

# *Interference with TRIM26 Expression on Proliferation of Glioma Cells*

**Rashar Almajed**

*American University in the Emirates, UAE*

**Keywords:** TRIM26, Glioma Cells, Cell Proliferation, Cell Migration

**Abstract:** Colloid tumor, as the most common malignant tumor in the brain, seriously endangers human life and health. TRIM 26 expression is closely related to the rapid growth and cell metastasis of most cancer cells. This article mainly studies the effect of interference with TRIM 26 expression on the proliferation of colloid tumor cells. Randomly selected 60 cases of colloid tumor specimens and the brain tissue of 10 cases of brain trauma patients as a normal control group. First, cell culture is performed to observe the growth status of the cells. Subsequently, the SiRNA interference technique was used to interfere with the expression of TRIM 26 on the liver cancer cell lines HepG 2 and Bel-720. The cells were collected for TRIM 26 protein westernBlot and fluorescence quantitative PCR to verify the knockout effect, and the cell proliferation, clone formation, transfer, and invasion experiments were performed. Experimental data showed that among 60 glioma patients, TRIM26 protein samples were positive in 46 cases, and 10 normal brain tissue samples were all negative. The performance of TRIM26 glioma was compared with the performance in normal brain tissue. The difference was significant and statistically significant ( $p < 0.01$ ). The results show that TRIM 26 can reduce the protein level of PBX1 through the correlation of time gradient and concentration gradient. TRIM 26 can reduce the mRNA level of RNF6, the downstream target gene of PBX1, without affecting the mRNA level of PBX1.

## **1. Introduction**

In the clinical treatment of glioma, surgery, radiotherapy, chemotherapy and other methods are used, but the treatment effect is relatively low, most patients with tumor recurrence in a short period of time, most of the patients with high malignant tumor survival time less than 1 year. Therefore, improving the therapeutic effect of glioma is the focus of clinical and scientific research in neurosurgery community. It plays an important role in the progression of respiratory diseases, HIV-1 infection and tolerance to aspirin syndrome disorder.

The surgical resection of colloidal tumors and postoperative chemotherapy have not completely

suppressed the occurrence of colloidal tumors. The recurrence rate of brain colloidal tumors is still very high, and the prognosis is not optimistic. TRIM 26 shares the physical location with PBX1 and is located in the core. TRIM26 combines SPRY structure region and PBX1, decomposes PBX1, inhibits its transcriptional activity, and promotes the proliferation of glioma cells.

In tumor necrosis factor-related apoptosis-inducing ligand (TRAIL) resistant glioma cells, TRAIL combined with sub-toxic dose of rottlerin treatment can induce rapid apoptosis. Eun believes that the proteolytic process of procaspase-3 by TRAIL is partially blocked in these cells, and treatment with rottlerin can effectively restore TRAIL-induced caspase activation. He down-regulated cyclin A, cyclin B and Cdc2 protein, and treated with rottlerin can significantly reduce Cdc2 activity, while rottlerin's sensitizing effect on TRAIL-induced apoptosis was not related to PKCdelta activity. In addition, he treated rottlerin down-regulated the protein levels of two major caspase inhibitors, survivin and X-linked IAP (XIAP). The forced expression of Cdc2 and cyclin B mediated down-regulation through the down-regulation of survivin and XIAP protein levels, thereby attenuating rottlerin-enhanced TRAIL-induced apoptosis [1]. His research process lacks experimental control, and research has no practical value.

Lijuan tested the effects of tanshinone IIA (Tan IIA) on the cell viability, cycle, apoptosis and autophagy of human glioma cell U251. Tan IIA and PI3K agonists (740 Y-P) are used to treat glioma cell U251. He found that Tan IIA reduced the expression of p-PI3K and p-Akt proteins, inhibited cell viability and promoted apoptosis. At the same time, Bax expression increased while Bcl-2 expression decreased. In addition, Tan IIA promoted autophagy in U251 glioma cells and increased the expression of LC3B and Beclin 1. However, 740Y-P plays an opposite role in the cell viability, cycle, apoptosis and autophagy of U251 cells [2]. His research did not determine how long the cells were active.

Zhu believes that the occurrence of inherent or acquired resistance to temozolomide (TMZ) is the main burden of glioma patients. He found that microRNA plays an important role in the regulation of cancer tumor characteristics. However, it is not fully understood whether miR-497 contributes to the resistance of glioma to chemotherapy. In this study, he found that miR-497 expression was significantly up-regulated in TMZ-resistant glioma cells. The high expression level of miR-497 is related to the TMZ resistance phenotype of glioma cells. The down-regulation of miR-497 in glioma cells enhanced the apoptosis induction and growth inhibition of TMZ in vitro and in vivo, while promoting miR-497 promoted the chemical sensitivity of glioma cells to TMZ. Simultaneous upregulation of miR-IRS1 pathway-related proteins (ie IGF1R, IRS1) in TMZ-resistant glioma cells, mammalian targets of rapamycin (mTOR) and Bcl-2. In addition, knockdown of mTOR and Bcl-2 reduced the tolerance of glioma cells to TMZ [3]. His research sample data is insufficient and the experiment is incomplete.

The innovation of this paper is to interfere with the expression of TRIM26 by siRNA transfection, and carry out cell proliferation, infiltration, transfer and cloning. These have important significance and clinical value for the future theoretical research and clinical individual treatment guidance of glioma.

## 2. TRIM26 and Glioma Cell Proliferation

### 2.1. TRIM26

TRIM 26 is a member of the triple coupled protein (Tripartite motif protein TRIM) pan-coupling enzyme family. It has been found that its biological functions are mainly concentrated in two aspects. TRIM 26 mediates the degradation of IF3 through the polymerization of K48, and further inhibits the production of IFN- $\beta$ , thereby inhibiting the antiviral response. Under the interference of the RNA virus, TRIM 26 binds to the NEMO protein after generalization, and further binds the

NEMO protein to the TBL to activate it, thereby having an effect on the RNA virus. In liver cancer, the expression of TRIM 26 is lower in cancer tissues and is related to the poor prognosis of liver cancer patients. Low TRIM 26 can promote the proliferation, invasion and metastasis of liver cancer cells. In breast cancer cells, short-term TGF- $\beta$  stimulation induces TRIM 26 transcription, which further mediates the degradation of TAF7 protein, thereby producing an inhibitory effect on the tumor [4-5].

These three domain family proteins have evolutionary and conserved structural regions and are considered to be important coordinators in cell biology. There are three N-terminal structural regions in TRIM members, including three zinc-binding structural regions, a RING structural region, a B-box 1 and B-box 2 and a coiled structural region. Therefore, it has many characteristics of opsin E3 universal ligase. TRIM protein affects cell proliferation, differentiation, non-specific immunity, cell death, cell migration, etc. [6]. For example, TRIM27 and TRIM 24 are previous oncogenes, while TRIM 16 can block the cell cycle and inhibit the growth of nerve cell aneurysms. TRIM 28 has anti-proliferative activity by inhibiting the transcription factors of the E2F family in lung cancer. In gastrointestinal tumors, TRIM 40 prevents cancer progression by promoting synaptic nuclear factor B kinase subgroup gamma inhibitors (IK $\gamma$ ). In colon cancer, TRIM 25 is highly expressed in colon cancer tissues, while TRIM 25 is exogenously expressed in colon cancer cells. Transforming growth factor (TGF- $\beta$ ) signaling pathway regulates the growth and invasion of colon cancer cells, and its growth and migration rate is more than twice that of control cells [7].

The mechanism of action of TRIM26 in cells is expressed by the following formula:

$$\min_G \max_D E_{x \sim P_{data}} \log[D(x)] + E_{Z \sim P_Z} \log[1 - D(G(z))] \quad (1)$$

In the formula,  $\min_G \max_D$  represents the number of cells, and  $\log[D(x)]$  represents the rate of action of TRIM26.

The survival rate of glioma cell proliferation is calculated by the following formula.

$$J(\theta) = E[R_T | S_{0,\theta}] = \sum_{y \in Y} G_\theta(y_{||S_0}) \bullet Q_{D_\phi}^{G_\theta}(S_0, y_1) \quad (2)$$

Among them,  $J(\theta)$  is the survival rate of glioma.

The cell growth cycle is as follows:

$$\min_\phi - E_{Y \sim P_{data}} [\log D_\phi(Y)] - E_{Y \sim G_\theta} [\log(1 - D_\phi(Y))] \quad (3)$$

The cell proliferation activity is as follows:

$$\Delta_\theta J(\theta) = EY_{1:t-1} \sim G_\theta \left[ \sum_{y_t \in Y} \Delta_\theta G_\theta(y_t | Y_{1:t-1}) \bullet Q_{D_\phi}^{G_\theta}(y_t, Y_{1:t-1}) \right] \quad (4)$$

TNFRSF6B is a polypeptide composed of 300 amino acids, including a guide sequence at the end of the amino acid, followed by four functional regions rich in cysteine. Because TNFRSF6 can secrete proteins, it lacks a clear transmembrane sequence. Compared with OPG, all four cysteine residues in the TNFRSF6B CDR are conserved and lack 101 residues at the carboxyl terminus. The molecular weight of TNFRSF6B is 35KD, which appears on the acrylic phthalocyanine gel electrophoresis plate. TRIM 26 belongs to the C-IV category of the TRIM series [8-9]. In the protein structure, the TRIM family has a characteristic RBC (RJNG, Bbox and helix domain) structure at the N-terminus, and the PRY-SCPRY structure in which the substrate protein and substrate are combined is specific at the C-terminus. TRIM 26 cleaves the target into proteasomes, which are formed by the multiplication of substrate protein specifically mediated by pancreas ligase

E3. TRIM 26 mediates the formation of the K48-linked polyprimary chain of NEIL1, which is related to the repair process of DNA damage, because mass spectrometry indicates that TRIM 26 is a universal ligase for NEIL1, and NEIL1 may be degraded at this time [10].

## 2.2. Glioma Cell Proliferation

Glioma is a malignant tumor of the central nervous system. It is produced by nerve interstitial cells and is divided into astrocytoma, myeloma, ependymoma, and pineal tumor. The main symptom of the patient is neurological deficit caused by headache, infiltration and brain tissue destruction caused by hypercranial pressure [11].

GOLPH3 is also closely related to the glycosylation modification of proteins. The secreted protein formed by the small cell body enters from the front of the Golgi body and is transported between the membrane sacs [12-13]. A series of orderly processing and modification takes place, and most of the original sugar chain Mannos is excised. However, various types of sugars were added in the order of various carbohydrate aminotransferases, resulting in specific carbohydrate aminotransferase modifications. GOLPH3 is attached to the N-terminus of ribose isoflavone kinase exposed on the cytoplasm. These lipases are fixed to the GOJI body along the mesenteric sac to regulate the processing and modification of carbohydrate proteins. The reduction of GOLPH3 may affect the fixation of pectin glycosylaminotransferase, forming low-sterol secreted protein [14]. Changes in glycosylation of proteins are usually associated with tumor formation, which may cause changes in cell proliferation, adhesion, invasiveness, and immune cognition, as well as changes in intracellular signal transmission. Sterolification plays an important role in the transmission of membrane penetration signals caused by growth factors. Moreover, sterolization modification may also change the activity of membrane receptors and affect the reaction with corresponding ligands. The glyceride modification fixes EGFR on the intact membrane and protects the terminal nervous system of the cell, thereby increasing the role of growth factors.

As an important molecule in the cell,  $\beta$ -catechin has many functions closely related to the intracellular distribution.  $\beta$ -catechin is distributed in cell membrane, nucleus and cytoplasm. On the cell membrane,  $\beta$ -catechin and E-cadmium hailin form a complex and participate in cell adhesion;  $\beta$ -catechin in the nucleus participates in intracellular signal transmission. Under normal circumstances,  $\beta$ -catechin in the cytoplasm is phosphorylated by GSK-3 $\beta$  and decomposed by pantothenic acid, so the concentration of  $\beta$ -catechin in the cytoplasm is kept low. In some cases such as Wnt signal activation, the function of GSK-3 $\beta$  is blocked,  $\beta$ -catechin in the cytoplasm cannot be phosphorylated, and the concentration of  $\beta$ -catechin in the cytoplasm increases [15].

The reason for the failure of colloid tumor treatment is relapse. Postoperative radiotherapy and chemotherapy are common treatments. However, radiation tolerance of tumor cells can cause recurrence of residual lesions in radiation therapy. Radiation therapy after surgery allows the survival time of patients with colloid varicose veins to be extended, but the proportion of local recurrence is still high. As the dose increases, the patient's cognitive dysfunction will continue to increase. Chemotherapy plays an important role in killing residual tumor cells. It is an important adjunct therapy for the treatment of brain colloid tumors, which can easily cause severe liver damage and bone marrow suppression.

## 2.3. Effect of TRIM26 Expression on Glioma Cell Proliferation

Viral infection induces the emergence of TRIM26, which acts as a mechanism to avoid the natural immune system by regulating IRF3 and interferon. In addition, the transfer group TRIM26 can directly decompose NEIL1 into viscose in vitro, and then adjust the protein level of NEIL1 in vitro. The lack of TRIM26 greatly increases the resistance to IR cells. This is partly due to the

increase in NEIL1 protein levels. This indicates that TRIM26 can play multiple roles. Some TRIM families target antiviral immunity, and may also be involved in the regulation of the main BER enzyme in the repair of cellular DNA damage. As a new tumor suppressor of liver cancer, TRIM26 is significantly reduced in liver cancer compared with normal liver tissue, and is related to the malignancy of liver cancer. Moreover, TRIM26 can promote the proliferation and metastasis of HCC cells. In HEK293 cells, TRIM26 interacts with TRAF2 through the optical coil domain. This plays an important role in regulating cell activation and tumor death [16].

### 3. Experiment of the Effect of TRIM26 on the Proliferation of Glioma Cells

#### 3.1. Specimen Collection

60 cases of colloidal aneurysms were randomly collected, including 15 cases of degree II neuropathy, 20 cases of degree III neuropathy, and 25 cases of degree IV neuropathy. After surgical resection, each patient's specimen is divided into two parts. Some samples were fixed in 10% formaldehyde solution. The remaining specimens are placed in sterile centrifuge tubes. Liquid nitrogen is kept frozen. Brain tissue was randomly selected from 10 brain trauma patients as a normal control group.

#### 3.2. Experimental Instruments and Main Reagents

The main instruments and reagents used in the experiment are shown in Table 1 and Table 2.

*Table 1. Main instruments*

Serial Number	Equipment Name	Manufacturer
1	Ultra-Clean Workbench Hf Safe 1200	Germany Heraeus and Lishen
2	CO2 Constant Humidity Incubator Heraeus Bb 16uv	Germany Heraeus and Lishen
3	Inverted Phase Contrast Microscope Olympus Ckx4	Olympus Corporation of Japan
4	Small Desktop Centrifuge 1-14	American Sigma
5	Electronic Balance Cpa2245	Germany Sartorius
6	Liquid Nitrogen Container Yds-35-1	Leshan Dongya Electromechanical Industry and Trade Co Ltd
7	Ph Meter Phs-3c	Shanghai Jingke Company
8	Small Desktop Multifunctional Oscillator Ika-Ms 3	Germany IKA
9	Micro-Adjustable Pipette (10, 100, 1000µl)	American Gilson Corporation
10	Microplate Reader Infitem200	American Tecan Company

*Table 2. Main reagents*

Serial Number	Reagent Name	Manufacturer
1	BCATM Protein Kit	American Pierce Company
2	Rabbit Anti-Human Hsp90 Antibody	British Abeam
3	HRP Anti-Rabbit Secondary Antibody	British Abeam
4	RNA Reverse Transcription Kit	Toyobo
5	BCA Protein Concentration Detection Kit	Pierce
6	PMSF	Roche
7	Cocktail	Roche

### 3.3. Preparation of Reagents

(1) Mixing of GDNF: 1 mg BSA was added into 1 ml PBS with 0.1% concentration, and then 10ug GDNF was added to mix into 10ug/ul GDNF mother liquor. After filtration and sterilization, it was frozen and stored in a refrigerator at -20°C. When used, it can be diluted with 0.1% BSA according to the required concentration.

(2) The molecular weight of the 0.1 mg/ml polylysine acid solution (mother liquor) is 30,000 to 70,000. Dissolve 10 mg of polylysine in 100 ml of ultrapure water and use a 0.22 µm caliber disposable filter Bacteria, store the prepared solution in the refrigerator at -20°C.

### 3.4. Cell Culture

(1) Preparation of culture medium:

Add 10% volume of fetal bovine serum and 1% penicillin strep antibiotic solution (100X) to the high glucose DEM medium, mix and store.

(2) Cell recovery:

Remove the frozen cells from the liquid nitrogen tank and pinch the cells with tweezers, taking care to avoid frostbite. Immediately place the frozen storage tube in a 37 °C water bath and shake it constantly. Thaw the cryopreservation solution into the cryopreservation tube in about 1 minute. Under sterile conditions, absorb the ice and store the liquid. After discarding the liquid above, you can see the sedimented cells in the heart. Blow dry and mix the cells evenly, add a small amount of medium to allow them to settle, then transfer to a cell culture flask and gently blow into the culture flask to mix. Transfer to 37 °C and incubate in 5% CO<sub>2</sub> environment. The next day, change the medium and continue to grow, and observe the growth.

### 3.5. Immunohistochemistry

(1) Insert the tissue slice into the slice frame, add distilled water to the washing box, place it on the shaker 3 times at a rate of 3 times/minute, discard the distilled water, add PBS buffer, and then place it on the shaker. Leave and wash 3 times for 3 minutes each time.

(2) Insert the tissue slice into the slice box, put it into a washing box containing PBS buffer memory, put it into a blender, and wash for 3 minutes.

(3) Tilt the remaining PBS buffer in the tissue section to suspend the undiluted sheep serum solution on the whole tissue, seal at room temperature for 20 minutes, and do not wash if tilted.

(4) Using the DAB developer kit, collect 1 ml of distilled water, add A, B, and C reagents to the kit one drop at a time, add 100 ul to the slice, and then add the microscope to the microscope. Control hair color for 1-3 minutes. When the color is light, add 0.01 MPBS to stop the reaction, and immediately wash the distilled water.

(5) Rinse tap water and re-stain hematoxylin for 1 minute, wash with ethanol for 3 to 5 seconds, and then rinse with tap water. After rinsing, the ammonia water turned blue and after rinsing for 10 seconds, the gradient alcohol was dehydrated and dried.

### 3.6. Statistical Analysis and Data Statistics

The results of RT-PCR and Western Blot were collected, processed and analyzed with Image J software, and the relevant data was statistically analyzed with SPSS16.0.  $p < 0.05$  is statistically significant. The MTT method was used to compare the cell absorption photometric values of the target group and the control group, and the floating control method was used to detect the mortality of the target group and the control group by unidirectional dispersion analysis.



#### 4. Effect of TRIM26 on the Proliferation of Glioma Cells

##### 4.1. Expression Analysis of TRIM26 in Immunohistochemical Staining

After immunohistochemical staining, the expression of TRIM26 in the control group and glioma group is shown in Figure 1 and Table 3. It can be seen that there was only one person with higher expression of TRIM26 in the control group, while 7 people with higher expression of glioma tertiary grade were significantly higher than the control group. The expression of TRIM26 in glioma tissue is mainly concentrated in the cytoplasm of tumor cells, but no expression in the nucleus. Cationic liposome method is the latest method in liposome-mediated transfection method, and is one of the non-viral transfection methods with the highest transfection efficiency and the least toxicity. This method obtains a high transfection efficiency that was previously unavailable in a variety of eukaryotic cell types, and it is easy to use and guarantees reproducible and consistent results. It is used for cell lines for protein expression (such as COS-7, CHO And HEK293), Lipofectamine2000 transfection reagent is particularly effective, the efficiency can exceed 90%. When the cationic liposome reagent is added to water under optimized conditions, it can form tiny (average size about 100-400 nm) monolayer liposomes. Compared with other methods, negatively charged DNA automatically binds to positively charged liposomes to form DNA cationic liposome complexes. These complexes are positively charged and can be bound to the surface of negatively charged cell membranes by electrostatic action, and the captured DNA will be introduced into the cultured cells. Liposome-mediated transfection can be used to establish cell lines containing DNA that stably integrates the target DNA. Tumor cells have a strong proliferative ability, which is reflected in the increase of cells in the division phase in the cell cycle. After knocking out oncogenes, tumor cells can reduce cell division and reduce cell proliferation capacity.

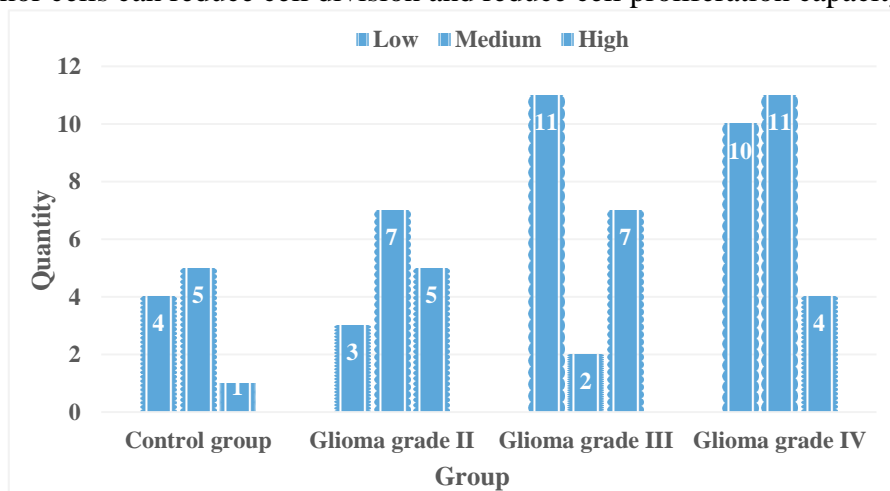


Figure 1. Immunohistochemical staining TRIM26 expression results

Table 3. TRIM26 immunohistochemical staining results

Group	Low	Medium	High
Control group	4	5	1
Glioma grade II	3	7	5
Glioma grade III	11	2	7
Glioma grade IV	10	11	4

The expression of TRIM26 in gliomas and normal brain tissue was detected. The statistical results are shown in Figure 2. Of the 60 glioma samples, 46 cases were positive for TRIM26 protein; all 10 cases of normal brain tissue samples were negative expression; comparing the expression of TRIM26 in glioma with the expression in normal brain tissue, the difference was significant and statistically significant ( $p < 0.01$ ). In human cells, there are many tumor suppressor genes, such as p53, which is a protein with a half-life of about 30 minutes. If normal cells are damaged, p53 will become more stable after such damage, and then the entire cell will die. There is a great correlation between DNA damage and the corresponding increase in p53. The final response level of p53 mainly depends on the actual nature of the entire damage. Studies have also shown that this is related to the corresponding p53 caused by the entire ubiquitin-proteasome system. The degradation ability is related to down regulation. Another series of studies suggest that if there is a heat-resistant E1 genotype in the cell line, p53 accumulation will occur, and once the E1 gene is transferred, the accumulation of p53 will be blocked. Taking human herpes virus HPV as an example, the E6 oncoprotein of high-risk HPV virus cells binds to p53 through E6AP and promotes the degradation of p53 through the ubiquitinated protease pathway. On the contrary, many viruses, such as low-risk viruses, cannot be combined with p53 and cannot be degraded. Under the action of E2 and E3 enzymes, the combination of ubiquitin and E6AP is mainly able to produce the corresponding thiol ester complex, and this substance has the function and effect of activating enzyme, so that p53 is finally compounded by the protease 26S body degradation.

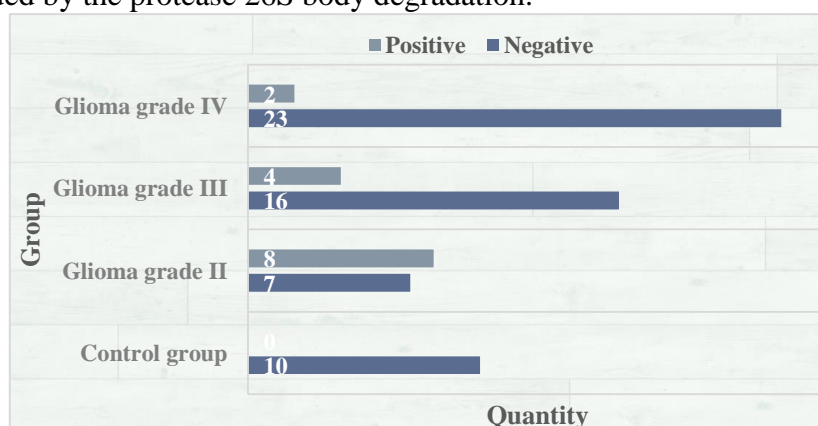


Figure 2. Statistical results

#### 4.2. Relationship between TRIM26 Expression Level and Clinical Parameters of Glioma

A total of 60 cases of glioma patients were collected with complete clinical data. The statistical results are shown in Figure 3. Correlation analysis of TRIM26 expression level and clinicopathological characteristics (tumor size, tumor site, tumor grade, gender, age) found that TRIM26 expression level was correlated with tumor size ( $p < 0.01$ ,  $r = 0.391$ ), and tumor site, Tumor grade, gender, and age were not statistically different ( $p > 0.05$ ). Due to the infiltrative growth of gliomas, the particularity and functionality of brain tissue, it is difficult to cure it with surgery, radiotherapy and chemotherapy, and the recurrence rate is extremely high. The growth rate and anti-apoptosis ability of glioma determine its progress. Therefore, the research on the growth and apoptosis of glioma is of great significance for its treatment. Through the detoxification of the carbonyl group by the cell, it can regulate the balance and steady state of retinoic acid and regulate the synthesis of fatty acid and lipid metabolism, affect the proliferation, invasion and metastasis of tumors, and are closely related to the occurrence and development of tumors. TRIM26 is mainly expressed in non-small cell lung cancer (pulmonary squamous cell carcinoma and adenocarcinoma),



and TRIM26 overexpression in lung squamous cell carcinoma is closely related to smoking. TRIM26 activates carcinogens such as polycyclic aromatic hydrocarbons (PAH) in tobacco Biological precursor, leading to lung tumors. In addition, TRIM26 promotes tumorigenesis in breast cancer and colon cancer by regulating lipid anabolism, protects tumor cells and regulates tumorigenesis by detoxifying carbonyl groups.

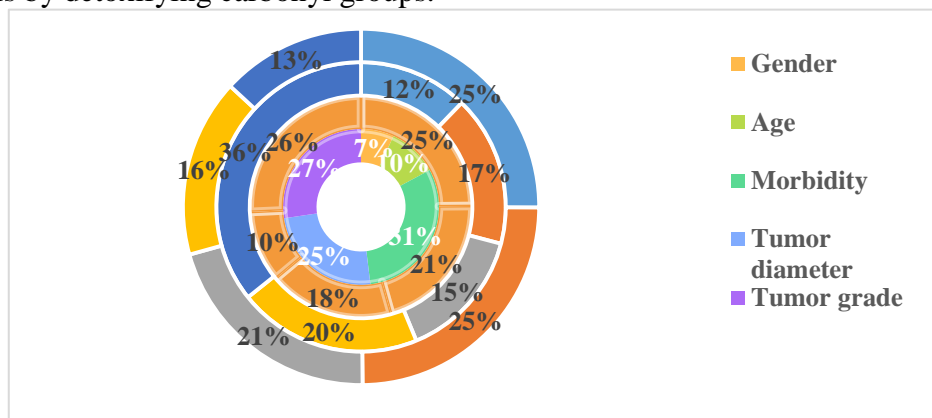


Figure 3. Statistical results of patients' clinical data

In cell growth, many cell growth-related proteins are the target substrates of ubiquitinated proteasome complexes, such as rapid and periodic B-cell degradation. In the study of cancer cells, it can be found that mainly when the entire cell cycle exits the rapid division period, Cell cycleB, E and D1 basically show a certain overexpression in the process of cell carcinogenesis. Moreover, Cell cycleB, E and D1 are also substrates of the proteasome complex after ubiquitination throughout the cell growth cycle. Their overexpression is caused by the decrease of ubiquitin-proteasome activity.

#### 4.3. Role of TRIM26 in the Proliferation of Glioma Cells

The expression of TRIM26 in glioma cell proliferation is shown in Figure 4. The free amino and sulfhydryl groups in proteins and amino acids can form covalently modified compounds with carbonyl groups, that is, the cross-linking reaction of carbon and ammonia, which results in the inability of the protein to hydrolyze and lose its original function, leading to the disorder of cell structure and function; the protein modified by carbonyl group can be aggregated it can form proteasome resistance, further prevent abnormal protein degradation, and may cause tumors and other diseases through a series of biochemical reactions. Carbonyl groups can also mutate and damage DNA, leading to disease. HE staining is mainly used to evaluate the heterogeneity of CHG-5 and U87 glioma cells. The expression level of TRIM26 is an important indicator of the degree of differentiation of glioma cells, and the degree of differentiation is inversely related to the degree of malignancy of tumor cells. The expression level of TRIM26 is an important indicator that reflects the malignant progress of gliomas. The expression level of TRIM26 is positively correlated with the malignant degree of gliomas. The glioma cells with 1p/19q chromosome combined deletion are highly sensitive to PCV chemotherapy, and the prognosis of these patients is better. In a further study of clinical glioma samples, it was found that TMZ simultaneous chemoradiotherapy and subsequent adjuvant therapy for patients with MGMT promoter methylation can significantly improve the patient's prognosis and prolong the survival of patients, while the MGMT promoter is not After methylated patients received the same condition of treatment, their prognostic survival level was significantly lower than that of patients with MGMT promoter methylation. KLF6 is an

important regulatory pathway of MEF2D in hippocampal neurons. Down-regulation of KLF6 expression can promote the death of neurons, and promote MEF2D to promote the apoptosis of hippocampal neurons through negative regulation. In addition, MEF2 can regulate the expression of various genes in the human brain. MEF2 plays an important role in the regulation and development of the human nervous system. Among them, epilepsy, autism, mental retardation and other diseases are related to mutations of MEF2 factors and MEF2 target genes. In the case of insufficient MEF2C single dose, severe mental retardation can be formed, and when certain genes regulated by MEF2 are mutated, it can lead to imbalance of synaptic excitation and induce epilepsy. In the lung cancer cell line A549 with low expression of TRIM26, overexpression of TRIM26 will degrade the protein level of PBX1 and promote the proliferation of A549 cells, while knocking down TRIM26 in lung cancer cell line H226 with high expression of TRIM26 will upregulate the protein level of PBX1 and inhibit the proliferation of H226 cells. Furthermore, after knocking down TRIM26 in lung cancer cell line H226 with high expression of TRIM26 and overexpressing TRIM26 in lung cancer cell line A549 with low expression of TRIM26, clone formation experiments were conducted. Experimental results show that knocking down TRIM26 inhibits the formation of H226 cell clones, and overexpressing TRIM26 can promote the formation of A549 cell clones. In summary, the high expression of TRIM26 in lung cancer tissue is related to the low expression of PBX1. TRIM26 promotes the proliferation of non-small cell lung cancer cells by inducing the degradation of PBX1.

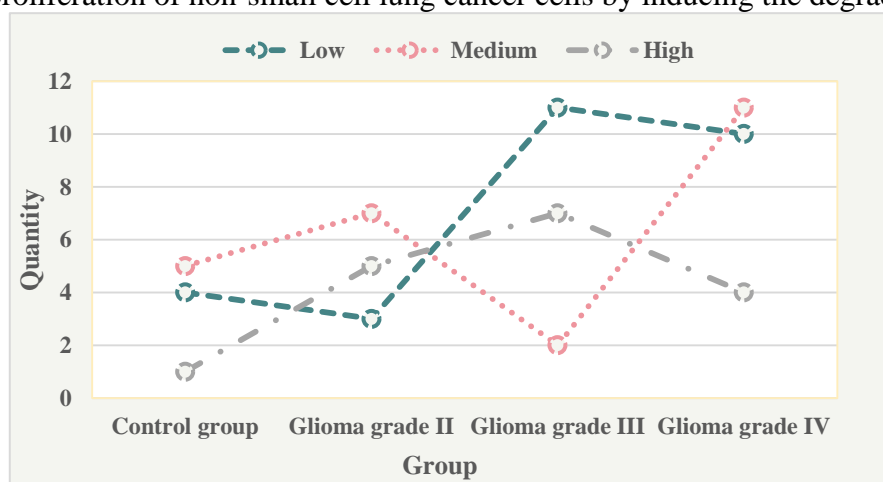


Figure 4. Expression of TRIM26 in glioma cell proliferation

TRIM26 can inhibit proliferation and promote apoptosis of glioma cells. However, glioma U251 and U87 cells are PTEN-deficient cell lines. Therefore, the mechanism of TRIM26 exerting tumor suppressive effect in glioma cells remains to be explored. NPP4B is a member of the inositol polyphosphate phosphatase family, about 105KD, has two subtypes, appearing at 514bp and 395bp, it has a recognized phosphatase catalytic site and a highly conserved C2 structural site, Its structure is highly similar in rodents and humans, and is expressed in brain tissue and many other tissues. TRIM26 phosphorylates the 336 tyrosine site of PTEN to maintain the stability of PTEN and play a role in cancer suppression. NPP4B has a specific phosphorylation site. Therefore, in gliomas, TRIM26 may play a role in suppressing cancer through INPP4B. Wnt plays an important role in various systems of the body, and is an important way to regulate the growth and development of cancer cells. Therefore, it is reasonable to speculate that TRIM26 may affect the function of Wnt signaling and cause cancer-related activities. The Wnt gene is a proto-oncogene. The Wnt egg self-sufficient secreted glycoprotein encodes a hydrophobic signal sequence, followed by several glycosylation sites, and is rich in cysteine. Knocking out TRIM26 can directly increase cell proliferation and promote cell transfer. Bioinformatics analysis indicates that TRIM26 may be an

important regulator of cancer cell metabolism.

## 5. Conclusion

This article mainly studies the effect of the interference of TRIM 26 expression on the growth of colloidal tumor cells. Through immunoprecipitation experiments, it was found that the C-terminal SPRY structure region of TRIM 26 bound to PBX1. After analyzing the patient samples, we found that the expression of TRIM 26 mRNA in glioma tissue was significantly higher than that in cancer tissue, which proved that TRIM 26 can actually inhibit the transcription activity of PBX 1. The expression of TRIM 26 and IF3 protein in nscic was negatively correlated. Co-immunoprecipitation studies were conducted by rotating TRIM 26 and IF3 particles in H299 cells, and it was found that TRIM 26 and IF3 interacted.

The essence of cell carcinogenesis is the infinite proliferation of cells due to protein dysfunction related to the redirection of cell growth signals. Molecular targeted therapy has become one of the current hot spots in neuroma research. Molecular targeted therapy is a treatment method that regulates interference at the target level, such as tumor cell receptors, cell transduction pathways, regulatory factors and essential genes at the molecular level.

Many members of the TRIM family target various substances such as cell cycle regulators, tumors or tumor suppressors, and play an important role as regulators in cancer. The discovery of TRIM26 hindered the proliferation of H11299 cells and promoted cell death. In addition, TRIM26 hindered the migration of H11299 cells and A549 cells, so it was confirmed that TRIM26 plays a role in promoting carcinogenesis in nerve cells. TRIM26 affects the stability of IFRF3 through ubiquitination and may reduce the amount of PBX1 bound to other factors.

## Funding

This article is not supported by any foundation.

## Data Availability

Data sharing is not applicable to this article as no new data were created or analysed in this study.

## Conflict of Interest

The author states that this article has no conflict of interest.

## References

- [1] Eun Hee Kim, Seung U Kim, & Kyeong Sook Choi. (2016). "Rottlerin Sensitizes Glioma Cells to Trail-Induced Apoptosis by Inhibition of Cdc2 and the Subsequent Downregulation of Survivin and Xiap", *Oncogene*, 24(5), pp.838. <https://doi.org/10.1038/sj.onc.1208241>
- [2] Lijuan Ding, Shudong Wang, Weiyao Wang, Peng Lv, Donghai Zhao, & Feier Chen. (2016). "Tanshinone Iia Affects Autophagy and Apoptosis of Glioma Cells by Inhibiting Phosphatidylinositol 3-Kinase/Akt/Mammalian Target of Rapamycin Signaling Pathway", *Pharmacology*, 99(1-2), pp.99. <https://doi.org/10.1159/000452340>
- [3] Zhu, D., Tu, M., Zeng, B., Cai, L., Zheng, W., & Su, Z., et al. (2017). "Up - Regulation of Mir - 497 Confers Resistance to Temozolomide in Human Glioma Cells by Targeting Mtor/Bcl - 2", *Cancer Medicine*, 6(2), pp.452-462. <https://doi.org/10.1002/cam4.987>

- [4] Song, H., Zhang, Y., Liu, N., Wan, C., Zhang, D., & Zhao, S., et al. (2016). "Mir-92b Regulates Glioma Cells Proliferation, Migration, Invasion, and Apoptosis Via Pten/Akt Signaling Pathway", *Journal of Physiology & Biochemistry*, 72(2), pp.201-211. <https://doi.org/10.1007/s13105-016-0470-z>
- [5] Zhi-Qiang, X., Teng-Kun, Y., Ya-Xing, L., Jian-He, Z., & Jian-Jun, G. (2017). "Mir-130b Regulates the Proliferation, Invasion and Apoptosis of Glioma Cells Via Targeting of Cyld", *Oncology Reports*, 38(1), pp.167-174. <https://doi.org/10.3892/or.2017.5651>
- [6] Tang, J.H., Ma, Z.X., Huang, G.H., Xu, Q.F., Xiang, Y., & Li, N., et al. (2016). "Downregulation of Hif-1 $\alpha$  Sensitizes u251 Glioma Cells to the Temozolomide (Tmz) Treatment", *Experimental Cell Research*, 343(2), pp.148-158. <https://doi.org/10.1016/j.yexcr.2016.04.011>
- [7] Qiao, D., Meyer, K., & Friedl, A. (2016). "Retraction for Qiao et al. Glypican 1 Stimulates s Phase Entry and Dna Replication in Human Glioma Cells and Normal Astrocytes", *Molecular & Cellular Biology*, 36(15), pp.2105. <https://doi.org/10.1128/MCB.00321-16>
- [8] Mohammed I. Alghamdi, Neutrosophic set with Adaptive Neuro-Fuzzy Inference System for Liver Tumor Segmentation and Classification Model, *International Journal of Neutrosophic Science*, 2022, Vol. 18, No. 2, pp: 174-185 (Do i: <https://doi.org/10.54216/IJNS.180202> ) <https://doi.org/10.54216/IJNS.180202>
- [9] Qin, Q., Yang, X., Zhang, R., Liu, M., and Ma, Y. (2022). An Application of Deep Belief Networks in Early Warning for Cerebrovascular Disease Risk. *Journal of Organizational and End User Computing*(forthcoming). <https://doi.org/10.4018/JOEUC.287574>
- [10] Antonietti, P., Linder, B., Hehlhans, S., Mildemberger, I.C., & Kogel, D. (2016). "Interference with the Hsf1/Hsp70/Bag3 Pathway Primes Glioma Cells to Matrix Detachment and Bh3 Mimetic-Induced Apoptosis", *Molecular Cancer Therapeutics*, 16(1), pp.156. <https://doi.org/10.1158/1535-7163.MCT-16-0262>
- [11] Liyang, G., Bing, C., Jinhong, L., Fan, Y., Xuecheng, C., & Zhuangbing, L., et al. (2017). "Wnt/ $\beta$ -Catenin Signaling Pathway Inhibits the Proliferation and Apoptosis of u87 Glioma Cells Via Different Mechanisms", *Plos One*, 12(8), pp.e0181346. <https://doi.org/10.1371/journal.pone.0181346>
- [12] Ngan Thi Roan, Florentin Smarandache, H-Max Single-Valued Neutrosophic Distance Measure in Medical Diagnosis, *International Journal of Neutrosophic Science*, 2021, Vol. 15, No. 2, pp: 62-76 (Doi : <https://doi.org/10.54216/IJNS.150201> )
- [13] M. Krichen, S. Mechti, R. Alrooba, E. Said, P. Singh et al. (2021) "A formal testing model for operating room control system using internet of things," *Computers, Materials & Continua*, vol. 66, no.3, pp. 2997-3011. <https://doi.org/10.32604/cmc.2021.014090>
- [14] Guangzhi, W., Mingna, L., Hongjun, W., Shan, Y., Zhenfeng, J., & Jiahang, S., et al. (2016). "Centrosomal Protein of 55 Regulates Glucose Metabolism, Proliferation and Apoptosis of Glioma Cells Via the Akt/Mtor Signaling Pathway", *Journal of Cancer*, 7(11), pp.1431-1440. <https://doi.org/10.7150/jca.15497>
- [15] Nitta, Y., Shimizu, S., Shishido-Hara, Y., Suzuki, K., Shiokawa, Y., & Nagane, M. (2016). "Nimotuzumab Enhances Temozolomide -Induced Growth Suppression of Glioma Cells Expressing Mutant Egfr in Vivo", *Cancer Medicine*, 5(3), pp.486-499. <https://doi.org/10.1002/cam4.614>
- [16] Lu, T., Bao, Z., Wang, Y., Yang, L., & Cui, G. (2016). "Karyopherin $\beta$ 1 Regulates Proliferation of Human Glioma Cells Via Wnt/ $\beta$ -Catenin Pathway", *Biochemical & Biophysical Research Communications*, 478(3), pp.1189-1197. <https://doi.org/10.1016/j.bbrc.2016.08.093>