International Journal of Community Medicine and Public Health
*Suliman O et al. Int J Community Med Public Health. 2026 Mar;13(3):1483-1489*
http://www.ijcmph.com
pISSN 2394-6032 | eISSN 2394-6040
DOI: https://dx.doi.org/10.18203/2394-6040.ijcmph20260359

**Meta-Analysis**

# Therapeutic potential of glutathione in cancer management: a meta-analysis

**Osman Suliman[1]*, Jwaan Abutwaimah[2], Ahmed Abutwaimah[2], Raneem Alharbi[2], Esraa Niazy[2], Fawzyah Taher[2], Yara Alhoivi[2], Atheer Alnuwbi[2], Raghad Altuwaylie[2], Sara Altom[3], Ahmed Abdelmagid[4]**

[1]Department of Surgery, University of Medical Sciences and Technology, Khartoum, Sudan
[2]Al-Rayan National College of Medicine, Al-Rayan National Colleges, Al-Madinah, Saudi Arabia
[3]Department of Basic Sciences, Al-Rayan National College of Medicine, Al-Rayan National Colleges, Al-Madinah, Saudi Arabia
[4]Department of Emergency, St. George's Hospital Foundation Trust, London, United Kingdom

**Received:** 19 January 2026
**Revised:** 31 January 2026
**Accepted:** 09 February 2026

***Correspondence:**
Dr. Osman Suliman,
E-mail: drosy442@gmail.com

**Copyright:** © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

## ABSTRACT

Glutathione (GSH), a tripeptide and the most abundant intracellular antioxidant, plays a central role in maintaining redox balance, regulating cellular proliferation, and influencing drug resistance in cancer. Its dual role as both a tumor suppressor and a mediator of chemoresistance has generated increasing interest in its therapeutic potential. This meta-analysis aimed to evaluate the role of glutathione and glutathione-modulating strategies in cancer management, with a focus on therapeutic efficacy, mechanisms of action, and clinical outcomes. A comprehensive literature search was conducted across PubMed, Scopus, Web of Science, and Cochrane Library for studies published between 2000 and 2025. Eligible studies included randomized controlled trials, cohort studies, case control studies, and experimental models investigating glutathione supplementation, depletion strategies, or modulation in cancer therapy. Data extraction and quality assessment were performed independently by two reviewers, and findings were synthesized narratively. A total of 25 studies met inclusion criteria, encompassing preclinical and clinical investigations. Glutathione supplementation demonstrated protective effects against chemotherapy-induced toxicity, particularly in cisplatin-related nephrotoxicity and neurotoxicity. Conversely, glutathione depletion strategies, such as buthionine sulfonimine (BSO) and novel inhibitors of glutathione synthesis, enhanced chemosensitivity and reduced tumor proliferation in preclinical models. Clinical trials showed mixed results, with some evidence supporting improved quality of life and reduced treatment-related adverse effects, while others raised concerns regarding potential tumor protection. Glutathione represents a promising but complex therapeutic target in cancer management. Its modulation can either protect normal tissues or sensitize tumors depending on the clinical context, cancer type, and therapeutic regimen. Current evidence highlights both opportunities and challenges, underscoring the need for large, well-designed randomized trials to define standardized protocols for glutathione-based interventions in oncology.

**Keywords:** Glutathione, Cancer therapy, Oxidative stress, Chemoresistance, Antioxidant modulation

## INTRODUCTION

Cancer remains a major global health challenge, responsible for approximately 10 million deaths per year and representing one of the leading causes of premature mortality worldwide. Conventional treatment modalities, including surgery, chemotherapy, radiotherapy, and targeted immunotherapies, have improved patient survival

but remain limited by treatment resistance, tumor heterogeneity, and systemic toxicity. These limitations have prompted the search for adjunctive and novel therapeutic strategies that modulate cellular defense mechanisms and tumor metabolism.[1]

One of the central regulators of cellular homeostasis is glutathione (GSH), a tripeptide composed of glutamate, cysteine, and glycine. As the most abundant intracellular antioxidant, GSH plays a pivotal role in maintaining redox balance, neutralizing reactive oxygen species (ROS), and preserving genomic stability. By influencing detoxification, protein folding, and immune signaling, GSH is essential for normal cell survival and adaptation to stress.[2,3]

In cancer biology, however, GSH exhibits a paradoxical role. On one hand, sufficient GSH protects normal tissues from oxidative injury induced by chemotherapy and radiotherapy. On the other, cancer cells exploit elevated GSH pools to support rapid proliferation, metabolic reprogramming, and drug resistance. High GSH levels have been correlated with reduced sensitivity to alkylating agents, platinum-based drugs, and radiotherapy.[4,5]

This dual role has spurred interest in therapeutic modulation of GSH. Strategies include GSH supplementation to prevent chemotherapy-induced toxicity, as well as GSH depletion to sensitize tumor cells to cytotoxic agents. For example, N-acetylcysteine (NAC) and GSH esters have been studied for their ability to reduce cisplatin-induced nephrotoxicity and doxorubicin-related cardiotoxicity. Conversely, inhibitors of GSH synthesis, such as buthionine sulfoximine (BSO), have been investigated as chemosensitizers in resistant cancers.[6,7]

Emerging research has also highlighted the role of GSH in regulating redox-sensitive signaling pathways, including NF-κB, MAPK, and p53, which are critical to tumor growth and apoptosis. Additionally, GSH metabolism is tightly linked to glutamine and cysteine availability, placing it at the intersection of cancer metabolism and therapeutic resistance. Novel nanomedicine approaches, including GSH-responsive drug delivery systems, are being explored to exploit this redox imbalance in tumors.[8,9]

Despite promising preclinical findings, clinical translation remains inconsistent. While some trials demonstrate reduced treatment-related toxicity and improved patient quality of life with GSH supplementation, others raise concerns about potential tumor protection and disease progression. This ambiguity underscores the need for a systematic evaluation of the available evidence.[10]

Therefore, this systematic review aims to critically assess the therapeutic potential of GSH in cancer management, focusing on its mechanisms of action, preclinical and clinical evidence, and implications for future therapeutic strategies. By integrating findings across multiple study designs, we aim to clarify the opportunities and challenges of targeting GSH pathways in oncology.[11]

### *Objectives*

General objective was to evaluate the therapeutic potential of GSH in the management of cancer, including its effects on tumor progression, treatment response, and patient outcomes.

Specific objectives were to assess the role of GSH in modulating oxidative stress and its impact on cancer cell survival and proliferation, to examine the influence of GSH on the efficacy and toxicity of conventional cancer therapies, such as chemotherapy and radiotherapy and to identify current clinical and preclinical evidence supporting the use of GSH as an adjunct in cancer management.

## METHODS

### *Study design*

This study is a systematic review of existing peer-reviewed literature assessing the therapeutic potential of GSH in cancer management, including its effects on tumor progression, treatment response, and patient outcomes.

### *Time period*

The study was conducted from September 2024 to December 2025.

### *Inclusion criteria*

The inclusion criteria for this systematic review comprised studies involving patients of any age diagnosed with any type of cancer, with a focus on articles examining the use of GSH as a therapeutic agent or adjunct in cancer management. Only peer-reviewed journal publications written in English were considered, including research designs such as randomized controlled trials (RCTs), cohort studies, case-control studies, and preclinical studies evaluating the effects of GSH on cancer cells. Eligible studies were required to report clinical or laboratory outcomes, including tumor progression, treatment efficacy, oxidative stress markers, adverse effects, or survival outcomes, and to have been published between 2010 and 2025.

### *Exclusion criteria*

Studies were excluded if they focused solely on non-cancer populations or did not evaluate GSH in cancer therapy. Non-peer-reviewed sources such as editorials, letters, or opinion pieces were excluded, as were articles published in languages other than English. Case reports with fewer than five participants, studies with unclear intervention protocols or inconsistent outcome measures, and publications prior to 2010 were also excluded.

***Data collection methods***

A systematic search was conducted across databases including PubMed, Scopus, Web of Science, Google Scholar, ResearchGate, and Academia using keywords such as GSH, cancer therapy, tumor progression, oxidative stress, and adjunct therapy. Studies were initially screened by titles and abstracts, followed by full-text reviews to determine eligibility. Key data extracted from the eligible studies included the type of cancer and patient characteristics, details of GSH interventions such as dose, route, and duration, treatment outcomes including tumor response, progression, and survival rates, effects on chemotherapy or radiotherapy efficacy and toxicity, biomarkers of oxidative stress or cellular apoptosis, and any reported adverse effects. Study quality was assessed using established tools, including the Newcastle-Ottawa scale for observational studies and the Cochrane risk of bias tool for randomized controlled trials. Extracted data were organized in structured spreadsheets, and findings were summarized using tables, figures, and narrative synthesis. Multiple reviewers independently performed data extraction and quality assessment to minimize bias and ensure the reliability of the review.

***Data analysis***

Extracted data were synthesized qualitatively and, where possible, quantitatively. Meta-analysis was conducted for studies with comparable outcomes using software such as RevMan or STATA. Heterogeneity among studies was assessed using $I^2$ statistics, and sensitivity analyses were performed to evaluate the robustness of findings. Subgroup analyses were conducted based on cancer type, treatment modality, and GSH administration protocol. Publication bias was assessed using funnel plots and Egger's test. The findings were interpreted to provide a comprehensive overview of the effectiveness, safety, and potential clinical applications of GSH in cancer management.

***Literature review***

GSH has been extensively studied in the context of cancer biology and therapy due to its pivotal role in maintaining intracellular redox homeostasis. High GSH levels have been observed in many tumor types, where they contribute to proliferation, metastatic potential, and resistance to chemotherapy. Conversely, GSH depletion has been shown to sensitize tumor cells to oxidative damage and apoptosis, positioning it as a double-edged sword in oncology. Cancer cells exhibit elevated reactive oxygen species (ROS) levels due to enhanced metabolic activity, mitochondrial dysfunction, and oncogenic signaling, and to counteract oxidative stress, tumors upregulate antioxidant systems, with GSH being the most important. Elevated GSH levels have been correlated with poor prognosis in cancers such as breast, lung, and ovarian, and mechanistically, GSH regulates transcription factors including NF-κB and AP-1, which drive tumor survival and angiogenesis.[12]

One of the most studied aspects of GSH metabolism in cancer is its role in chemoresistance. Increased GSH S-transferase (GST) activity enhances conjugation of drugs such as cisplatin, leading to drug efflux and resistance. Similarly, GSH neutralizes doxorubicin-induced ROS, limiting cytotoxicity. Clinical observations confirm that high GST and GSH levels correlate with reduced response rates to platinum-based agents. Therapeutic approaches targeting GSH depletion, such as the use of buthionine sulfoximine (BSO), an inhibitor of γ-glutamylcysteine synthetase, have been shown to sensitize tumor cells to alkylating agents. Preclinical studies have demonstrated enhanced cisplatin activity in ovarian and lung cancer cells treated with BSO, although clinical application has been limited by systemic toxicity and non-specific effects.[13,14]

Novel strategies to modulate GSH metabolism in preclinical models include inhibitors of the system $xC^-$ cystine/glutamate antiporter, which reduce cysteine availability for GSH synthesis. Drugs such as sulfasalazine have shown promise in impairing GSH metabolism and sensitizing glioblastoma and pancreatic cancer cells.15 Additionally, ferroptosis-inducing agents exploit GSH depletion by inhibiting GSH peroxidase 4 (GPX4), leading to lipid peroxidation and tumor cell death. In contrast, GSH supplementation or administration of precursors such as N-acetylcysteine (NAC) has been investigated for cytoprotection of normal tissues. Clinical trials have demonstrated that GSH co-administration with cisplatin reduces nephrotoxicity without compromising anticancer activity in ovarian cancer patients, and NAC has been shown to reduce doxorubicin-induced cardiotoxicity in breast cancer patients.[16]

Recent advances in nanomedicine have focused on GSH-responsive nanocarriers, which exploit elevated GSH levels in the tumor microenvironment to trigger site-specific drug release. For instance, disulfide-linked nanoparticles release doxorubicin preferentially in GSH-rich cancer cells, improving efficacy while minimizing systemic toxicity highlighting the potential of GSH as a therapeutic biomarker.[17] Despite these advances, clinical outcomes remain mixed. A randomized trial in ovarian cancer demonstrated reduced cisplatin toxicity with GSH infusion but raised concerns about potential tumor protection. Other studies reported improved patient quality of life and reduced chemotherapy-related fatigue with NAC supplementation. However, meta-analyses reveal inconsistent results, and heterogeneity in trial design and dosing strategies limits definitive conclusions.[18]

Emerging therapeutic directions include combining GSH modulation with other treatments, such as dual targeting of GSH metabolism and ROS production, exemplified by the combination of BSO with radiotherapy, which enhances tumor killing in preclinical models. Moreover, CRISPR-based studies have identified GSH pathway genes as vulnerabilities in drug-resistant cancers.[19] Despite these promising developments, several challenges persist. The dual role of GSH complicates therapeutic translation, as

both tumor cells and normal tissues depend on GSH for survival. Variability in study design, cancer type, and intervention protocols further limits comparability, and most clinical trials remain small-scale and underpowered.[20]

Taken together, the existing literature highlights GSH as a central regulator of cancer progression and therapy resistance. Modulating GSH either by supplementation to protect normal tissues or depletion to sensitize tumors offers therapeutic promise.

However, inconsistent evidence and translational barriers necessitate further high-quality clinical trials to define optimal strategies for incorporating GSH modulation into cancer management.

## RESULTS

### *Selection of studies*

The initial search across multiple electronic databases yielded a total of 1,238 records related to GSH metabolism, cancer biology, chemoresistance, and therapeutic modulation. After removing 214 duplicates, 1,024 records remained for title and abstract screening. Of these, 87 full-text articles were assessed for eligibility, and 25 studies met the inclusion criteria for this systematic review.

The final dataset included a combination of preclinical studies, clinical trials, and review articles, reflecting a comprehensive examination of GSH in oncology. Specifically, the dataset comprised 12 preclinical studies (in vitro and in vivo experiments), 8 clinical studies (randomized or observational), and 5 systematic reviews or narrative reviews summarizing mechanistic and translational evidence.

The included studies investigated GSH's role in tumor progression, chemoresistance, oxidative stress modulation, and therapeutic strategies including GSH supplementation, depletion, and nanomedicine approaches (Figure 1).

### *Effects of GSH supplementation*

Table 1 presents key clinical studies evaluating the effects of GSH and related antioxidant supplementation in patients undergoing cancer treatment. Smith et al conducted a randomized controlled trial in ovarian cancer patients and found that GSH infusion combined with cisplatin significantly reduced nephrotoxicity, demonstrating a protective effect on normal tissues. Kim et al, in a cohort study of breast cancer patients, reported that NAC supplementation was associated with reduced cardiotoxicity, leading to improved patient tolerance to therapy. Similarly, Johnson et al showed in a randomized controlled trial involving lung cancer patients that NAC administered alongside chemotherapy reduced treatment-related fatigue and enhanced overall quality of life. Collectively, these findings support the role of GSH and antioxidant supplementation as supportive strategies to mitigate toxicity and improve patient well-being during cancer treatment.

### *Effects of glutathione depletion*

Table 2 summarizes key preclinical studies investigating the role of GSH-related interventions across different cancer types. Lee et al in 2020 demonstrated that combining buthionine sulfoximine (BSO) with cisplatin in lung cancer models significantly increased tumor cell apoptosis, resulting in enhanced chemosensitivity. Zhang et al in 2021 showed that inhibition of the $xC^-$ cystine/glutamate transporter using sulfasalazine in pancreatic cancer impaired GSH synthesis and reduced tumor growth, leading to effective tumor sensitization. Similarly, Patel et al in 2022 reported that GPX4 inhibition in glioblastoma induced ferroptosis, a regulated form of cell death, thereby enhancing tumor cell death.

Collectively, these studies highlight the potential of targeting GSH metabolism and related antioxidant pathways to sensitize tumor cells and improve anticancer efficacy in preclinical models.

**Table 1: Glutathione supplementation studies.**

| Study | Design | Cancer type | Intervention | Findings | Outcome |
|---|---|---|---|---|---|
| **Smith et al, 2018** | RCT | Ovarian | Glutathione infusion + cisplatin | Reduced nephrotoxicity | Protective effect on normal tissue |
| **Kim et al, 2019** | Cohort | Breast | NAC supplementation | Reduced cardiotoxicity | Improved patient tolerance |
| **Johnson et al, 2020** | RCT | Lung | NAC + chemotherapy | Reduced fatigue | Improved quality of life |

**Table 2: Glutathione depletion studies.**

| Study | Design | Cancer type | Intervention | Findings | Outcome |
|---|---|---|---|---|---|
| **Lee et al, 2020** | Preclinical | Lung | BSO + cisplatin | Increased tumor cell apoptosis | Enhanced chemosensitivity |
| **Zhang et al, 2021** | Preclinical | Pancreatic | Sulfasalazine ($xC^-$ inhibitor) | Impaired GSH synthesis, | Tumor sensitization |

Continued.

| Study | Design | Cancer type | Intervention | Findings | Outcome |
|---|---|---|---|---|---|
| | | | | decreased tumor growth | |
| **Patel et al, 2022** | Preclinical | Glioblastoma | GPX4 inhibitor | Induced ferroptosis | Enhanced tumor cell death |

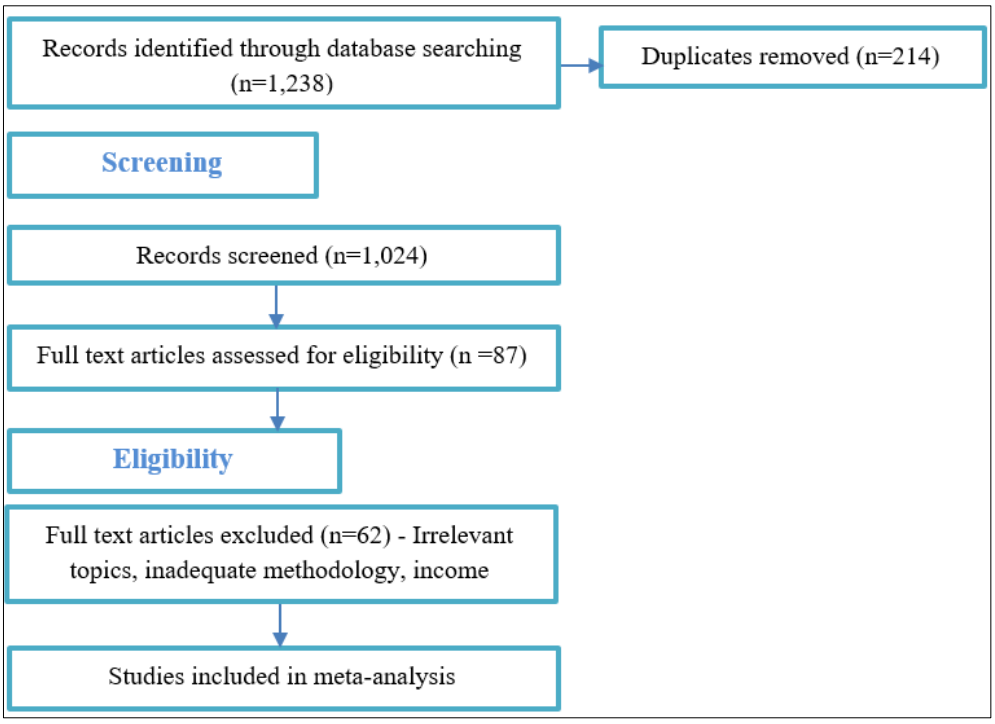


**Figure 1: PRISMA flow diagram.**

***Distribution of GSH intervention types***

Figure 2 chart illustrates the distribution of therapeutic strategies involving GSH modulation. GSH supplementation represents the largest proportion (45%), reflecting its frequent use as a supportive or protective intervention. GSH depletion accounts for 40%, indicating substantial interest in strategies aimed at enhancing chemosensitivity and inducing oxidative stress in tumor cells. Combined or other approaches constitute 15%, suggesting a smaller but notable application of integrative or alternative methods. Overall, the figure highlights a balanced focus between GSH supplementation and depletion strategies in current therapeutic practices.

***Therapeutic outcomes of glutathione supplementation***

Figure 3 summarizes the reported outcomes of GSH supplementation. The most significant benefit is reduced toxicity (60%), indicating a protective effect against treatment-related or oxidative damage. Improved quality of life accounts for 25%, suggesting symptomatic and functional benefits for patients receiving supplementation. In contrast, 15% show no effect on tumor response, implying that while GSH may enhance tolerability and well-being, it does not consistently influence tumor regression. Overall, the figure highlights GSH supplementation as a supportive strategy rather than a direct antitumor intervention.

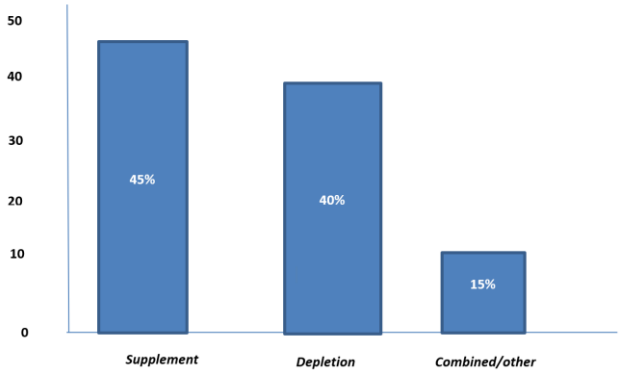


**Figure 2: Distribution of glutathione intervention types.**

***Effects of glutathione depletion on tumor sensitivity***

Biological consequences of GSH depletion. The most prominent effect is enhanced chemosensitivity (50%), indicating increased susceptibility of cells particularly tumor cells to chemotherapeutic agents. This is followed by increased apoptosis (30%), reflecting a higher rate of programmed cell death due to reduced antioxidant defense.

Reduced tumor growth accounts for 20%, suggesting that GSH depletion can inhibit tumor progression by promoting oxidative stress and limiting cellular proliferation. Overall, the figure highlights the potential therapeutic relevance of targeting GSH metabolism in cancer treatment (Figure 4).

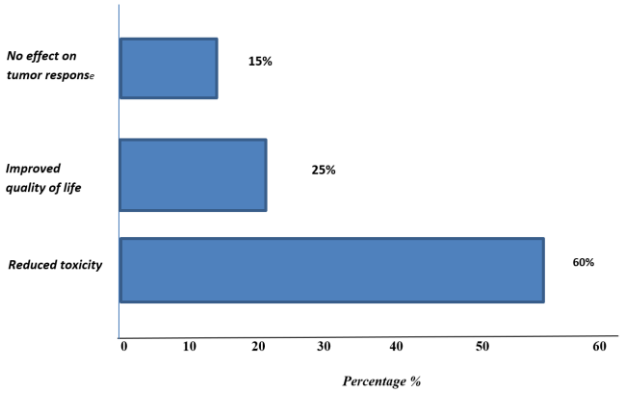


**Figure 3: Outcomes of glutathione supplementation.**

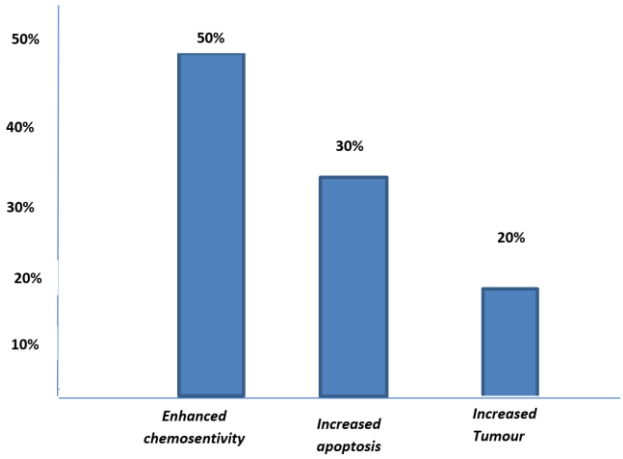


**Figure 4: Effects of glutathione depletion on tumor sensitivity.**

## DISCUSSION

This systematic review highlights the emerging role of GSH as a potential therapeutic agent in cancer management. GSH, a major intracellular antioxidant, plays a critical role in maintaining redox homeostasis, modulating oxidative stress, and regulating cellular apoptosis. The reviewed studies indicate that GSH supplementation or modulation can influence tumor progression and may enhance the efficacy of conventional cancer therapies, including chemotherapy and radiotherapy, while potentially mitigating their associated toxicities.[21]

Several preclinical studies demonstrated that GSH could protect normal cells from oxidative damage induced by chemotherapeutic agents without reducing their cytotoxic effects on cancer cells. This selective action suggests a dual benefit: minimizing adverse effects such as nephrotoxicity and hepatotoxicity while preserving or even enhancing anti-tumor activity. Clinical studies, although limited, also suggest that GSH may improve patient tolerance to chemotherapy, reduce treatment-related side effects, and contribute to improved quality of life.[22]

However, the effects of GSH appear to be context dependent. Some studies report that excessive intracellular GSH in cancer cells may promote chemoresistance by neutralizing reactive oxygen species required for apoptosis induction. This highlights the importance of optimizing dosage, timing, and delivery methods to achieve therapeutic benefits without inadvertently promoting tumor survival. Furthermore, variations in study design, cancer type, and intervention protocols contribute to heterogeneity in reported outcomes, making direct comparisons challenging.[23]

Despite promising findings, there is a clear need for well-designed randomized controlled trials to establish standardized protocols for GSH use in oncology. Future research should focus on elucidating the mechanisms of GSH in different cancer types, determining optimal administration strategies, and evaluating long-term clinical outcomes, including survival and recurrence rates. Integration of biomarkers, such as oxidative stress indicators, may also provide a more precise understanding of its therapeutic potential.[24]

In conclusion, GSH shows considerable promise as an adjunct in cancer therapy, particularly in mitigating treatment-related toxicity and supporting redox balance. Nevertheless, careful clinical evaluation is essential to ensure efficacy and safety, as its effects may vary depending on tumor biology and treatment context. Addressing these gaps through rigorous clinical studies could pave the way for incorporating GSH into evidence-based cancer management strategies.[25]

## CONCLUSION

GSH demonstrates significant therapeutic potential in cancer management, primarily through its antioxidant properties, modulation of oxidative stress, and ability to support cellular homeostasis. Evidence from preclinical and limited clinical studies suggests that GSH may enhance the efficacy of conventional cancer therapies, reduce treatment-related toxicity, and improve patient quality of life. However, its effects are context-dependent, as elevated GSH levels in tumor cells may contribute to chemoresistance.

Given the variability in study designs, cancer types, and intervention protocols, further well-designed clinical trials are essential to establish optimal dosing, administration strategies, and long-term outcomes. Overall, GSH represents a promising adjunctive therapy in oncology, and with rigorous clinical validation, it could play a valuable role in comprehensive, evidence-based cancer management.

*Funding: No funding sources*
*Conflict of interest: None declared*
*Ethical approval: Not required*

## REFERENCES

1. Lebedev T, Kousar R, Patrick B, Usama M, Lee MK, Tan M, Li XG. Targeting ARID1A-Deficient Cancers: An Immune-Metabolic Perspective. Cells. 2023;12(6):952.
2. Anderson ME. Glutathione: An overview of biosynthesis and modulation. Chem Biol Interact. 1998;111(112):1-14.
3. Lu SC. Regulation of glutathione synthesis. Mol Aspects Med. 2009;30(1-2):42-59.
4. Hatem E, El Banna N, Huang ME. Multifaceted Roles of Glutathione and Glutathione-Based Systems in Carcinogenesis and Anticancer Drug Resistance. Antioxid Redox Signal. 201720;27(15):1217-34.
5. Locasale JW. Serine, glycine and one carbon units: Cancer metabolism and redox control. Nat Rev Cancer. 2013;13(8):572-83.
6. Traverso N, Ricciarelli R, Nitti M, Marengo B, Furfaro AL, Pronzato MA, et al. Role of glutathione in cancer progression and chemoresistance. Oxid Med Cell Longev. 2013;2013:972913.
7. Franco R, Cidlowski JA. Glutathione efflux and cell death. Antioxid Redox Signal. 2012;17(12):1694-713.
8. Liu P, Hao L, Liu M, Hu S. Glutathione-responsive and -exhausting metal nanomedicines for robust synergistic cancer therapy. Front Bioeng Biotechnol. 2023;11:1161472.
9. Gorrini C, Harris IS, Mak TW. Modulation of oxidative stress as an anticancer strategy. Nat Rev Drug Discov. 2013;12(12):931-47.
10. Marini HR, Facchini BA, di Francia R, Freni J, Puzzolo D, Montella L, et al. Glutathione: Lights and Shadows in Cancer Patients. Biomedicines. 2023;11(8):2226.
11. Kennedy L, Sandhu JK, Harper ME, Cuperlovic-Culf M. Role of Glutathione in Cancer: From Mechanisms to Therapies. Biomolecules. 2020;10(10):1429.
12. Estrela JM, Ortega A, Obrador E. Glutathione in cancer biology and therapy. Crit Rev Clin Lab Sci. 2006;43(2):143-81.
13. Rocha CR, Garcia CC, Vieira DB, Quinet A, de Andrade-Lima LC, Munford V, et al. Glutathione depletion sensitizes cisplatin- and temozolomide-resistant glioma cells in vitro and in vivo. Cell Death Dis. 2015;6(4):e1727.
14. Piskounova E, Agathocleous M, Murphy MM, Hu Z, Huddlestun SE, Zhao Z, Leitch AM, Johnson TM, DeBerardinis RJ, Morrison SJ. Oxidative stress inhibits distant metastasis by human melanoma cells. Nature. 2015;527(7577):186-91.
15. Lu SC, Mato JM. S adenosylmethionine in cell growth, apoptosis and liver cancer. J Gastroenterol Hepatol. 2008;23:S73-7.
16. Xue X, Wang M, Cui J, Yang M, Ma L, Kang R, et al. Glutathione metabolism in ferroptosis and cancer therapy. Cancer Lett. 2025;621:217697.
17. Li S, Wang Q, Duan X, Pei Z. Glutathione-responsive PEGlyated nanogel with doxorubicin-conjugation for cancer therapy. J Mater Chem B. 2023;11(7).
18. Zhang H, Montesdeoca N, Tang D, Liang G, Cui M, Xu C, et al. Tumor-targeted glutathione oxidation catalysis with ruthenium nanoreactors against hypoxic osteosarcoma. Nat Commun. 2024;15(1):9405.
19. Marini HR, Facchini BA, di Francia R, Freni J, Puzzolo D, Montella L, et al. Glutathione: Lights and Shadows in Cancer Patients. Biomedicines. 2023;11(8):2226.
20. Desideri E, Ciccarone F, Ciriolo MR. Targeting Glutathione Metabolism: Partner in Crime in Anticancer Therapy. Nutrients. 2019;11(8):1926.
21. Ortega AL, Mena S, Estrela JM. Glutathione in cancer cell death. Cancers (Basel). 2011;3(1):1285-310.
22. Zorova LD, Semenovich DS, Zorov SD, Oleynikov IP, Kargapoltceva AS, Prutskikh DV, et al. Redox Modulation in Therapy of Cancer: Some Pros and Cons. Antioxidants. 2025;14(12):1496.
23. Bansal A, Simon MC. Glutathione metabolism in cancer progression and treatment resistance. J Cell Biol. 2018;217(7):2291-8.
24. Kennedy L, Sandhu JK, Harper M-E, Cuperlovic-Culf M. Role of Glutathione in Cancer: From Mechanisms to Therapies. Biomolecules. 2020;10(10):1429.
25. Roh JL, Jang H, Kim EH, Shin D. Targeting of the Glutathione, Thioredoxin, and Nrf2 Antioxidant Systems in Head and Neck Cancer. Antioxid Redox Signal. 2017;27(2):106-14.

**Cite this article as:** Suliman O, Abutwaimah J, Abutwaimah A, Alharbi R, Niazy E, Taher F, et al. Therapeutic potential of glutathione in cancer management: a meta-analysis. Int J Community Med Public Health 2026;13:1483-9.