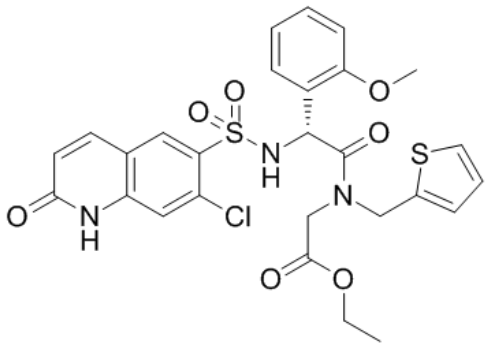
Product Data Sheet

Product Name: OSMI-4
Cat. No.: GC31517

Chemical Properties

Cas. No.	2260791-14-6		
SMILES	O=C1C=CC2=C(C=C(Cl)C(S(N[C@@H](C(N(CC(OCC)=O)CC3=CC=CS3)=O)C4=C(OC)C=CC=C4)(=O)=O)=C2)N1		
Formula	$C_{27}H_{26}ClN_3O_7S_2$	M.Wt	604.09
Solubility	DMSO : 125 mg/mL (206.92 mM)	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure



Protocol

Cell experiment [1]:	
Cell lines	Neural stem cells (NSCs) from SD rats

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Preparation Method	Rat NSCs were cultured in DMEM/F12 medium, supplemented with 2% B-27 supplements, 20ng/ml EGF, 20ng/ml FGF, 100U/ml penicillin, and 100U/ml streptomycin at 37°C in an incubator with 5% CO_2. Cells were seeded onto polyornithine-laminin-coated 24-well plates with a density of 3×10^4 cells/well. After 24h culture, the medium with different concentrations of OSMI-4 (0, 0.2, 1, and 2μM) was added. After 7 days of culture, the differentiation of the NSCs was evaluated through immunofluorescent staining.
Reaction Conditions	0, 0.2, 1, and 2μM; 7 days
Applications	OSMI-4 treatment enhanced the differentiation of the rat NSCs in a dose-dependent manner, accompanied by an increase in $MAP2^+$ cells and $Tuj1^+$ cells.
Animal experiment [2]:	
Animal models	Female BALB/c nude mice
Preparation Method	Female BALB/c nude mice (4 weeks old; 22-25g) were used. Each mouse was subcutaneously injected with 1×10^6 TE10 cells suspended in 200μl of sterile PBS. Tumor growth was monitored twice weekly by measuring dimensions with calipers, and volume was calculated using the formula $0.5\times length\times width^2$. When tumors reached $150mm^3$, mice were randomized into treatment groups. OSMI-4 was administered daily at 20mg/kg via intraperitoneal injection for 20 days. At the end of the experiment, mice were euthanized, and tumors were excised for analysis.
Dosage form	20mg/kg/day; 20 days; i.p.

Caution: Product has not been fully validated for medical applications. For research use only.
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Applications	OSMI-4 significantly inhibited the tumor volume in mice with TE10 xenografts.

References:

[1] Liu X, Song S, Chen Z, et al. Release of O-GlcNAc transferase inhibitor promotes neuronal differentiation of neural stem cells in 3D bioprinted supramolecular hydrogel scaffold for spinal cord injury repair[J]. Acta biomaterialia, 2022, 151: 148-162.

[2] Liu J, Cao D, Xu T, et al. O-GlcNAcylation stabilizes RSK4 by antagonizing GSK3β-mediated phosphorylation to enhance radioresistance in esophageal squamous cell carcinoma[J]. International Journal of Biological Sciences, 2026, 22(5): 2418.

Background

OSMI-4 is a potent O-GlcNAc transferase (OGT) inhibitor, with an EC_{50} value of 3μM[1]. OSMI-4 suppresses the catalytic activity of OGT, reduces the overall level of O-GlcNAc protein modification, and impairs homologous recombination repair and cell cycle progression[2]. OSMI-4 has been widely used in tumor research to reduce the overall O-GlcNAc levels and regulate cellular metabolic activity[3].

In vitro, OSMI-4 (2μM) incubated for 7 days induced the differentiation of rat neural stem cells and enhanced cell viability[4]. Treatment with 30μM OSMI-4 for 24 hours increased the levels of p62 and LC3-II proteins in CCD841CoN cells and disrupted the intracellular localization of lysosomes[5]. Treatment with 20μM OSMI-4 for 24 hours induced cell cycle arrest in HCT116 cells, resulting in increased expression of γH2AX, cleaved caspase 7, and cleaved PARP-1[6].

In vivo, OSMI-4 administration via intraperitoneal injection at a dose of 200μg every three days for 2 weeks markedly inhibited tumor growth and enhanced the number of cytotoxic $CD8^+$ T cells in the tumor tissue of Hepa1-6 xenograft mice[7]. Daily intraperitoneal injection of OSMI-4 (20mg/kg) for 20 days, combined with two rounds of localized X-ray irradiation (5Gy), significantly inhibited the tumor volume of TE10 xenograft mice[8].

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References:

[1] Martin S E S, Tan Z W, Itkonen H M, et al. Structure-Based Evolution of Low Nanomolar O-GlcNAc Transferase Inhibitors[J]. Journal of the American Chemical Society, 2018, 140(42): 13542-13545.

[2] Ping X, Stark J M. O-GlcNAc transferase is important for homology-directed repair[J]. DNA repair, 2022, 119: 103394.

[3] Loi E M, Weiss M, Pajk S, et al. Intracellular hydrolysis of small-molecule O-linked N-acetylglucosamine transferase inhibitors differs among cells and is not required for its inhibition[J]. Molecules, 2020, 25(15): 3381.

[4] Liu X, Song S, Chen Z, et al. Release of O-GlcNAc transferase inhibitor promotes neuronal differentiation of neural stem cells in 3D bioprinted supramolecular hydrogel scaffold for spinal cord injury repair[J]. Acta biomaterialia, 2022, 151: 148-162.

[5] Ahmed A B, Scache J, Mortuaire M, et al. Downregulation of O-GlcNAc transferase activity impairs basal autophagy and late endosome positioning under nutrient-rich conditions in human colon cells[J]. Biochemical and Biophysical Research Communications, 2024, 724: 150198.

[6] Loison I, Pioger A, Paget S, et al. O-GlcNAcylation inhibition redirects the response of colon cancer cells to chemotherapy from senescence to apoptosis[J]. Cell Death & Disease, 2024, 15(10): 762.

[7] Zhu Q, Wang H, Chai S, et al. O-GlcNAcylation promotes tumor immune evasion by inhibiting PD-L1 lysosomal degradation[J]. Proceedings of the National Academy of Sciences, 2023, 120(13): e2216796120.

[8] Liu J, Cao D, Xu T, et al. O-GlcNAcylation stabilizes RSK4 by antagonizing GSK3β-mediated phosphorylation to enhance radioresistance in esophageal squamous cell carcinoma[J]. International Journal of Biological Sciences, 2026, 22(5): 2418.

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