Triptonide

Cat. No.: HY-32736

CAS No.: 38647-11-9

Molecular Formula: $C_{20}H_{22}O_6$

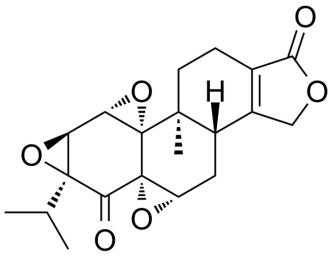
Molecular Weight: 358.39

Target: Wnt; β-catenin; Apoptosis; Autophagy

Pathway: Stem Cell/Wnt; Apoptosis; Autophagy

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (279.03 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.7903 mL	13.9513 mL	27.9026 mL
	5 mM	0.5581 mL	2.7903 mL	5.5805 mL
	10 mM	0.2790 mL	1.3951 mL	2.7903 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.98 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.98 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Triptonide (NSC 165677) is a natural product identified in Tripterygium wilfordii Hook F.. Triptonide is a Wnt signaling inhibitor with an IC_{50} of appropriately 0.3 nM. Triptonide has immunosuppression, anti-inflammatory, anti-fertility, neuroprotective and anti-lymphoma effects[1][2].

IC_{50} & Target

IC50: 0.3 nM (Wnt)[1]

In Vitro

Triptonide blocks Wnt/β-catenin signaling via C-terminal transactivation domain of β-catenin, and promots apoptosis in Wnt-dependent cancer cells[1].
Triptonide potently inhibits the proliferation of human B-lymphoma Raji and T-lymphoma Jurkat cells with IC_{50} of 5.7 nM and 4.8 nM, respectively[2].

Triptonide (2.5-10 nM; 6 days) significantly suppresses B-lymphoma cell colony-forming capability[2].
Triptonide (20 nM; 3 days) promotes apoptosis through activation of PARP and caspase 3, but reduction of BCL2 protein levels in the lymphoma cells[2].
Triptonide (5-10 nM; 72 hours) markedly reduces both total and phosphorylated Lyn proteins, and diminishes Lyn downstream ERK and ATK signal pathways[2].

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

Cell Proliferation Assay[2]

Cell Line:	B-lymphoma Raji cells, T-lymphoma Jurkat cells
Concentration:	0-80 nM
Incubation Time:	3 days, 6 days
Result:	Inhibited lymphoma cell tumorigenic capability in a dose-dependent manner.

Apoptosis Analysis[2]

Cell Line:	Raji cells
Concentration:	5 nM, 10 nM, 20 nM
Incubation Time:	3 days
Result:	Did not significantly induce apoptosis at the effective tumor growth-inhibitory (2.5-10 nM); moderately induced lymphoma cell apoptosis (20 nM).

Western Blot Analysis[2]

Cell Line:	Raji cells
Concentration:	5 nM, 10 nM, 20 nM
Incubation Time:	3 days
Result:	Did not vigorously activated pro-apoptotic proteins PARP and caspase 3 in lymphoma cells (5-10 nM); significantly activated PARP and caspase 3 (20 nM); significantly reduced anti-apoptotic BCL2 levels.

RT-PCR[2]

Cell Line:	Raji cells
Concentration:	5 nM, 10 nM
Incubation Time:	72 hours
Result:	Significantly diminished Lyn mRNA levels in the lymphoma cells.

In Vivo

Triptonide (5 mg/kg; i.p.; daily; for 34 days) exerts a strong anti-lymphoma effect in mice[2].

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

Animal Model:	Eight-week-old female NOD/SCID mice (18-22 g), with 3 x 10^7 Raji cells xenograft[2]
Dosage:	5 mg/kg
Administration:	Intraperitoneal injection, daily, for 34 days

Result:	Potently inhibited lymphoma cell growth and tumorigenic capability.

CUSTOMER VALIDATION

- Nat Commun. 2021 Feb 23;12(1):1253.
- Int J Mol Sci. 2023 Mar 22;24(6):6008.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Jessica Chinison, et al. Triptonide Effectively Inhibits Wnt/β-Catenin Signaling via C-terminal Transactivation Domain of β-catenin. Sci Rep. 2016; 6: 32779.

[2]. Ping Yang, et al. Triptonide acts as a novel potent anti-lymphoma agent with low toxicity mainly through inhibition of proto-oncogene Lyn transcription and suppression of Lyn signal pathway. Toxicol LettPing Yang. 2017 Aug 15;278:9-17.

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

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA