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# Pentagalloylglucose Suppresses Glioblastoma Progression via Wnt/β-Catenin Pathway Inhibition and EMT Reversal

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**ABSTRACT**

**Background:** Glioblastoma multiforme (GBM) is the most lethal primary brain tumor. GBM exhibits rapid growth and invasiveness along with a nadir prognosis. Epithelial–mesenchymal transition (EMT), combined with activated Wnt/β-catenin signaling, contributes to GBM progression and associated therapy resistance. *Pentagalloylglucose* (PGG), a polyphenolic compound from nature, has been shown to be anticancer in multiple cancers by inhibiting proliferation, migration, and EMT. The objectives are to demonstrate the effects of PGG on GBM cells and examine the modulation of EMT and the Wnt/β-catenin pathway.

**Methods:** U87-MG cells were treated with PGG (0.5–40 μM) for 24, 48, and 72 hours. Cell viability was assessed using the MTT assay. The expression levels of epithelial–mesenchymal transition (EMT) markers, including *E-cadherin*, *N-cadherin*, *Vimentin*, *Snail*, *Slug*, as well as *β-catenin*, were quantified by qRT-PCR. Cell migration was evaluated using a wound healing assay. Statistical analysis was performed using one-way ANOVA followed by Tukey's post hoc test.

**Results:** PGG demonstrated a marked reduction in cell viability in a dose- and duration-dependent manner with $IC_{50}$ values at 18.4, 12.7, and 8.9 μM at 24, 48, and 72 hours, respectively. The upregulation of *E-cadherin* and downregulation of *N-cadherin*, *Vimentin*, and the *Snail* protein show that mesenchymal markers are being transcriptionally silenced. It was also a striking loss of *β-catenin* expression, which suggests Wnt/β-catenin suppression. Wound healing assay showed that PGG treatment resulted in a marked reduction of cell migration.

**Conclusion**: PGG significantly inhibits the progression of GBM by inhibiting EMT and downregulating the Wnt/β-catenin signaling pathways. Overall, PGG has potential as a natural, low-toxicity therapeutic or combinatory drug for glioblastoma, and future studies in vivo and in human trials will be needed to reaffirm this conclusion.

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## INTRODUCTION

Glioblastoma multiforme (GBM) is a grade IV tumor by the World Health Organization and is the most aggressive of these cancer subtypes. GBM is characterized by high proliferation, high angiogenic ability, poor prognosis, and resistance to medication. The prevalence of glioblastoma multiforme (GBM) is approximately 2-3 per 100,000 population and is responsible for 15-20% of all primary brain tumours. GBM primarily presents in older patients

with a median age of diagnosis at 64 years. Despite developments in the field of neuro-oncology, the median survival in GBM patients remains around 12-15 months with a < 5% survival at 5 years (1, 2).

One of the key biological processes implicated in GBM development is epithelial–mesenchymal transition (EMT), a reversible transdifferentiation process wherein cells relinquish the epithelial characteristics (e.g., *E-cadherin*-mediated cell–cell attachment) and gain mesenchymal characteristics (e.g., heightened motility, invasiveness, and apoptosis resistance). Epithelial-mesenchymal transition (EMT) promotes the acquisition of cancer stem cell-like characteristics, which result in tumor recurrence and drug resistance (3, 4).

The Wnt/β-catenin signaling pathway is an essential and evolutionarily conserved mechanism that plays a key role in regulating epithelial–mesenchymal transition (EMT), embryogenesis, tissue homeostasis, and stem cell maintenance. In cancer, aberrant activation of this pathway leads to nuclear accumulation of *β-catenin*, which associates with TCF/LEF transcription factors to drive the expression of EMT-related genes, including Snail, Slug, ZEB1, and Twist. In Glioblastoma multiforme, dysregulation of Wnt/β-catenin signaling has been linked to enhanced tumor cell migration, invasion, angiogenesis, and therapeutic resistance. Therefore, targeting this pathway pharmacologically represents a promising strategy for reducing the aggressiveness of GBM (5-7).

1,2,3,4,6-*Pentagalloylglucose* (PGG), a natural polyphenolic molecule classified as a hydrolyzable tannin, is prevalent in medicinal plants such as Punica granatum, Rhus chinensis, and Terminalia chebula. The anticancer effects of PGG demonstrated inhibition of proliferation, induction of apoptosis and autophagy, inhibition of angiogenesis, and reduction of metastatic potential by regulating many oncogenic pathways populations (e.g., Wnt/β-catenin and GSK3β/β-catenin) (8, 9).

PGG has recently been shown to inhibit epithelial-mesenchymal transition (EMT) in various cancer models that displayed increased epithelial markers (e.g., *E-cadherin*) and reduced mesenchymal markers (e.g., *vimentin*, *N-cadherin*), as a result of inhibiting Wnt/β-catenin transcriptional activity (9, 10). Additionally, PGG has demonstrated advantageous effects in glioma cell lines by reducing fatty acid production, influencing energy metabolism, and lessening invasive properties (8). The results suggest that PGG may act as a significant inhibitor of GBM growth through various molecular pathways.

Given the significance of epithelial-mesenchymal transition (EMT) and Wnt/β-catenin signaling in the pathogenesis of glioblastoma (GBM), along with the increasing evidence supporting the anti-EMT properties of PGG- particularly in relation to the Wnt/β-catenin signaling pathway- this study aims to clarify the mechanistic connection between PGG treatment and the temozolomide (TMZ)-induced suppression of EMT in GBM cells. A deeper understanding of PGG may facilitate its potential application as either an adjunct or primary treatment for highly invasive brain tumors.

## MATERIALS AND METHODS

### Reagents and Chemicals

PGG was obtained from Sigma-Aldrich (St. Louis, MO, USA; CAS No.: 14937-32-7). A 10 mM stock solution was prepared by dissolving the compound in DMSO and stored at −20°C. Dulbecco's Modified Eagle Medium (DMEM), fetal bovine serum (FBS), penicillin–streptomycin (Pen/Strep), and trypsin–EDTA were purchased from Gibco (Thermo Fisher Scientific, USA). The Add Prep Total RNA Extraction Kit, AddScript cDNA Synthesis Kit, and SYBR Green qPCR Master Mix were supplied by Addbio. Primers targeting epithelial–mesenchymal transition (EMT)-associated genes, including *E-cadherin, N-cadherin, Vimentin, Snail, Slug, and β-catenin*, as well as the housekeeping gene *GAPDH*, were synthesized by GenScript in China.

### Cell Culture and Treatment

The U87-MG was obtained from IBRC, Iran (cell code: IBRC C10982), and maintained in DMEM medium supplemented with 10% fetal bovine serum (FBS) and 1% penicillin/streptomycin (Pen/Strep) under standard culture conditions at 37°C in a humidified incubator with 5% $CO_2$. Cells were seeded into 6-well plates at a density of $2 \times 10^5$ cells per well and incubated for 24 hours to allow attachment. Subsequently, cells were treated with PGG at concentrations of 0.5, 1, 5, 10, 20, and 40 µM for 24, 48, and 72 hours. Control groups received equal volumes of dimethyl sulfoxide (DMSO) at concentrations below 0.1%.

### Cell Viability Assay (MTT Assay)

Cell viability was assessed using the MTT assay. U87-MG cells were seeded in 96-well plates at a density of $5 \times 10^3$ cells per well and allowed to incubate overnight. Subsequently, the cells were treated with increasing concentrations of PGG (1–40 µM) for 24, 48, and 72 hours. Following treatment, 10 µL of MTT solution (5 mg/mL) was added to each well and incubated at 37°C for 4 hours. The resulting formazan crystals were dissolved in 100 µL of DMSO, and absorbance was measured at 570 nm using a microplate reader. Cell viability was expressed as a percentage relative to untreated control cells.

### RNA Extraction and Quantitative Real-Time PCR (qRT-PCR)

Total RNA was isolated using the Add Prep Total RNA Extraction Kit according to the manufacturer's protocol. RNA quantity and quality were assessed with a Nanodrop spectrophotometer. cDNA was synthesized from 1 µg of total RNA using the AddScript cDNA Synthesis Kit. Quantitative reverse transcription polymerase chain reaction (qRT-PCR) was carried out using SYBR Green Master Mix on a StepOnePlus Real-Time PCR System (Applied Biosystems, USA). Primers were designed based on sequences obtained from the NCBI database. Detailed primer specifications are provided in Table 1.

Relative gene expression was determined using the 2^-ΔΔct technique and normalized to *GAPDH*. All responses were conducted in triplicate.

### Cell Migration Assay

The wound healing assay approach solved for cell migration. In cell U87-MG, preparation in 6-well culture plates was continued until more than 90% confluency was attained. The cell suspension was scraped gently to form a wound, a cross-section. The cells were washed so that no debris was left, and subsequently, PGG was added; the cells were then moved to the plates microscopically. Captures were made during 0, 24, and 48 hours. Movement of the mouse and images captured were combined for data using the ImageJ software.

### Statistical Analysis

All experiments were carried out in triplicate, and the results are presented as mean ± standard deviation (SD). Statistical analysis was performed using GraphPad Prism 9 (GraphPad Software, CA, USA). Group comparisons were evaluated by one-way analysis of variance (ANOVA), followed by Tukey's post hoc test. A p-value less than 0.05 was considered indicative of statistical significance.

## RESULTS

### Cell Viability Assay (MTT Assay)

The MTT assay revealed that PGG reduced the viability of U87-MG glioblastoma cells in a clear dose- and time-dependent manner. At 24 h, low concentrations of PGG (0.5 and 1 µM) showed no statistically significant effect compared to the control group ($p > 0.05$). A mild but noticeable reduction in viability (~10–15%) was observed at 5 µM, whereas treatment with 10 µM resulted in approximately a 25% reduction in viability. Higher concentrations, i.e. 20 µM and 40 µM, significantly inhibited viability and reached ~50% and ~65% inhibition levels relative to the control ($p < 0.01$).

At 48 hours, the cytotoxic effects were more pronounced. At a concentration of 5 µM, PGG decreased cell viability by approximately 20%, whereas at 10 µM, the reduction was around 40%. At a concentration of 20 µM, cell viability declined to approximately 35% of the control level, while at 40 µM, viability fell below

**Table 1.** Primers used in qRT-PCR

| Gene | Primer sequence | Pcr product (bp) |
|---|---|---|
| ***E-cadherin*** | F: GAGAACGCATTGCCACATACA<br>R: ACCTTCCATGACAGACCCCTTAA | 158 |
| ***N-cadherin*** | F: TCTCTCACGCTGTGTCATCCAAC<br>R: CACAGAGGTTCCTGGAAGAGCAC | 152 |
| ***Vimentin*** | F: AGTCCACTGAGTACCGGAGAC<br>R: CATTTCACGCATCTGGCGTTC | 98 |
| ***Snail*** | F: TCCAGAGTTTACCCTTCCAGCA<br>R: CTTTCCCACTGTCCTCATCTG | 219 |
| ***Slug*** | F: CTCACCTCGGGAGCATACAG<br>R: GACTTACACGCCCCAAGGATG | 121 |
| ***β-catenin*** | F: CAATGACTCGAGCTCAGAGGGTAC<br>R: TTTAGCAGTTTTGTCAGTTCAGGGA | 223 |
| ***GAPDH*** | F: AATCCCATCACCATCTTCCAG<br>R: ATCAGCAGAGGGGGCAGAGA | 168 |

25% ($p < 0.001$).

Significant inhibitory effects were noted at 72 hours. Treatment with 5 μM PGG resulted in a decrease in viability of approximately 30% and treatment with 10 μM resulted in a decrease of about 55%. Viability decreased to less than 30% of controls with treatment of 20 μM. The maximum concentration tested, 40 μM, decreased cell viability to under 15%, demonstrating significant cytotoxicity ($p < 0.001$).

The calculated $IC_{50}$ values from the dose–response curves were approximately 18.4 μM at 24 hours, 12.7 μM at 48 hours, and 8.9 μM at 72 hours, indicating that extended exposure increases the anti-proliferative effects of PGG. The findings indicate that PGG significantly inhibits glioblastoma cell growth, with treatment concentration and duration being essential factors in its effectiveness.

### Gene Expression Analysis (qRT-PCR)

Real-time PCR analysis showed that PGG treatment significantly affected the expression of genes associated with EMT and the Wnt/β-catenin pathway in U87-MG glioblastoma cells in a manner consistent with suppression of EMT characteristics. The epithelial marker *E-cadherin* showed significant upregulation with transcript levels 2.0–3.5-fold more than the control, indicating enhancement of cell-to-cell adhesion characteristics. Conversely, the two mesenchymal markers *N-cadherin* and *Vimentin* were significantly downregulated with decreases to 0.4–0.6-fold or 0.3–0.5-fold of the control ($p < 0.001$), respectively. Expression of the EMT transcription factor *Snail* also showed a marked decrease. However, although *slug* expression decreased by a slight amount in the PGG treatment group relative to the untreated group, the difference was not statistically significant ($p > 0.05$), suggesting that treatment with PGG did not significantly impact *Slug* gene expression in the current experimental conditions.

*β-catenin*, a central mediator of the Wnt signaling pathway, was downregulated to 0.4–0.7-fold, suggesting inhibition of Wnt/β-catenin–dependent transcriptional activity.

### Cell migration (Wound healing assay)

The wound healing assay demonstrated that PGG treatment (10 μM) significantly diminished the migratory ability of U87-MG glioblastoma cells in comparison to the untreated control group. After 24 hours, the wound closure percentage in untreated cells was 62.66 ± 1.53%, while PGG-treated cells demonstrated a significantly reduced closure percentage of 40.67 ± 1.53% ($p < 0.01$). The inhibitory effect was significantly greater at 48 hours, with control cells demonstrating 89.67 ± 1.53% closure, whereas PGG-treated cells exhibited only 45.33 ± 1.53% closure ($p < 0.001$). The microscopy showed that at every time point, the PGG-treated group had a reduced number of cells invading the wound area, which was in accordance with the results of the MTT assay, which also supports the conclusion that PGG expresses time-dependent inhibition of glioblastoma cell migration.

## DISCUSSION

Among the many primary brain tumors, GBM can have the most aggressive, lethal, and devastating effects; it is characterized by rapid growth and invasiveness and is often resistant to treatment. Despite some advances in surgery, radiotherapy, and chemotherapy, patients appear to have a median overall survival of under 15 months (1). One of the key molecular processes involved in the progression of GBM is the EMT. This process enables cancer cells to improve their migration and invasion capabilities, as well as to develop resistance to drugs (3). The Wnt/β-catenin pathway is imperative for activation of EMT in GBM, and researchers are attempting its targeting as a potential new way to inhibit tumor aggressiveness (5).

The findings of this study demonstrated that the natural compound PGG decreased the viability of GBM (U87-MG) cells in both a dose- and time-dependent manner. qRT-PCR analysis revealed a marked upregulation of the epithelial marker *E-cadherin*, alongside a reduction in mesenchymal markers, including *N-cadherin*, vimentin, and Snail. In addition, the expression level of *β-catenin*, a central element of the Wnt/β-catenin signaling pathway, was diminished. Furthermore, migration assay results showed that PGG significantly suppressed cell motility, suggesting a decrease in tumor cell invasive capacity.

The present study indicated that PGG inhibits the EMT process in GBM cells by decreasing the expression of mesenchymal genes and increasing *E-cadherin*. Furthermore, the reduction of the *β-catenin* level indicated the inhibition of the Wnt/β-catenin pathway. Inhibition of the Wnt/β-catenin pathway by PGG and reduction of EMT factors are findings that have been confirmed not only in the present study but also in other independent studies. Abnormal activation of the Wnt/β-catenin pathway is considered a key mechanism in invasion and drug resistance in GBM. In this pathway, nuclear accumulation of *β-catenin* leads to activation of EMT genes such as *Snail* and *Slug*, which is accompanied by a decrease in *E-cadherin* and an increase in *N-cadherin* and *vimentin* (11, 12).

A recent study indicated that PGG extracted from Bouea macrophylla significantly reduced the migration, invasion, and EMT abilities of MDA-MB231 cells. The mediation was driven by a decrease in *vimentin* and *β-catenin* expression and an increase in *E-cadherin*, as well as an inhibition of STAT3 phosphorylation. The compound also synergistically en-

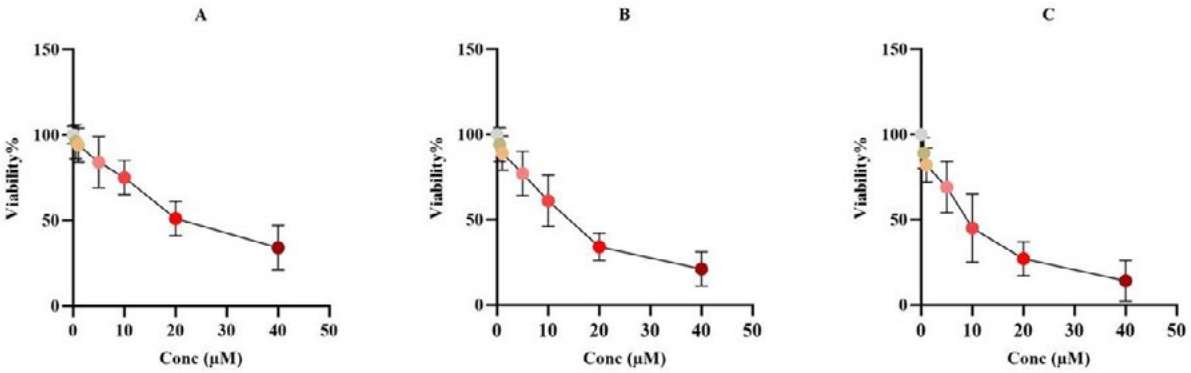


**Fig 1.** MTT assay to measure dose- and time-dependent inhibitory effects of PGG on U87-MG glioblastoma cell viability. Cells were treated with increasing concentrations of PGG (0.5–40 µM) for 24, 48, and 72 hours. Data are expressed as mean ± SD from three independent experiments. $p < 0.05$, $p < 0.01$, $p < 0.001$ versus control group.

hanced the effect of the drug doxorubicin (13). In a colon cancer model, PGG inhibited EMT and significantly reduced metastasis to the liver and lung by inhibiting the protease Cathepsin B and extracellular matrix-related pathways (FAK, cofilin). The expression of mesenchymal proteins was decreased, and epithelial markers were increased (14). PGG also inhibited cell migration and EMT in pancreatic cancer by suppressing stem cell factors (CD44v3, Nanog, and Sox-2) and inhibiting NF-κB and Foxo3 pathways (15).

The journal Biochemical and Biophysical Research Communications published research confirming the cytotoxic effects PGG has on glioblastoma cells, and as demonstrated with the data, PGG displayed the most significant level of inhibition on U251 glioma cells in comparison to other cell lines, including MDA-MB-231 and U87. The mechanism of this effect involves inhibition of the key enzyme of fatty acid synthesis (fatty acid synthase (FAS)), activation of the enzyme caspase-3, and induction of programmed cell death (apoptosis). Furthermore, at concentrations higher than 20 µM, PGG caused changes in the secondary structure of the FAS protein and its precipitation, indicating a strong inhibition of the function of this enzyme at the structural level. These findings indicate that PGG can act as an anti-glioblastoma agent not only through classical pathways such as inhibition of EMT and Wnt/β-catenin signaling but also through inhibition of lipid metabolism and induction of cell death (16).

Taken together, our data indicate that PGG may act as an anti-EMT and anti-proliferative agent to inhibit the growth of GBM cells. Since PGG was shown to enhance TMZ cytotoxicity through inhibition of EMT, it may offer a benefit when combined with TMZ in order to improve standard therapy effects. Additionally, due to the plant origin and distinct multi-mechanism activity of PGG, clinical investigation as a compound with low toxicity for use as an adjuvant treatment is also warranted.

**The limitations of this study are as follows:**

The study was performed only in an in vitro model with only the U87-MG cell line; therefore, results cannot necessarily be generalized to in vivo conditions or other cell lines, and further investigation is warranted. This study did not evaluate the impact of PGG on levels of the Wnt pathway or other associated signaling pathways. The decline of *Slug* gene expression, while a decline was observed, did not achieve statistical significance. This may be related to the duration of treatment and/or the sensitivity of the cell line.

## CONCLUSION

The findings of this study clearly demonstrate that the natural compound PGG was able to significantly limit the growth, survival, and migration of GBM cells by inhibiting the EMT, reducing the expression of mesenchymal genes, and suppressing the Wnt/β-catenin pathway. Also, the dose- and time-dependent cytotoxic effects and the ability to induce apoptosis in tumor cells strengthen the position of PGG as a potential therapeutic candidate or a complementary combination with standard drugs such as temozolomide. Due to the natural origin, low toxicity, and multimodality of this compound, we advocate further testing with in vivo models and clinical trials to establish its efficacy and safety. The available information outlines new opportunities to develop more selective and less harmful therapeutic options in glioblastoma treatment.

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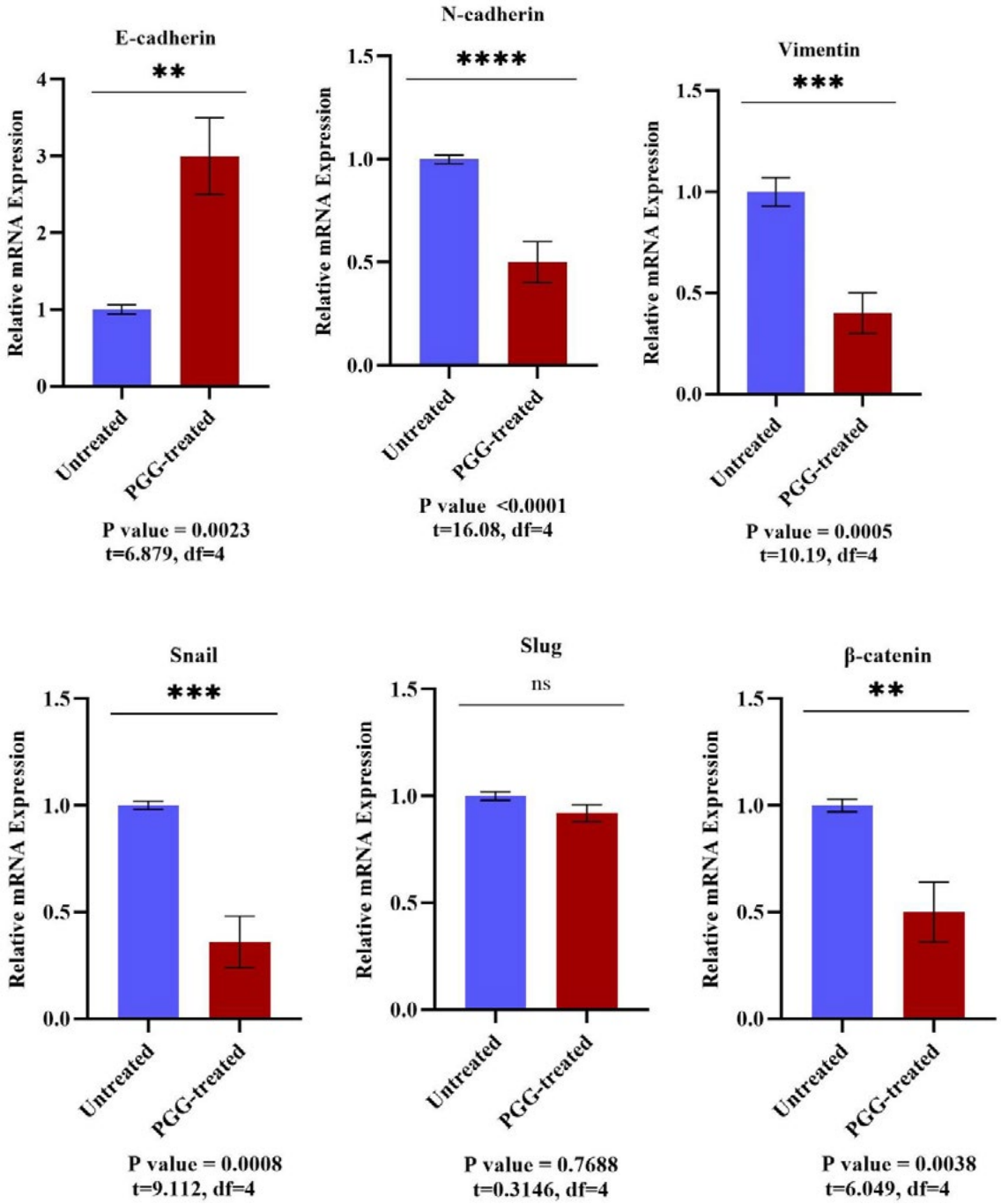


**Fig2.** The effect of PGG treatment on the expression of EMT-related genes and the level of β-catenin in U87-MG glioblastoma cells. The relative mRNA expression levels of E-cadherin, N-cadherin, Vimentin, Snail, Slug, and β-catenin were determined by qRT-PCR following 24 hours of treatment with PGG (10 μM). The relative expression levels are presented as fold change to the untreated control group and were normalised to GAPDH expression. PGG treatment significantly increased E-cadherin and decreased the mesenchymal markers (N-cadherin, Vimentin) and β-catenin. Snail expression was also significantly reduced, whereas the decrease in Slug was not statistically significant. Data represent mean ± SD of three independent experiments. $p < 0.05$, $p < 0.01$, $p < 0.001$ versus control group.

## Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

## Data Availability Statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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