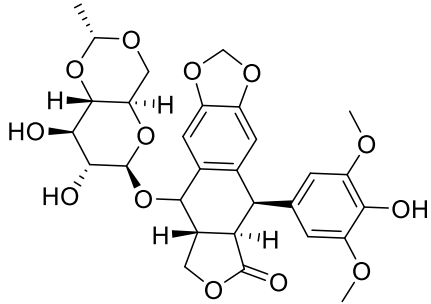


Product data sheet



MedKoo Cat#: 100330 Name: Etoposide CAS#: 33419-42-0 Chemical Formula: C ₂₉ H ₃₂ O ₁₃ Exact Mass: 588.18429 Molecular Weight: 588.55		
Product supplied as:		Powder
Purity (by HPLC):		≥ 98%
Shipping conditions		Ambient temperature
Storage conditions:		Powder: -20°C 3 years; 4°C 2 years. In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:

Etoposide is a semisynthetic derivative of podophyllotoxin and a substance extracted from the mandrake root *Podophyllum peltatum*. Possessing potent antineoplastic properties, etoposide binds to and inhibits topoisomerase II and its function in ligating cleaved DNA molecules, resulting in the accumulation of single- or double-strand DNA breaks, the inhibition of DNA replication and transcription, and apoptotic cell death. Etoposide acts primarily in the G₂ and S phases of the cell cycle. Check for active clinical trials or closed clinical trials using this agent. (NCI Thesaurus).

2. CoA, QC data, SDS, and handling instruction

SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under “QC And Documents” section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	100	169.91

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	1.70 mL	8.50 mL	16.99 mL
5 mM	0.34 mL	1.70 mL	3.40 mL
10 mM	0.17 mL	0.85 mL	1.70 mL
50 mM	0.03 mL	0.17 mL	0.34 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator

Please refer the product web page under section of “Calculator”

6. Recommended literature which reported protocols for in vitro and in vivo study

In vitro study

1. Lee KI, Su CC, Yang CY, Hung DZ, Lin CT, Lu TH, Liu SH, Huang CF. Etoposide induces pancreatic β -cells cytotoxicity via the JNK/ERK/GSK-3 signaling-mediated mitochondria-dependent apoptosis pathway. *Toxicol In Vitro*. 2016 Oct;36:142-152. doi: 10.1016/j.tiv.2016.07.018. Epub 2016 Jul 27. PMID: 27473919.

In vivo study

1. Slater LM, Stupecky M, Sweet P, Osann K, Eklof A, Arquilla ER. Etoposide induction of tumor immunity in Lewis lung cancer. *Cancer Chemother Pharmacol*. 2001 Oct;48(4):327-32. doi: 10.1007/s002800100357. PMID: 11710634.

7. Bioactivity

Biological target:

Etoposide (VP-16, VP-16213) is a semisynthetic derivative of podophyllotoxin, which inhibits DNA synthesis via topoisomerase II inhibition activity.

In vitro activity

Product data sheet



In RIN-m5F cells (a β -cell-derived cell line), the number of viable cells was significantly decreased after 24h of etoposide treatment and underwent mitochondria-dependent apoptotic signals accompanied by mitochondrial dysfunction, and increases in the population of sub-G1 hypodiploid cells and apoptotic cells, caspase-3 activity, and the activation of caspase cascades. Etoposide also increased the phosphorylation levels of glycogen synthase kinase (GSK)-3 α/β in treated RIN-m5F cells. Pretreatment with LiCl, a GSK-3 inhibitor, prevented etoposide-induced mitochondria-dependent apoptosis and GSK-3 protein phosphorylation in RIN-m5F cells. Furthermore, exposure of the cells to etoposide induced the phosphorylation of c-Jun N-terminal kinase (JNK) and extracellular signal-related kinase (ERK)1/2 but not p38-MAPK.

Reference: Toxicol In Vitro. 2016 Oct;36:142-152. [https://linkinghub.elsevier.com/retrieve/pii/S0887-2333\(16\)30147-3](https://linkinghub.elsevier.com/retrieve/pii/S0887-2333(16)30147-3)

In vivo activity

Of C57B1/6 mice injected with 10(6) Lewis lung cancer (3LL) cells followed by treatment with a single 50 mg/kg dose of etoposide (VP-16), 60% survived over 60 days, in contrast to untreated control mice which died within 30 days. Approximately 40% of surviving mice rejected a subsequent challenge with 3LL. Their splenocytes protected naive mice injected with 3LL. To test if VP-16 treatment produced alterations in 3LL cells, which induce host immunity, leading to tumor rejection, C57B1/6 mice were injected with 3LL cells that had survived an 80-90% lethal concentration of VP-16 in vitro. These cells killed 75% of recipient mice but 60% of the surviving mice rejected challenge with 3LL. Splenocytes harvested from tumor-rejecting mice protected naive mice injected with 3LL.

Reference: Cancer Chemother Pharmacol. 2001 Oct;48(4):327-32. <https://dx.doi.org/10.1007/s002800100357>

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.