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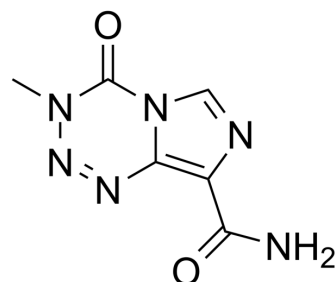
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Temozolomide

Cat. No.:	HY-17364
CAS No.:	85622-93-1
Molecular Formula:	C ₆ H ₆ N ₆ O ₂
Molecular Weight:	194.15
Target:	Autophagy; DNA Alkylator/Crosslinker; Apoptosis
Pathway:	Autophagy; Cell Cycle/DNA Damage; Apoptosis
Storage:	-20°C, protect from light, stored under nitrogen * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light, stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 20.83 mg/mL (107.29 mM; Need ultrasonic)
H₂O : 2.86 mg/mL (14.73 mM; Need ultrasonic)

	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
Preparing Stock Solutions	1 mM	5.1507 mL	25.7533 mL	51.5066 mL
	5 mM	1.0301 mL	5.1507 mL	10.3013 mL
	10 mM	0.5151 mL	2.5753 mL	5.1507 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

- Add each solvent one by one: PBS
Solubility: 9.09 mg/mL (46.82 mM); Clear solution; Need ultrasonic and warming and heat to 60°C
- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 1.25 mg/mL (6.44 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 1.25 mg/mL (6.44 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 1.25 mg/mL (6.44 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Temozolomide (NSC 362856) is an oral active DNA alkylating agent that crosses the blood-brain barrier. Temozolomide is also a proautophagic and proapoptotic agent. Temozolomide is effective against tumor cells that are characterized by low levels of O6-alkylguanine DNA alkyltransferase (OGAT) and a functional mismatch repair system. Temozolomide has antitumor and antiangiogenic effects^{[1][2]}.

IC₅₀ & Target	DNA alkylator ^[1]
In Vitro	<p>Temozolomide (TZA) is a methylating agent that crosses the blood-brain barrier and is indicated for malignant gliomas and metastatic melanomas. Temozolomide is effective against tumor cells that are characterized by low levels of O⁶-alkylguanine DNA alkyltransferase (OGAT) and a functional mismatch repair system (MR)^[1]. Determination of the IC₅₀ for Temozolomide (TZA) in different cell lines gave values ranging from 14.1 to 234.6 μM that fell into two clearly differentiated groups: cell lines with low IC₅₀ values (<50 μM), which include A172 (14.1±1.1 μM) and LN229 cells (14.5±1.1 μM), and those with high IC₅₀ values (>100 μM), which include SF268 (147.2±2.1 μM) and SK-N-SH cells (234.6±2.3 μM)^[3].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
In Vivo	<p>Temozolomide (TZA), as a single agent, does not significantly increase median survival time (MST) with respect to control. Noteworthy, intracranial injection of NU1025, immediately before the administration of 100 or 200 mg/kg Temozolomide, significantly increases lifespans with respect to controls or to groups treated with Temozolomide only. When Temozolomide is fractionated, the increase in lifespan (ILS) obtained with this schedule is higher than that observed when NU1025 is combined with a single injection of Temozolomide (statistical comparison of survival curves: NU1025 intracranially+Temozolomide 100 mg/kg×2 vs NU1025+Temozolomide 200 mg/kg; P=0.023)^[1].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

PROTOCOL

Cell Assay ^[1]	<p>The murine lymphoma cell line L5178Y of DBA/2 (H-2^d/H-2^d) origin is cultured in RPMI-1640 containing 10% fetal calf serum and antibiotics. Inhibition of PARP is obtained by treating cells (10⁵ cells/mL) with 8-hydroxy-2-methylquinazolin-4-[³H]-1 (NU1025), at a concentration (25 μM) that abrogates PARP activity. Cells are then exposed to Temozolomide (7.5-125 μM) and are cultured for 3 days. Cell growth is evaluated by counting viable cells in quadruplicate, and apoptosis is assessed by flow cytometry analysis of DNA content. Long-term survival is analyzed by colony-formation assay^[1].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
Animal Administration ^[1]	<p>Mice^[1]</p> <p>Male B6D2F1 (C57BL/6×DBA/2) mice are used. L5178Y cells (10⁴ in 0.03 mL RPMI-1640) are then injected intracranially, through the center-middle area of the frontal bone to a 2-mm depth, using a 0.1-mL glass microsyringe and a 27-gauge disposable needle. To evaluate tumor cell growth, brains are fixed in 10% phosphate-buffered formaldehyde, and histologic sections (5 μm) are cut along the axial plane, stained with hematoxylin-eosin, and analyzed by light microscopy.</p> <p>Temozolomide is dissolved in DMSO (40 mg/mL), diluted in saline (5 mg/mL), and administered intraperitoneally on day 2 after tumor injection at 100 mg/kg or 200 mg/kg, doses commonly used for in vivo preclinical studies. Because cytotoxicity induced by Temozolomide and PARP inhibitors can be improved by fractionated modality of treatment, in selected groups a total dose of 200 mg/kg Temozolomide is divided in 2 doses of 100 mg/kg given on days 2 and 3.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

CUSTOMER VALIDATION

- Nat Photonics. 2024 Jan 18.
- Cell Discov. 2023 Aug 29;9(1):90.
- Neuro Oncol. 2019 Nov 4;21(11):1423-1435.
- Adv Sci (Weinh). 2024 Mar 13:e2309290.
- Adv Sci (Weinh). 2024 Feb 14:e2306027.

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REFERENCES

- [1]. Tentori L, et al. Combined treatment with temozolomide and poly(ADP-ribose) polymerase inhibitor enhances survival of mice bearing hematologic malignancy at the central nervous system site. *Blood*. 2002 Mar 15;99(6):2241-4.
- [2]. Perazzoli G, et al. Temozolomide Resistance in Glioblastoma Cell Lines: Implication of MGMT, MMR, P-Glycoprotein and CD133 Expression. *PLoS One*. 2015 Oct 8;10(10):e0140131.
- [3]. Mathieu V, et al. Combining Anti-Human VEGF with temozolomide increases the antitumor efficacy of temozolomide in a human glioblastoma orthotopic xenograft model. *Neoplasia*. 2008 Dec;10(12):1383-92.
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Caution: Product has not been fully validated for medical applications. For research use only.

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