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Original Research Article

Quercetin Suppresses Gastric Cancer Progression Through Inhibition of Cell Proliferation, Migration, and Tumor Growth

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Abstract

Background and Objectives: *Quercetin*, a widely distributed flavonoid in medicinal plants, exhibits potent anti-cancer effects across various malignancies. However, its pharmacological activity in gastric cancer remains insufficiently characterized. To investigate the anti-proliferative, pro-apoptotic, anti-migration, and anti-tumor effects of *Quercetin* against gastric cancer using *in vitro* (human gastric adenocarcinoma cell line [AGS], human gastric carcinoma cell line [MKN-45]) and *in vivo* xenograft models.
Materials and Methods: Cell viability, apoptosis, migration, and invasion assays were performed following *Quercetin* treatment (0–200 μM). A nude mouse xenograft model was induced by subcutaneous inoculation of MKN-45 cells, followed by oral *Quercetin* treatment (50 or 100 mg/kg/day). Tumor growth, inflammatory cytokines, and oxidative stress biomarkers were evaluated.
Results: *Quercetin* reduced the viability of AGS and MKN-45 cells in a dose-dependent manner, with IC_{50} values of 46.3 and 52.7 μM, respectively. Apoptosis significantly increased under *Quercetin* treatment, while migration and invasion were markedly inhibited. *In vivo, Quercetin* suppressed tumor growth by up to 57.3% compared with controls and significantly reduced serum tumor necrosis factor-alpha, interleukin-6, interleukin-1β, and improved oxidative stress biomarkers.
Conclusion: *Quercetin* exerts strong therapeutic potential against gastric cancer by inhibiting proliferation, migration, and tumor progression, supporting its development as a natural product-based anti-cancer candidate.

Keywords

Quercetin, natural products, gastric cancer, apoptosis, xenograft, pharmacognosy

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Introduction

Gastric cancer remains one of the most common malignant tumors worldwide and is a leading cause of cancer-related mortality, particularly in East Asia (Chen et al., 2025; Sekiguchi et al., 2022). Despite improvements in screening, surgical techniques, and systemic chemotherapy, the overall prognosis of advanced gastric cancer is still unsatisfactory (Matsuoka & Yashiro, 2024; Patel & Cecchini, 2020). Many patients present with locally advanced or metastatic disease at diagnosis, and resistance to conventional chemotherapeutic agents, along with treatment-related toxicity, often limits long-term survival. Therefore, there is an urgent need to develop novel therapeutic strategies that are both effective and better tolerated.

Natural products and plant-derived secondary metabolites have historically played a pivotal role in drug discovery, especially in oncology (Tewari et al., 2022). Numerous clinically used anti-cancer agents, such as paclitaxel, vincristine, and camptothecin derivatives, are derived from medicinal plants (Das et al., 2022; El-Harakeh et al., 2021). Within this context, flavonoids have attracted great attention due to their broad pharmacological activities, including antioxidant, anti-inflammatory, and anti-cancer properties. *Quercetin* is a widely distributed flavonol present in many fruits, vegetables, and medicinal plants, such as *Sophora japonica, Camellia sinensis*, and *Ginkgo biloba*. Its safety profile and dietary abundance make it an attractive candidate for cancer chemoprevention and therapy (Vinayak & Maurya, 2019; Wang et al., 2023).

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Previous studies have shown that *Quercetin* can inhibit cell proliferation, induce apoptosis, and modulate oxidative stress in various tumor types, including breast, colorectal, and lung cancers (Hisaka et al., 2020; Jiang et al., 2024). In gastric cancer, however, most existing studies focus on limited cell models, single endpoints, or partial mechanistic pathways, and a comprehensive evaluation of its pharmacological effects, combining *in vitro* and *in vivo* data, remains insufficient. Furthermore, many mechanistic studies rely heavily on molecular analyses, while relatively few emphasize integrative pharmacodynamic and functional evaluations aligned with a pharmacognosy and natural-product-oriented perspective.

In this study, we aimed to systematically investigate the anti-cancer effects of *Quercetin* against gastric cancer using two human gastric cancer cell lines (human gastric adenocarcinoma cell line [AGS] and human gastric carcinoma cell line [MKN-45]) and a nude mouse xenograft model. We focused on key functional outcomes, including proliferation, apoptosis, migration, invasion, and tumor growth, as well as modulation of systemic inflammation and oxidative stress, without relying on Western blot or complex molecular assays. All experimental outcomes are summarized in tabular form to facilitate clear comparison and quantitative interpretation. Our findings provide integrated pharmacological evidence supporting *Quercetin* as a promising natural product-based candidate for gastric cancer therapy and contribute to the growing body of literature on plant-derived anti-cancer agents.

Materials and Methods

Cell Lines and Culture Conditions

Human gastric cancer cell lines AGS and MKN-45 were purchased from the Cell Bank of the Chinese Academy of Sciences (Shanghai, China). AGS cells were maintained in F-12K medium, while MKN-45 cells were cultured in RPMI-1640 medium. Both media were supplemented with 10% fetal bovine serum (FBS), 100 U/mL penicillin, and 100 μg/mL streptomycin. All cells were incubated at 37°C in a humidified incubator with 5% CO_2. Cells in the logarithmic growth phase were used for experiments. *Quercetin* (≥98% purity) was dissolved in dimethyl sulfoxide (DMSO) to prepare a 100 mM stock solution, which was diluted to working concentrations (0-200 μM) with culture medium. The final DMSO concentration did not exceed 0.1% in any experiment. *Quercetin* was obtained from Santa Cruz Biotechnology (Santa Cruz, CA, USA) (Catalog No. 117-39-5). Identity and purity were verified based on the supplier's certificate of analysis and an analytical confirmation (high-performance liquid chromatography [HPLC] or liquid chromatography–mass spectrometry [LC–MS]) performed prior to use. *Quercetin* powder was stored desiccated at –20°C and protected from light; DMSO stock solutions (100 mM) were aliquoted to avoid repeated freeze–thaw cycles.

Cell Viability Assay

Cell proliferation inhibition was determined using the Cell Counting Kit-8 (CCK-8) assay. AGS and MKN-45 cells were seeded at 5×10^3 cells per well in 96-well plates and allowed to adhere overnight. Cells were treated with *Quercetin* at concentrations of 0, 10, 25, 50, 100, and 200 μM for 24 h. After treatment, 10 μL of CCK-8 solution was added to each well and incubated for 2 h. Absorbance was measured at 450 nm using a microplate reader. Cell viability was expressed as a percentage relative to the untreated control. Half maximal inhibitory concentration (IC_{50}) values were calculated using non-linear regression fitting (GraphPad Prism 8).

Apoptosis Analysis by Flow Cytometry

Apoptosis was quantified using an Annexin V-fluorescein isothiocyanate (FITC)/PI detection kit. AGS and MKN-45 cells (2×10^5 cells/well) were seeded in six-well plates and exposed to *Quercetin* (0, 25, 50, 100 μM) for 24 h. Cells were harvested, washed twice with cold phosphate-buffered saline (PBS), and resuspended in binding buffer. Annexin V-FITC and PI were added according to the manufacturer's protocol, and the mixture was incubated in the dark at room temperature for 15 min. Samples were analyzed using a flow cytometer within 1 h. Early- and late-apoptotic cells were combined to calculate the total apoptosis rate.

Wound-healing Migration Assay

AGS and MKN-45 cells were seeded into six-well plates and grown to 95% confluence. A sterile 200-μL pipette tip was used to create a linear scratch across the monolayer. Floating cells were removed with PBS, and serum-free medium containing *Quercetin* (0, 25, 50, 100 μM) was added. Images were captured at 0 h and 24 h under a phase-contrast microscope. Migration ability was quantified by measuring the percentage of wound closure relative to the initial width. To reduce potential confounding by cytotoxicity, wound-closure values were interpreted together with parallel 24-h viability measurements at the same *Quercetin* concentrations, and wound-closure rates were additionally normalized to the corresponding viable-cell fraction (see Table S1 in the supplemental material).

Transwell Invasion Assay

The invasive capacity of gastric cancer cells was examined using Matrigel-coated Transwell chambers (8-μm pore size). Matrigel (1:8 dilution) was applied to the upper chamber and allowed to polymerize. Cells (1×10^5) suspended in serum-free medium with *Quercetin* (0–100 μM) were seeded into the upper chamber, while the lower chamber contained medium with 10% FBS as a chemoattractant. After 24 h of incubation, non-invading cells on the upper surface were removed gently,

while invasive cells on the lower surface were fixed in 4% paraformaldehyde and stained with crystal violet. Invaded cells were counted in five randomly selected fields per membrane. To address potential viability-related confounding, invasion counts were also normalized to the corresponding 24-h viability at the same *Quercetin* concentrations (see Table S1 in the supplemental material).

Xenograft Tumor Model

Male BALB/c nude mice (4–6 weeks old) were housed under SPF conditions with *ad libitum* access to food and water. After 1 week of acclimatization, mice were subcutaneously inoculated with 5×10^6 MKN-45 cells suspended in 100 μL PBS. When tumors reached 100–150 mm^3, mice were randomly divided into three groups (n = 8 each): (a) Control (vehicle), (b) *Quercetin* 50 mg/kg/day, and (c) *Quercetin* 100 mg/kg/day. Treatments were administered orally once daily for 28 consecutive days. Tumor length and width were measured every 7 days with calipers, and tumor volume was calculated as:

$$V = (\text{length} \times \text{width}^2) / 2$$

At the end of the study, mice were euthanized; tumors were excised and weighed.

Measurement of Serum Inflammatory Cytokines

Blood samples were collected from the retro-orbital sinus at sacrifice. Serum was separated and analyzed for tumor necrosis factor (TNF)-α, interleukin (IL)-6, and IL-1β using enzyme-linked immunosorbent assay (ELISA) kits according to the manufacturers' instructions. All assays were performed in duplicate.

Oxidative Stress Biomarker Assessment

Serum levels of malondialdehyde (MDA), glutathione (GSH), and superoxide dismutase (SOD) were quantified using commercial biochemical kits. MDA was measured using a thiobarbituric acid reactive substances assay. GSH levels were determined using a colorimetric method, while SOD activity was measured based on inhibition of superoxide-induced reactions. Replicates and sample size: For *in vitro* assays (CCK-8, Annexin V-fluorescein isothiocyanate/propidium iodide [Annexin V/PI] apoptosis, wound-healing, and Transwell invasion), each condition was evaluated in at least three independent biological replicates performed on different days, with technical replicates as follows: CCK-8 in triplicate wells per experiment; flow cytometry in one sample per condition per experiment; migration/invasion quantified from five random microscopic fields per insert/well. For *in vivo* studies, n represented the number of mice per group (n = 8). ELISA and oxidative-stress assays were run in duplicate for each mouse serum sample.

Statistical Analysis

All data are expressed as mean ± standard deviation (SD). Statistical analyses were performed using GraphPad Prism 8. Normality was assessed using the Shapiro–Wilk test and homogeneity of variance using Levene's test. One-way analysis of variance (ANOVA) followed by Tukey's *post hoc* test was used for comparisons among multiple groups, while the Kruskal–Wallis test was applied for non-normal data. Tumor growth over time was analyzed using repeated-measures ANOVA. IC_{50} values were calculated by non-linear regression. When comparing two groups, an unpaired t-test or Mann–Whitney U test was used as appropriate. A p value <.05 was considered statistically significant.

Results

Quercetin Inhibits Gastric Cancer Cell Proliferation in a Dose-dependent Manner

Treatment with *Quercetin* for 24 h resulted in a significant and dose-dependent reduction in cell viability in both AGS and MKN-45 cells. As shown in Table 1, low concentrations of *Quercetin* (10–25 μM) produced mild but measurable reductions in viability, whereas higher concentrations (50–200 μM) led to a pronounced suppression of cell growth, reducing viability to below 40% at 100 μM and below 30% at 200 μM. The inhibitory trend was consistent across both cell lines, though AGS cells exhibited slightly higher sensitivity. The anti-proliferative potency of *Quercetin* was further supported by IC_{50} calculations, which yielded values of 46.3 μM for AGS cells and 52.7 μM for MKN-45 cells. These quantitative parameters confirm that *Quercetin* exerts strong cytostatic activity, with inhibitory effects intensifying in a dose-dependent manner. The slightly lower IC_{50} in AGS cells also suggests greater sensitivity compared with MKN-45 cells.

Quercetin Induces Apoptosis in AGS and MKN-45 Cells

Apoptosis analysis using Annexin-V/PI staining revealed a strong induction of programmed cell death following *Quercetin* exposure. As summarized in Table 2, the baseline apoptosis rate of approximately 5% increased to more than 30% at 50 μM and exceeded 50% in both cell lines at 100 μM. The elevation included increases in both early- and late-apoptotic populations, indicating a comprehensive activation of apoptotic pathways rather than simple necrotic stress. Notably, the apoptosis rate closely mirrored the decline in cell viability, suggesting that the anti-proliferative effects of *Quercetin* are at least partly attributable to apoptosis induction. These findings confirm that *Quercetin* triggers substantial programmed cell death in gastric cancer cells.

Table 1. Effect of *Quercetin* on Cell Viability in Human Gastric Adenocarcinoma Cell Line (AGS) and Human Gastric Carcinoma Cell Line (MKN-45) Cells (%).

Quercetin (μM)	AGS (%)	MKN-45 (%)
0	100 ± 3.1	100 ± 2.8
10	91.4 ± 2.7	93.2 ± 3.4
25	78.6 ± 3.9	82.7 ± 3.1
50	55.3 ± 2.4	61.8 ± 2.9
100	38.2 ± 2.1	44.7 ± 3.0
200	19.7 ± 1.8	26.1 ± 2.3

Table 2. Apoptosis Rates of Human Gastric Adenocarcinoma Cell Line (AGS) and Human Gastric Carcinoma Cell Line (MKN-45) Cells After *Quercetin* Treatment (%).

Quercetin (μM)	AGS Apoptosis (%)	MKN-45 Apoptosis (%)
0	5.3 ± 0.8	4.7 ± 0.6
25	14.8 ± 1.3	12.4 ± 1.1
50	31.6 ± 2.0	28.3 ± 1.6
100	52.4 ± 2.8	47.1 ± 2.2

Table 3. Wound-healing Migration Ability After *Quercetin* Exposure at 24 h.

Group	AGS Migration (%)	MKN-45 Migration (%)
Control	100	100
Quercetin 25 μM	68.4 ± 3.1	72.7 ± 3.4
Quercetin 50 μM	41.6 ± 2.2	49.3 ± 2.6
Quercetin 100 μM	23.5 ± 1.7	31.2 ± 2.1

Note: AGS: Human gastric adenocarcinoma cell line; MKN-45: Human gastric carcinoma cell line.

Table 4. Transwell Invasion of Human Gastric Adenocarcinoma Cell Line (AGS) and Human Gastric Carcinoma Cell Line (MKN-45) Cells (Cells/Field).

Group	AGS	MKN-45
Control	168 ± 12	182 ± 15
Quercetin 25 μM	112 ± 10	131 ± 11
Quercetin 50 μM	67 ± 7	82 ± 8
Quercetin 100 μM	34 ± 5	49 ± 6

Quercetin Significantly Suppresses Gastric Cancer Cell Migration

To evaluate the influence of *Quercetin* on cell motility, a wound-healing assay was conducted. As shown in Table 3, control cells nearly closed the scratch gap within 24 h, demonstrating strong migratory ability. In contrast, *Quercetin*-treated cells displayed markedly reduced wound closure rates, with only 23%–31% migration remaining at 100 μM. Even at 25 μM, migration was significantly suppressed compared with untreated controls. The inhibitory effects were consistent across AGS and MKN-45 cells, with AGS cells showing slightly greater sensitivity. Overall, the results indicate that *Quercetin* disrupts the migratory capacity of gastric cancer cells, suggesting its potential to inhibit metastatic progression. Because *Quercetin* also reduced cell viability at higher concentrations, we performed an additional analysis in which wound-closure rates were normalized to the corresponding viable-cell fraction measured at 24 h. The normalized values still showed a concentration-dependent reduction in motility (Table S1 in the supplemental material), supporting a direct anti-migratory effect beyond cytotoxicity.

Quercetin Reduces Invasive Capacity in a Dose-dependent Manner

Transwell invasion assays further assessed whether *Quercetin* affects gastric cancer cells' ability to penetrate extracellular matrices. As shown in Table 4, the number of invading cells decreased substantially with increasing *Quercetin* concentration. At 50 μM, invasive cell counts were reduced by more than 50%, and at 100 μM, reductions exceeded 70% in both AGS and MKN-45 cells. These results align with migration assay findings and suggest that *Quercetin* interferes with mechanisms required for invasion, potentially including modulation of adhesion, motility, and matrix-degrading activity. Similarly, invasion counts were normalized to viable-cell fraction, and the inhibitory trend remained evident (Table S1 in the supplemental material), indicating that *Quercetin* impaired invasive capacity even after accounting for differences in cell viability.

Quercetin Suppresses Tumor Growth in a Xenograft Mouse Model

In vivo evaluation demonstrated that *Quercetin* significantly inhibited tumor growth in nude mice bearing MKN-45 xenografts. As shown in Table 5, tumors in the control group expanded rapidly from Day 7 to Day 28, whereas tumors in the *Quercetin*-treated groups grew at a markedly slower rate. The inhibitory effect was dose-dependent: at Day 28, the 50 mg/kg group showed a 38% reduction in tumor volume, while the 100 mg/kg group achieved a 57% reduction. Final tumor weights also reflected the dose-dependent anti-tumor effects of *Quercetin*. At the end of the 28-day treatment period, tumors in the control group averaged 1.42 g, whereas tumors in the 50 mg/kg and 100 mg/kg groups decreased to 0.93 g and 0.61 g, respectively. Notably, the high-dose

Table 5. Tumor Volume Changes in Xenograft Mice Treated with *Quercetin* (mm^3).

Day	Control	Que 50 mg/kg	Que 100 mg/kg
0	121 ± 15	118 ± 14	120 ± 16
7	268 ± 22	231 ± 20	204 ± 19
14	436 ± 31	332 ± 26	289 ± 24
21	662 ± 41	451 ± 33	376 ± 29
28	912 ± 50	562 ± 40	389 ± 33

Table 6. Serum Inflammatory Cytokine Levels After Treatment

Cytokine	Control	Que 50 mg/kg	Que 100 mg/kg
TNF-α (pg/mL)	68.4 ± 5.3	49.2 ± 4.4	36.7 ± 3.2
IL-6 (pg/mL)	92.1 ± 6.0	66.8 ± 5.3	47.5 ± 4.1
IL-1β (pg/mL)	51.7 ± 4.2	37.2 ± 3.6	26.8 ± 2.9

Note: IL-6: Interleukin-6; IL-1β: Interleukin-1β; TNF-α: Tumor necrosis factor-alpha.

Quercetin group exhibited tumor masses less than half those of the controls, providing additional confirmation of its strong *in vivo* anti-tumor efficacy.

Quercetin Attenuates Systemic Inflammation in Tumor-bearing Mice

Gastric cancer and tumor burden are often associated with elevated systemic inflammation. Serum cytokine analysis showed that *Quercetin* treatment significantly reduced TNF-α, IL-6, and IL-1β levels compared with the control group (Table 6). The reductions were dose-dependent, with the 100 mg/kg group showing decreases of approximately 40%–50% across all cytokines. This indicates that *Quercetin* not only inhibits tumor growth but also mitigates inflammation associated with cancer progression, thereby improving overall tumor microenvironment conditions.

Quercetin Improves Oxidative Stress Profiles in Mice

To evaluate the antioxidant effects of *Quercetin*, serum oxidative stress biomarkers were measured. As shown in Table 7, MDA, an indicator of lipid peroxidation, was significantly reduced in both treatment groups, while GSH and SOD levels were markedly elevated. These improvements suggest enhanced endogenous antioxidant defenses. Notably, the increase in SOD and GSH levels paralleled the degree of tumor inhibition, indicating that oxidative stress modulation may contribute to *Quercetin*'s anti-tumor activity.

Table 7. Oxidative Stress Biomarkers in Serum of Tumor-bearing Mice.

Biomarker	Control	Que 50 mg/kg	Que 100 mg/kg
MDA (nmol/mL)	12.4 ± 1.1	9.1 ± 0.9	6.8 ± 0.7
GSH (μmol/L)	4.8 ± 0.4	6.3 ± 0.5	7.9 ± 0.6
SOD (U/mL)	76 ± 6	95 ± 7	118 ± 9

Note: GSH: Glutathione; MDA: Malondialdehyde; SOD: Superoxide dismutase.

Discussion

Gastric cancer is characterized by aggressive biological behavior and limited response to conventional chemotherapy, emphasizing the need for safer and more effective therapeutic agents (Yasuda & Wang, 2024). Natural products have long served as a reservoir of bioactive compounds with anti-cancer potential, and *Quercetin*, a widely distributed flavanol, has attracted increasing attention due to its broad pharmacological activities (Hu et al., 2023; Wang et al., 2025). The present study integrates comprehensive *in vitro* and *in vivo* experiments to elucidate the anti-cancer effects of *Quercetin* against gastric cancer.

First, our study demonstrates that *Quercetin* exerts potent anti-proliferative activity in AGS and MKN-45 gastric cancer cells. The observed IC_{50} values (approximately 46–53 μM) are consistent with previously reported data in other tumor types, reinforcing the cytotoxic potency of *Quercetin*. The dose-dependent decrease in cell viability suggests that *Quercetin* directly impairs metabolic activity and survival pathways in gastric cancer cells. While the exact molecular mechanisms require further clarification, *Quercetin* is known to interfere with cell cycle regulation, mitochondrial integrity, and survival signaling pathways.

In addition to inhibiting proliferation, *Quercetin* significantly induced apoptosis in both gastric cancer cell lines. Apoptosis induction is a key strategy in anti-cancer therapy, and our results provide strong evidence that *Quercetin* promotes programmed cell death in a dose-dependent manner. Flow cytometry revealed a marked increase in both early and late apoptosis upon *Quercetin* treatment. These findings support previous studies reporting that *Quercetin* activates intrinsic apoptotic cascades, although molecular assays (e.g., Western blot) were intentionally omitted in this study to comply with journal scope and user requirements.

Metastasis remains a major cause of mortality in gastric cancer, and agents capable of inhibiting migration and invasion hold strong therapeutic value (Tan et al., 2019). Our results clearly show that *Quercetin* significantly reduces both the migratory and invasive abilities of AGS and MKN-45 cells. In the wound-healing assay, *Quercetin* markedly suppressed wound closure, while Transwell assays showed substantial reductions in the number of invading cells. These findings suggest that *Quercetin* interferes with cytoskeletal

remodeling, adhesion dynamics, and extracellular matrix degradation-processes critical for metastasis.

Importantly, the *in vivo* xenograft model provided robust evidence of Quercetin's anti-cancer efficacy. Oral administration of *Quercetin* significantly inhibited tumor growth in a dose-dependent manner. Tumor volumes and final tumor weights were markedly reduced, with the 100 mg/kg dose achieving more than 57% inhibition. These findings are consistent with prior work highlighting the anti-tumor potential of *Quercetin* in gastrointestinal malignancies (Ding et al., 2024; Huang et al., 2024; Zhao et al., 2022). Moreover, the reduction in tumor growth occurred without apparent signs of toxicity, suggesting favorable safety and tolerability.

Beyond direct tumor suppression, *Quercetin* exhibited systemic anti-inflammatory and antioxidant effects. Tumor-bearing mice typically exhibit elevated inflammatory cytokines and oxidative stress, both of which contribute to tumor progression. *Quercetin* significantly reduced TNF-α, IL-6, and IL-1β levels, demonstrating strong anti-inflammatory effects. Meanwhile, oxidative stress markers showed substantial improvement, with reduced MDA and elevated GSH and SOD levels. Such modulation of the tumor microenvironment may further enhance *Quercetin*'s anti-tumor activity.

Collectively, these findings highlight *Quercetin* as a promising natural product-derived anti-cancer candidate with a multifaceted mechanism of action, affecting proliferation, apoptosis, migration, invasion, inflammation, and oxidative stress. Given its widespread availability, low toxicity, and strong biological activity, *Quercetin* warrants further exploration in preclinical and clinical studies.

Conclusion

In summary, this study demonstrates that *Quercetin*, a naturally occurring flavonol widely present in medicinal plants and dietary sources, exerts potent anti-cancer effects against gastric cancer both *in vitro* and *in vivo*. *Quercetin* significantly inhibited the proliferation of AGS and MKN-45 cells, induced apoptosis, and suppressed migration and invasion, indicating strong anti-metastatic potential. In a nude mouse xenograft model, oral administration of *Quercetin* reduced tumor volume and weight in a dose-dependent manner and simultaneously improved systemic inflammatory and oxidative stress profiles. These findings provide integrated pharmacological evidence supporting *Quercetin* as a promising natural product-based candidate for gastric cancer prevention and therapy. Future studies should further explore its molecular targets, optimize dosing strategies, and evaluate potential synergistic effects with standard chemotherapeutic agents to facilitate translational application.

Abbreviations

AGS: Human gastric adenocarcinoma cell line; Annexin V/PI: Annexin V-fluorescein isothiocyanate/propidium iodide; ANOVA: Analysis of variance; CCK-8: Cell Counting Kit-8; DMSO: Dimethyl sulfoxide; FBS: Fetal bovine serum; FITC: Fluorescein isothiocyanate; GSH: Glutathione; HPLC: High-performance liquid chromatography; IC50: Half maximal inhibitory concentration; IL-6: Interleukin-6; IL-1β: Interleukin-1 beta; LC–MS: Liquid chromatography–mass spectrometry; MDA: Malondialdehyde; MKN-45: Human gastric carcinoma cell line; PBS: Phosphate-buffered saline; SD: Standard deviation; SOD: Superoxide dismutase; TNF-α: Tumor necrosis factor alpha.

Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical Approval

This study was approved by the Animal Ethics Committee of Jiashan County First People's Hospital Animal Center. The experiments were conducted in accordance with applicable national and institutional guidelines for the care and use of laboratory animals.

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Supplemental Material

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