cells compared to superficial tumor tissues, particularly myeloid cells and fibroblasts. GPX3 was predominantly expressed in fibroblasts and myeloid cells, while its expression in T cells was relatively low, with no significant differences across different tumor layers. Moreover, GPX3 exhibited weak correlations with PD-1 and CTLA-4, suggesting that GPX3 may not directly mediate immune evasion via immune checkpoint pathways. These findings characterize the cellular distribution of GPX3 within the STAD immune microenvironment and provide initial insights into its potential regulatory function.

☆ Audio abstract available in Supplementary material.

Peer review under responsibility of Pontificia Universidad Católica de Valparaíso.

* Corresponding author.
E-mail address: ljuanjljljlmamm@finmail.com (L. Ma).

https://doi.org/10.1016/j.ejbt.2026.100713

Predictor
Selenoproteins
Stomach neoplasms
Tumor microenvironment

Conclusions: Although GPX3 may not directly influence immune checkpoint pathways, its high expression in myeloid cells and fibroblasts suggests that it might indirectly modulate immune responses by regulating the tumor microenvironment. These results lay a theoretical foundation for future research on GPX3 in gastric cancer and its potential as a therapeutic target.

How to cite: Shao Y, Cui S, Zhang C, et al. Glutathione peroxidase 3 as a predictor of immune modulation in gastric adenocarcinoma. Electron J Biotechnol 2026;81. https://doi.org/10.1016/j.ejbt.2026.100713.

1. Introduction

Gastric cancer (GC) ranks as the fifth most common malignancy worldwide and the fourth leading cause of cancer-related mortality [1]. The overall survival rate is 31% in the United States and 25% globally [2]. Its etiology is associated with *Helicobacter pylori* infection, unhealthy lifestyle habits, and other risk factors, with a five-year survival rate of less than 50% in most advanced cases [3]. Although extensive research has been conducted on gastric cancer, more effective molecularly driven therapeutic strategies are still needed. As an emerging therapeutic approach, immunotherapy has demonstrated significant potential in treating various malignancies in recent years [4]. However, its application in gastric cancer faces several challenges. Gastric cancer patients exhibit substantial heterogeneity in response and resistance to immunotherapy, leading to considerable variation in individual treatment outcomes [5]. Therefore, a comprehensive understanding of the immune molecular characteristics and immune evasion mechanisms in gastric cancer is essential for developing more personalized and effective treatment strategies.

Trace elements play crucial biological roles in living organisms, but their low concentrations in the human body often result in them being overlooked. These essential elements are primarily acquired through diet, inhalation, and skin contact. Recent research has shown that Se, Cu, and Zn contribute to the development of gastrointestinal tumors. Studies indicate that selenium serves as the active center of antioxidant enzymes, enabling lipid peroxidation inhibition and free radical scavenging, which in turn influences cancer risk [6]. The immune-regulating effects of selenium are mainly attributed to the diverse biological activities of selenoproteins, particularly their involvement in redox balance. To date, 25 selenoprotein-coding genes have been identified in humans, including glutathione peroxidase 3 (GPX3), which plays a key role in antioxidant defense mechanisms [7]. GPX3, as a secreted enzyme, can influence the extracellular redox environment and modulate immune cell functions [8]. Abnormal GPX3 expression has been found to be associated with the onset and progression of various cancers, including melanoma [9], esophageal cancer [10], ovarian cancer [11], colorectal cancer [12], gastric cancer [13], and other malignancies.

Furthermore, selenium supplementation has been shown to influence both innate and adaptive immune cells within the tumor microenvironment (TME). It affects innate immune cells, including neutrophils, macrophages, and natural killer (NK) cells, as well as adaptive immune cells, particularly T lymphocytes. As a critical trace element, selenium is essential for regulating immune function, primarily by enhancing the antioxidant capacity of immune cells and modulating immune responses [14]. In the TME, Se may regulate macrophage polarization, modulate T cell activity and function, and influence the response of other immune cells, thereby shaping the immune microenvironment [15,16]. These findings suggest that selenium may mediate immune evasion in gastric cancer by altering tumor-infiltrating immune cell populations, though its precise mechanisms remain to be further explored.

This study aims to investigate the regulatory role of GPX3 in the immune microenvironment of gastric cancer and its potential mechanisms, with the goal of elucidating its impact on immune evasion in gastric cancer and providing new theoretical insights for personalized immunotherapy strategies.

2. Materials and methods

Fig. 1 illustrates the overall workflow of this study, integrating data from GEO, TCGA, and TIMER databases.

2.1. Single-cell sequencing data acquisition and analysis

The GSE167297 dataset was obtained from the GEO database, containing 10X single-cell sequencing data from five gastric cancer patients, including three groups (normal tissue, deep cancerous tissue, and superficial cancerous tissue). The Seurat package was used to import the 10X data through the Create Seurat Object function, constructing a Seurat object. The subset function was applied to select cell datasets with UMI counts greater than 500 and genes expressed in at least three cells, while the Which Cells function was used to filter out cells that did not meet these criteria. The Run Harmony function from the harmony package was used to integrate all samples, reducing batch effects among samples. The Run UMAP function (UMAP dimensionality reduction method) and the Run TSNE function (t-SNE dimensionality reduction method) were used for dataset dimensionality reduction, and the Dim Plot function was used for visualization after reduction. The Find Clusters function was used to determine the appropriate number of clusters by setting the resolution parameter. Based on single-cell gastric cancer data, all clusters were manually annotated according to marker genes reported in the literature. The Find Markers function was used to compare differentially expressed genes between normal tissue and superficial cancerous tissue, as well as between normal tissue and deep cancerous tissue, and the VlnPlot function was used for visualization.

2.2. TCGA gastric adenocarcinoma data acquisition and analysis

Gastric cancer-related data were retrieved from the TCGA-STAD dataset. Our methodological approach, utilizing well-established public repositories like TCGA, is consistent with prior successful investigations in cancer genomics [17,18]. However, we also recognize the inherent technical and biological biases associated with bulk transcriptomic data mining [19,20], and thus, our findings based on these datasets should be interpreted with this context in mind. Based on clinical information, samples were categorized into normal and cancer groups. RNA expression data were grouped accordingly, and the CIBERSORT function from the CIBERSORT package was used to analyse the composition and proportions of immune cell subtypes in different groups. The results were visualized using the ggpubr and ggplot2 packages.

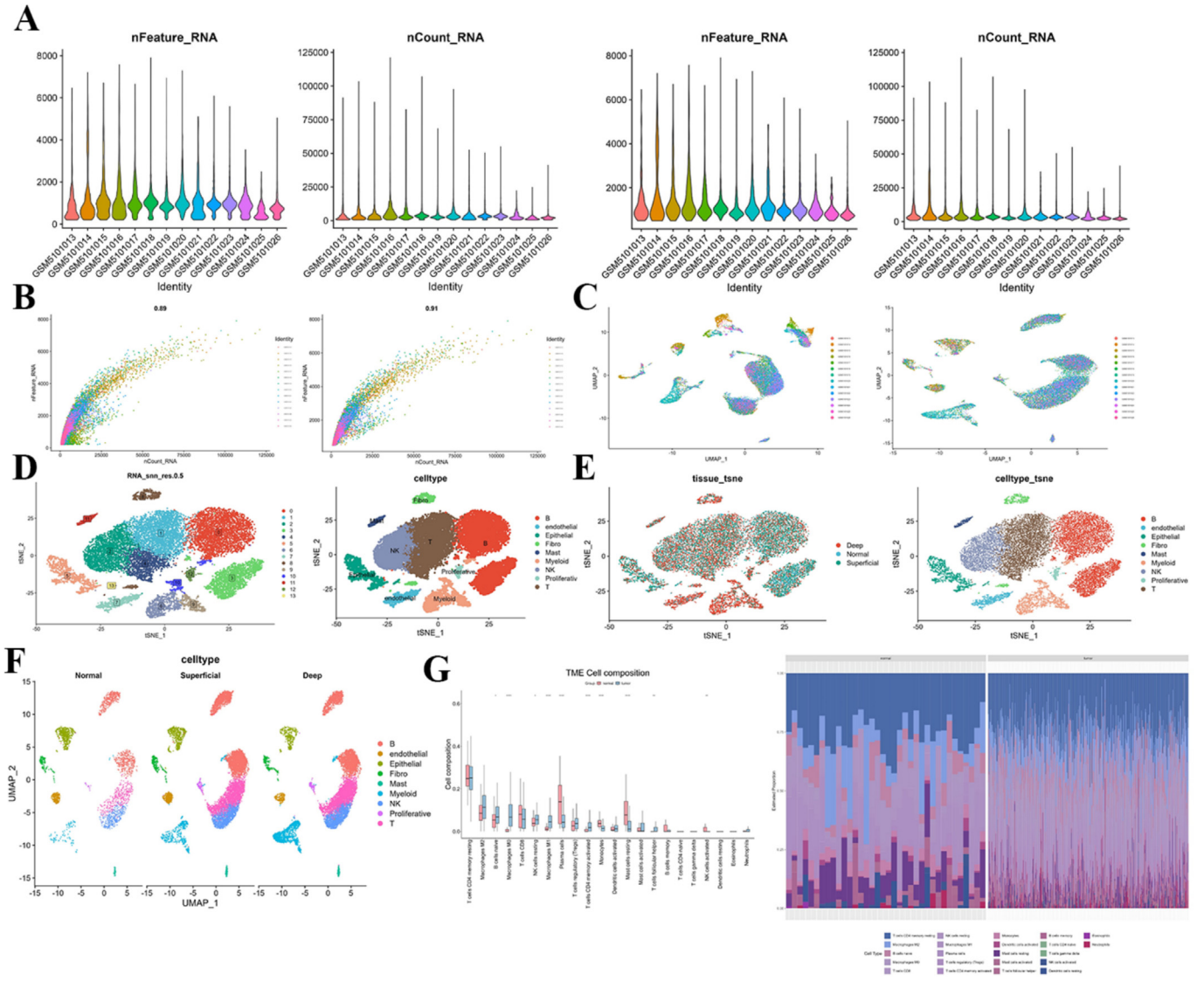


Fig. 1. Distribution of cells in superficial and deep gastric adenocarcinoma tissues. (A) Quality control of single-cell data: The subset function selected cells with UMI > 500, gene expression in ≥3 cells, mitochondrial ratio < 25%, ribosomal ratio > 3%, and hemoglobin ratio < 1%. The Which Cells function filtered out non-conforming cells. Left: UMI and gene counts before filtering; Right: after filtering. (B) Correlation between UMI and gene counts before (left) and after (right) filtering, showing improved data quality. (C) UMAP clustering before and after sample integration: Left: before integration; Right: after integration, showing reduced batch effects. (D) t-SNE clustering and cell type annotation: Left: t-SNE clustering identifying 14 clusters; Right: annotated clusters based on marker genes. (E) Comparison of cell distribution across normal, superficial cancer, and deep cancer tissues: Left: distribution of different tissue types; Right: annotated cell clusters. (F) UMAP visualization of cell distribution: Left: normal tissue; Middle: superficial cancer tissue; Right: deep cancer tissue, showing increased immune cells and decreased epithelial/endothelial cells in deeper cancer tissues. (G) Tumor microenvironment (TME) immune cell composition: Left: immune cell differences between normal and cancer tissues (X-axis: immune cell types, Y-axis: proportion). Right: immune cell composition in individual samples (each bar represents a sample; different colors indicate different immune cell types). Left: normal samples; Right: cancer samples.

2.3. TIMER data acquisition and analysis

Data on the correlation coefficients and *p*-values of GPX3, PDCD1, and CTL4 genes in gastric adenocarcinoma were retrieved from the TIMER database.

3. Results

3.1. Distribution of cells in superficial and deep gastric adenocarcinoma tissues

To determine the cellular composition of superficial and deep gastric adenocarcinoma tissues, we obtained the GSE167297 dataset from the GEO database, which includes single-cell 10X data from normal tissue, deep cancerous tissue, and superficial cancerous tissue. Data quality control was performed using the subset and Which Cells functions, and sample integration was conducted with the Run Harmony function to reduce batch effects among samples. As shown in Fig. 1A–C, the filtered data exhibited improved structure, stronger correlation between unique molecular identifiers (UMIs) and gene expression, and reduced heterogeneity in the integrated data, meeting the requirements for subsequent analyses. Using t-distributed stochastic neighbour embedding (t-SNE), we categorized the cells into 14 clusters, which were further classified into nine distinct cell types (Fig. 1D). The analysis revealed that cells from normal tissue were distributed across all clusters, whereas B cells were more abundant in superficial cancerous tissue, and myeloid cells and fibroblasts were predominantly found in deep cancerous tissue, suggesting potential differences in cell subpopulations at different tumor depths

(Fig. 1E). Moreover, as the tumor tissue extended deeper, the number of immune cells increased, while the proportion of epithelial or endothelial cells decreased (Fig. 1F). Additionally, data retrieved from the TCGA database further confirmed that the composition of the tumor microenvironment in gastric adenocarcinoma underwent significant alterations, particularly in macrophages, monocytes, T cells, and mast cells (Fig. 1G). These findings indicate that, compared to normal tissue, the cellular distribution in both superficial and deep gastric adenocarcinoma tissues exhibited significant changes, especially in immune cell subpopulations dominated by macrophages and T cells.

3.2. Low expression of GPX3 in T cells in gastric cancer

Next, we analyzed the expression of selenoprotein GPX3 in gastric adenocarcinoma tissues. As shown in Fig. 2A, GPX3 is highly expressed in fibroblasts and myeloid cells, with detectable expression in epithelial cells as well. Notably, GPX3 expression is relatively low in T cells (Fig. 2A). Furthermore, an analysis of GPX3 expression levels in T cells from normal tissue, superficial cancer tissue, and deep cancer tissue revealed no statistically significant differences among these groups (Fig. 2B).

3.3. GPX3 in gastric cancer does not directly regulate immune checkpoints

To investigate whether GPX3 is involved in immune checkpoint–mediated tumor immune evasion, we examined its correlations with the immune inhibitory receptors PD-1 and CTLA-4 in stomach adenocarcinoma. As shown in Fig. 3, GPX3 expression exhibited only a weak positive correlation with PD-1 (r = 0.203) and an even weaker association with CTLA-4 (r = 0.161). These results suggest that GPX3, although functionally related to oxidative stress regulation, may not directly participate in immune suppression via checkpoint pathways. Instead, it is more likely to modulate immune activity indirectly through redox-dependent signaling or stromal remodeling within the tumor microenvironment.

4. Discussion

This study, through the analysis of GEO and TCGA databases, reveals the potential role of the selenoprotein GPX3 in the immune microenvironment of stomach adenocarcinoma (STAD). The results indicate that deep STAD tissues exhibit more extensive immune cell infiltration compared to superficial tumor tissues, characterized by an increase in myeloid cells and fibroblasts, whereas B cells are more predominant in superficial cancer tissues. Additionally, GPX3 is highly expressed in fibroblasts and myeloid cells but shows low expression in T cells, with no significant difference in GPX3 expression levels among T cells in different tumor layers. These findings suggest that GPX3 primarily exerts its effects in the gastric cancer microenvironment through myeloid cells and fibroblasts rather than directly modulating T cell function.

Furthermore, the well-established role of *Helicobacter pylori* infection in gastric carcinogenesis provides a compelling context for our findings. *H. pylori* is known to induce oxidative stress and shape the tumor immune microenvironment. Recent evidence sug-

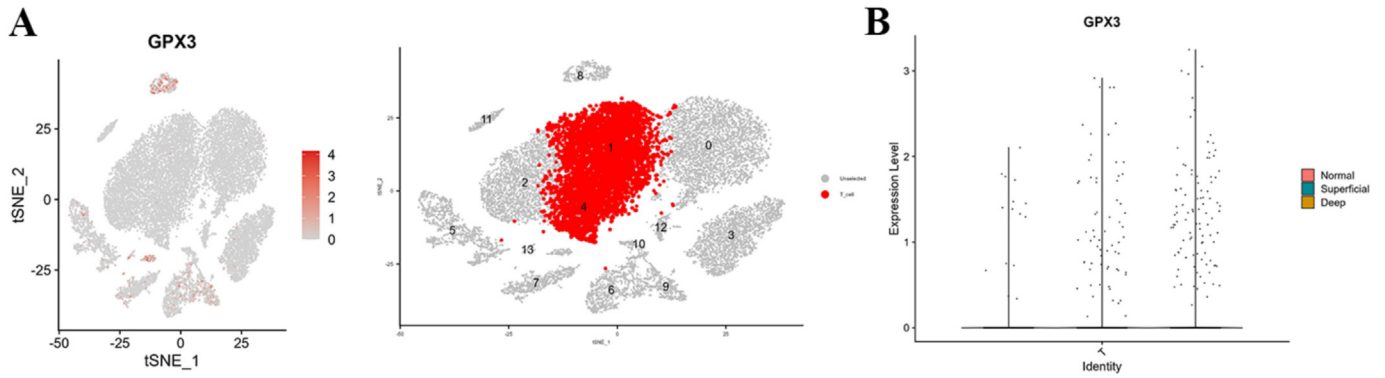


Fig. 2. Cellular origin of GPX3 in gastric cancer tissues. (A) t-SNE plot showing GPX3 expression distribution across different cell populations (left). The right panel highlights GPX3 expression specifically in T cells. (B) GPX3 expression levels in T cells across different tissue groups (Normal, Superficial, Deep) show no significant differences among them.

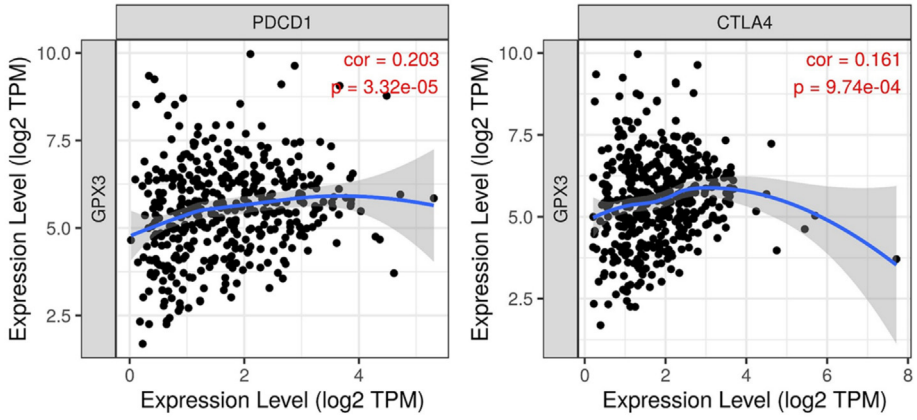


Fig. 3. Correlation analysis between GPX3 and PD-1/CTLA-4. This figure illustrates the correlation between GPX3 and PD-1 (PDCD1 gene) as well as CTLA-4 (CTL4 gene) in gastric cancer. The left panel presents the scatter plot of GPX3 and PD-1 correlation, while the right panel shows the scatter plot of GPX3 and CTLA-4 correlation.

gests that *H. pylori* infection can directly influence the phenotypes of macrophages and fibroblasts within the gastric tumor niche [21]. Given that our study identified fibroblasts and myeloid cells (including macrophages) as the primary sites of GPX3 expression, it is plausible that *H. pylori*-induced oxidative stress may regulate or be counterbalanced by GPX3 in these specific cell compartments. This potential interplay between a major bacterial pathogen, redox regulation by GPX3, and immune cell modulation warrants further investigation.

To fully understand GPX3′s role, future studies should also consider its position within broader transcriptional networks. For instance, circuits like the BRD9-p53-E2F1 axis, which integrates redox regulation with cell-cycle control and DNA damage response in gastric cancer [22], might operate in parallel or upstream of GPX3-mediated antioxidant pathways. Similarly, the expanding field of non-coding RNAs offers another layer of regulation. As highlighted in recent reviews, long non-coding RNAs (lncRNAs) are key transcriptional tuners of numerous redox and immune regulators [23]. It is conceivable that GPX3 expression itself is modulated by specific lncRNAs in STAD, a promising area for future exploration that could reveal new regulatory mechanisms and therapeutic targets.

The study also analyzed the correlation between GPX3 and immune checkpoint genes (PD-1 and CTLA-4), revealing only a weak positive correlation. This suggests that GPX3 may not directly promote immune evasion in gastric cancer by enhancing immune suppression but rather influences the tumor immune microenvironment through alternative mechanisms, such as regulating redox balance, modulating tumor-associated fibroblasts, or affecting myeloid cell function.

It is also important to acknowledge the limitations of our study. The single-cell RNA sequencing analysis, while informative, was based on a cohort of five patients. This limited sample size may affect the generalizability of our findings, and future validation with larger scRNA-seq cohorts is recommended.

Existing studies have demonstrated that selenoprotein GPX3 plays a crucial role in various malignancies, including melanoma, esophageal cancer, ovarian cancer, and colorectal cancer [9,10,11,12,13]. In gastric cancer, GPX3 may influence tumor-associated cell functions by regulating oxidative stress within the tumor microenvironment. However, its specific mechanisms remain unclear and require further investigation. Potential areas of exploration include whether GPX3 affects myeloid cell polarization, modulates tumor-associated fibroblast activity, or regulates inflammatory cytokines to influence immune cell infiltration. To experimentally validate and extend our computational findings, several approaches are proposed for future work. Firstly, immunohistochemistry (IHC) or multiplex immunofluorescence (mIF) on gastric cancer tissue sections could directly visualize the co-localization of GPX3 protein with canonical markers for macrophages (e.g., CD68) and fibroblasts (e.g., FAP). Secondly, analyzing correlations between GPX3 and these cellular markers in additional independent bulk or single-cell transcriptomic datasets would strengthen the robustness of our observations. Such validation steps are crucial for confirming the cellular sources of GPX3 within the TME. Finally, given the intimate link between redox balance and cellular metabolism, it is tempting to speculate that GPX3′s antioxidant activity might influence immunometabolic reprogramming within the gastric TME. By scavenging peroxides, GPX3 could potentially alter metabolic pathways in tumor or stromal cells, thereby indirectly shaping the metabolic crosstalk that defines immune cell function and infiltration patterns [24]. Exploring this potential nexus between GPX3, oxidative stress, and tumor immunometabolism represents a fascinating future research avenue.

In summary, this study highlights the expression characteristics of GPX3 within the immune microenvironment of gastric cancer and preliminarily explores its potential role in tumor immune evasion. Although GPX3 shows a weak direct correlation with immune checkpoint pathways, its high expression in myeloid cells and fibroblasts suggests that it may indirectly influence immune responses by modulating the tumor microenvironment. These findings provide new theoretical insights for personalized gastric cancer therapy and establish a foundation for further research on GPX3 as a potential therapeutic target.

5. Conclusions

This study revealed significant differences in the immune microenvironment of gastric adenocarcinoma at different tissue depths, with deep cancerous tissues being enriched in immune cells. GPX3 was predominantly expressed in fibroblasts and myeloid cells, with low expression in T cells, and showed no significant variation in T cell expression across different tissue types. Furthermore, GPX3 exhibited only weak correlations with immune checkpoints PD-1 and CTLA-4, suggesting that it may not directly contribute to immune evasion. Future research should further explore the mechanistic role of GPX3 in gastric adenocarcinoma to identify potential therapeutic targets.

CRediT authorship contribution statement

Ying Shao: Writing – original draft. **Shanpeng Cui:** Writing – original draft. **Chunfeng Zhang:** Writing – original draft. **Li Li:** Writing – original draft. **Lijuan Ma:** Writing – original draft.

Ethical approval and consent to participate

This study has been approved by the Fourth Affiliated Hospital of Harbin Medical University. All the experiments of this study were conducted in accordance with the relevant guidelines and regulations or in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants.

Financial support

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Declaration of competing interest

The authors declare no competing interests.

Supplementary material

https://doi.org/10.1016/j.ejbt.2026.100713.

Data availability

All data generated or analyzed during this study are included in this published article.

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