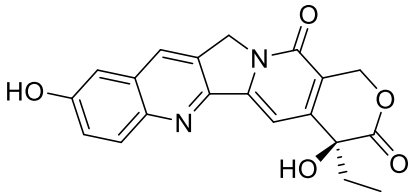
Product data sheet

MedKoo Cat#: 406281 Name: 10-Hydroxycamptothecin CAS#: 19685-09-7 Chemical Formula: $C_{20}H_{16}N_2O_5$ Exact Mass: 364.10592 Molecular Weight: 364.35	
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:
10-Hydroxycamptothecin (10-HCPT), an indole alkaloid isolated from a Chinese tree, Camptotheca acuminate, inhibits the activity of topoisomerase I and has a broad spectrum of anticancer activity in vitro and in vivo.

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	2.0	5.49
DMF	2.0	5.49

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.74 mL	13.72 mL	27.45 mL
5 mM	0.55 mL	2.74 mL	5.49 mL
10 mM	0.27 mL	1.37 mL	2.74 mL
50 mM	0.05 mL	0.27 mL	0.55 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. Zeng L, Sun Y, Li X, Wang J, Yan L. 10-Hydroxycamptothecin induces apoptosis in human fibroblasts by regulating miRNA-23b-3p expression. Mol Med Rep. 2019 Apr;19(4):2680-2686. doi: 10.3892/mmr.2019.9927. Epub 2019 Feb 1. PMID: 30720099; PMCID: PMC6423607.
2. Song M, Yin S, Zhao R, Liu K, Kundu JK, Shim JH, Lee MH, Dong Z. (S)-10-Hydroxycamptothecin Inhibits Esophageal Squamous Cell Carcinoma Growth In Vitro and In Vivo Via Decreasing Topoisomerase I Enzyme Activity. Cancers (Basel). 2019 Dec 6;11(12):1964. doi: 10.3390/cancers11121964. PMID: 31817790; PMCID: PMC6966462.

In vivo study

1. Song M, Yin S, Zhao R, Liu K, Kundu JK, Shim JH, Lee MH, Dong Z. (S)-10-Hydroxycamptothecin Inhibits Esophageal Squamous Cell Carcinoma Growth In Vitro and In Vivo Via Decreasing Topoisomerase I Enzyme Activity. Cancers (Basel). 2019 Dec 6;11(12):1964. doi: 10.3390/cancers11121964. PMID: 31817790; PMCID: PMC6966462.
2. Dai J, Sun Y, Yan L, Wang J, Li X, He J. Upregulation of NOXA by 10-Hydroxycamptothecin plays a key role in inducing fibroblasts apoptosis and reducing epidural fibrosis. PeerJ. 2017 Jan 12;5:e2858. doi: 10.7717/peerj.2858. PMID: 28097065; PMCID: PMC5237371.

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7. Bioactivity

Biological target:

(S)-10-Hydroxycamptothecin (10-HCPT;10-Hydroxycampto thecin) is a DNA topoisomerase I inhibitor of isolated from the Chinese plant Camptotheca accuminata

In vitro activity

To verify the role of miR-23b-3p in HCPT-induced fibroblast apoptosis, human fibroblasts were transfected with Lv-miR-23b-3p and Lv-anti-miR-23b-3p, followed by co-treatment with HCPT. TUNEL assays revealed that miR-23b-3p increased HCPT-induced fibroblast apoptosis (Fig. 4A). Flow cytometric analysis confirmed the TUNEL assay results; HCPT treatment increased the fibroblast apoptotic rate compare to the control group, and transfection with Lv-anti-miR-23b-3p partially reversed the increased apoptosis caused by HCPT, whereas transfection with Lv-miR-23b-3p significantly increased cell apoptosis caused by HCPT (Fig. 4B). Consistent with these apoptosis data, treatment with HCPT alone or with Lv-miR-23b-3p co-transfection increased the expression of apoptosis-related proteins cleaved-PARP and BAX, and decreased the expression of Bcl-2, compared with the Control group (Fig. 4C). Transfecting Lv-anti-miR-23b-3p into the fibroblasts partially attenuated the HCPT-induced increased expression of these proteins. These data indicated that upregulation of miR-23b-3p expression by HCPT treatment increases human fibroblast apoptosis.

Reference: Mol Med Rep. 2019 Apr; 19(4): 2680–2686. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6423607/

In vivo activity

To examine the possible inhibitory effects of HCPT on PDX tumor growth, patient-derived LEG104 and LEG110 ESCC tumors with high expression levels of TOP I were adopted in this investigation. The treatment of PDX tumors grown in mice with HCPT (4 mg/kg and 8 mg/kg body weight) decreased the tumor volume and tumor weight significantly (Figure 5A–D). The weight and size of tumors were also dramatically decreased after HCPT administration for 18 or 29 days (Figure 5B,C,E,F). While eliciting antitumor activity in PDX tumor-bearing mice, HCPT application did not exhibit any remarkable signs of toxicity in mice (Figure S3). The IHC and HE staining of the PDX tumor sections showed more regression of tumor cells and changes in morphology, such as cell enlargement, caryolysis, variable nucleus size, nuclear pyknosis, and nucleolar margin blurring in the treatment group in comparison with the vehicle group (Figure 5G). Darker staining in the cytoplasm by eosin in the HCPT-treated groups indicated apoptosis phenomenon which was not evident in the vehicle group (Figure 5G). Moreover, there was a significant decrease in the expression of Ki-67 and elevation in cleaved caspase-3, which indicated that the cell proliferation was inhibited and apoptosis was induced after HCPT application. The IHC analysis of HCPT-treated tumors also showed decreased TOP I expression and increased phosphorylated rH2A.X level (Figure 5G,H, Figure S4). These findings mean that TOP I appears to be a valid therapeutic target and HCPT may be considered for further clinical application in ESCC.

Reference: Cancers (Basel). 2019 Dec; 11(12): 1964. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6966462/