Etoposide

Cat. No.: HY-13629

CAS No.: 33419-42-0

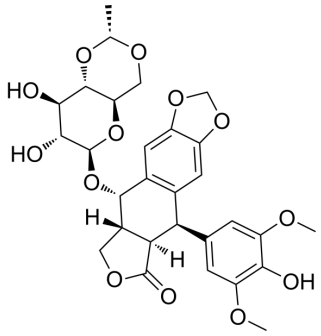
Molecular Formula: $C_{29}H_{32}O_{13}$

Molecular Weight: 588.56

Target: Topoisomerase; Autophagy; Mitophagy; Apoptosis; Bacterial; Antibiotic

Pathway: Cell Cycle/DNA Damage; Autophagy; Apoptosis; Anti-infection

Storage: 4°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 39 mg/mL (66.26 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.6991 mL	8.4953 mL	16.9906 mL
	5 mM	0.3398 mL	1.6991 mL	3.3981 mL
	10 mM	0.1699 mL	0.8495 mL	1.6991 mL

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 (4.25 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.25 mM); Clear solution
3. Add each solvent one by one: 5% DMSO >> 40% PEG300 >> 5% Tween-80 >> 50% saline
   Solubility: ≥ 2.5 mg/mL (4.25 mM); Clear solution
4. Add each solvent one by one: 5% DMSO >> 95% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (4.25 mM); Clear solution
5. Add each solvent one by one: 1% DMSO >> 99% saline
   Solubility: ≥ 0.5 mg/mL (0.85 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Etoposide (VP-16; VP-16-213) is an anti-cancer chemotherapy agent. Etoposide inhibits topoisomerase II, thus stopping DNA replication. Etoposide induces cell cycle arrest, apoptosis and autophagy[1].

IC_{50} & Target	Topoisomerase II

In Vitro

Etoposide is capable of causing cytotoxicity on pancreatic β-cells by inducing apoptosis through the JNK/ERK-mediated GSK-3 downstream-triggered mitochondria-dependent signaling pathway in RIN-m5F cells[1].
Etoposide and Anti-Human VEGF significantly abolish P1 sphere-forming ability, an effect associated with apoptosis of this subset of cells[2].
Etoposide phosphate (0-1μM; 72 hours) inhibits HCT116 $FBXW^{+/+}$, $FBXW^{-/-}$ and $p53^{-/-}$ as a dose-dependent manner, exhibits IC_{50}s of 0.945 μM; 0.375 μM; and 1.437 μM, respectively[5].
Etoposide (25 μM; 6 hours) delays p53 recover in FBXW7-deficient cells. In addition, FBXW7 expression is disappeared in $FBXW7^{-/-}$ cells[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[5]

Cell Line:	HCT116 $FBXW^{+/+}$, $FBXW^{-/-}$ and $p53^{-/-}$ cells
Concentration:	0.025 μM, 0.05 μM, 0.075 μM, 0.1 μM, 0.2 μM, 0.4 μM, 0.6 μM, 0.8 μM, 1 μM
Incubation Time:	72 hours
Result:	Inhibits HCT116 $FBXW^{+/+}$p>, $FBXW^{-/-}$ and $p53^{-/-}$ cell growth as a concentration manner.

Western Blot Analysis[5]

Cell Line:	HCT116 $FBXW7^{+/+}$ or $FBXW7^{-/-}$ cells
Concentration:	25 μM
Incubation Time:	6 hours
Result:	Exhibited that the recovery of p53 levels after DNA damage is mediated by FBXW7.

In Vivo

Etoposide (50 μM) and Anti-Human VEGF-treated hypoxic cells injected intravenously into immunodeficient mice reveals a reduced capacity to induce lung colonies, which also appear with a longer latency period[2]. Etoposide (10 mg/kg/day, i.v.) with NSC 109724 and NSC 241240, reduces the tumor volume in the hepatoblastoma cell injected NMRI nude mice[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Immunity. 2022 Aug 9;55(8):1370-1385.e8.
- Protein Cell. 2022 Jan;13(1):47-64.
- J Extracell Vesicles. 2022 Apr;11(4):e12206.
- Adv Sci (Weinh). 2020 Sep 28;7(21):2001364.
- Cell Rep Med. 2023 Jan 10;100911.

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REFERENCES

[1]. Lee KI, et al. Etoposide induces pancreatic β-cells cytotoxicity via the JNK/ERK/GSK-3 signaling-mediated mitochondria-dependent apoptosis pathway. Toxicol In Vitro. 2016 Jul 26. pii: S0887-2333(16)30147-3.

[2]. Calvani M, det al. Etoposide-Anti-Human VEGF a new strategy against human melanoma cells expressing stem-like traits. Oncotarget. 2016 Jun 9. doi: 10.18632/oncotarget.9939.

[3]. Fuchs, J., et al. Comparative activity of NSC 119875, NSC 109724, NSC 123127, NSC 241240, and etoposide in heterotransplanted hepatoblastoma. Cancer, 1998. 83(11): p. 2400-7.

[4]. Hande KR, et al. The Importance of Drug Scheduling in Cancer Chemotherapy: Etoposide as an Example. Oncologist. 1996;1(4):234-239.

[5]. Cui D, et al. FBXW7 Confers Radiation Survival by Targeting p53 for Degradation.Cell Rep. 2020 Jan 14;30(2):497-509.e4.

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