Teniposide

Cat. No.:	HY-13761
CAS No.:	29767-20-2
Molecular Formula:	C ₃₂ H ₃₂ O ₁₃ S
Molecular Weight:	656.65
Target:	Topoisomerase
Pathway:	Cell Cycle/DNA Damage
Storage:	4°C, protect from light
	* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)

Product Data Sheet

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SOLVENT & SOLUBILITY

	* "≥" means soluble, b	ut saturation unknown.					
		Solvent Mass Concentration	1 mg	5 mg	10 mg		
	Preparing Stock Solutions	1 mM	1.5229 mL	7.6144 mL	15.2288 mL		
		5 mM	0.3046 mL	1.5229 mL	3.0458 mL		
		10 mM	0.1523 mL	0.7614 mL	1.5229 mL		
	Please refer to the sol	Please refer to the solubility information to select the appropriate solvent.					
In Vivo	1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: ≥ 2.5 mg/mL (3.81 mM); Clear solution						
		2. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.5 mg/mL (3.81 mM); Clear solution					

BIOLOGICAL ACTIV	
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Description	Teniposide is a podophyllotoxin derivative, acts as a topoisomerase II inhibitor, and used as a chemotherapeutic agent.
IC ₅₀ & Target	Topoisomerase II
In Vitro	Teniposide is a topoisomerase II inhibitor. Teniposide (VM-26, 0.15-45 mg/L) inhibits the proliferation of Tca8113 cells in a dose-dependent manner, with an IC ₅₀ of 0.35 mg/L. Teniposide (5 mg/L) induces apoptosis of Tca8113 cells. Teniposide (5.0 mg/L) causes cell arrested at G2/M phase in Tca8113 cells ^[2] . Teniposide is active on primary cultured glioma cells from patients, when the level of miR-181b is high in the cells, with an IC ₅₀ of 1.3?±?0.34 µg/mL. Cells treated with teniposide with low MDM2 have decreased viability compared with control cells, and the IC ₅₀ decreases from 5.86?±?0.36 µg/mL to 2.90?±?0.35 µg/mL upon MDM2 suppression. Teniposide also inhibits the viability of glioma cell with high level of miR-181b,

	through mediation of MDM2 ^[3] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Teniposide (0.5 mg/kg, i.p.) significantly increases micronucleated polychromatic erythrocyte (MNPCE) frequencies, which is directly related to bone marrow toxicity as significant suppression of bone marrow is noted. Teniposide (24 mg/kg, i.p.) markedly decreases the frequencies of BrdU-labelled sperm. Teniposide (12, 24 mg/kg, i.p.) also dramatically induces disomic sperm in the germ cell of male mice ^[1] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL	
ROTOCOL	
Cell Assay ^[2]	Logarithmically growing Tca8113 cells are trypsinized and made into single cell suspension then plated in 9 plate at a concentration of 5 × 10 ⁴ cells/well, eight columns for Teniposide and seven columns for CDDP in e wells in each column. After 24 hours of incubation, the medium of the 3 wells in each column are replaced we containing Teniposide of 0.15 mg/L, 0.5 mg/L, 1.5 mg/L, 5.0 mg/L, 15 mg/L and 45 mg/L or CDDP of 0.1 mg/L mg/L, 3.0 mg/L and 9.0 mg/L, respectively. Blank control wells are added medium without drugs. Cells are t another 24 hours, 48 hours, 72 hours, 96 hours and 120 hours. The supernatants are removed and 20 µL MT added in each well, followed with another 4 hours of culture. The supernatants are discarded carefully and sulphoxide (DMSO) is added and shaken vigorously to dissolve the purple precipitation formation. Optical ceach well is tested using Spectrophotometer with a wavelength of 450 nm. The experiment is repeated in the MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration ^[1]	Animals (mice) are treated with 0.5 mg/kg teniposide and bone marrow is sampled 24 h after treatment. Co mitomycin C are used as a positive control aneugen and clastogen, respectively, at the dose of 2 mg/kg eac smears are prepared and stained with May-Gruenwald/Giemsa solutions. At least four slides are made for e allowed to dry overnight. One slide per animal is stained with May-Gruenwald/Giemsa solutions for conven assessment of the micronuclei (MN) frequencies in polychromatic erythrocytes (PCEs) and normochromatic (NCEs). The remaining unstained slides are stored at –20°C for the distinction between the clastogenic and effects by identifying the origin of MN with the mouse DNA probes. Per animal, 1000 PCE of coded slides are presence of MN. In addition, the number of PCEs among 1000 NCE per animal is recorded to evaluate bone suppression and mitotic activity is calculated as %PCE = [PCE/(PCE + NCE)] × 100 ^[1] .

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Immunother Cancer. 2022 Aug;10(8):e004006.
- Cell Death Dis. 2020 Nov 12;11(11):976.
- Acta Pharmacol Sin. 2021 Jan;42(1):108-114.
- Int Immunopharmacol. 2021 Oct 26;101(Pt A):108264.
- Mol Pharm. 2022 Oct 21.

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REFERENCES

[1]. Attia SM, et al. Molecular cytogenetic evaluation of the aneugenic effects of teniposide in somatic and germinal cells of male mice. Mutagenesis. 2012 Jan;27(1):31-9.

[2]. Li J, et al. Topoisomerase II trapping agent teniposide induces apoptosis and G2/M or S phase arrest of oral squamous cell carcinoma. World J Surg Oncol. 2006 Jul

6;4:41.

[3]. Sun YC, et al. MiR-181b sensitizes glioma cells to teniposide by targeting MDM2. BMC Cancer. 2014 Aug 25;14:611.

Caution: Product has not been fully validated for medical applications. For research use only.

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