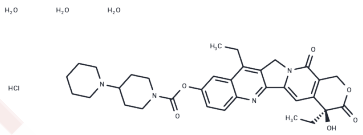
Data Sheet (Cat.No.T0486)

TargetMol

Irinotecan hydrochloride trihydrate

Chemical Properties



CAS No. : 136572-09-3

Formula: C33H45ClN4O9

Molecular Weight: 677.18

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	Irinotecan hydrochloride trihydrate (CPT-11 HCl Trihydrate) keeps DNA from unwinding by inhibiting topoisomerase 1.
Targets(IC50)	Autophagy,Topoisomerase
In vitro	In the liver, stomach, duodenum, and colon, a single dose of Irinotecan significantly increases the covalent binding of topoisomerase I to DNA. Compared to the control group, there is a marked increase in DNA strand breaks within the colonic mucosal cells in the Irinotecan-treated group. In COLO320 xenografts, Irinotecan induces a 92% maximum growth inhibition.
In vivo	In LoVo and HT-29 cell lines, Irinotecan induces a similar number of cleavable complexes at IC50 concentrations, with no significant difference observed between the two. The formation of cleavable complexes by SN-38 is concentration-dependent and consistent across both cell lines. Notably, intracellular accumulation of Irinotecan differs significantly, with consistently higher levels in HT-29 cells compared to LoVo cells. Carboxylesterase activation of Irinotecan to SN-38 (primarily in the liver) facilitates interaction with its target, topoisomerase I, in plasma, intestines, and tumor tissues. The hydrolysis of Irinotecan's lactone E-ring and SN-38 is reversible in aqueous solution, with the interconversion of their carboxylate and lactone forms dependent on temperature and pH. For the same concentration of SN-38 glucuronide and Irinotecan in tumor and normal tissues, the yield of SN-38 via β-glucuronidase mediation exceeds that of SN-38 produced from Irinotecan. In SCLC cell lines, Irinotecan exhibits markedly greater activity than in NSCLC cell lines, while tissue histology does not reveal significant differences in SN-38 efficacy.
Cell Research	Exponentially growing cells (LoVo and HT-29 cells) are seeded in 20 cm2 Petri dishes with an optimal cell number for each cell line (2 × 104 for LoVo cells, 105 for HT-29 cells). They are treated 2 days later with increasing concentrations of Irinotecan or SN-38 for one cell doubling time (24 hours for LoVo cells, 40 hours for HT-29 cells). After washing with 0.15 M NaCl, the cells are further grown for two doubling times in normal medium, detached from the support with trypsin-EDTA and counted in a hemocytometer. The IC50 values are then estimated as the Irinotecan or SN-38 concentrations responsible for 50% growth inhibition as compared with cells incubated without Irinotecan or SN-38. (Only for Reference)

Solubility Information

Solubility	H2O: 1 mg/mL (1.48 mM),Sonication is recommended. Ethanol: 7 mg/mL (10.34 mM),Sonication is recommended. DMSO: 50 mg/mL (73.84 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 2 mg/mL (2.95 mM),Sonication is recommended. *Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.*

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	1.4767 mL	7.3836 mL	14.7671 mL
5 mM	0.2953 mL	1.4767 mL	2.9534 mL
10 mM	0.1477 mL	0.7384 mL	1.4767 mL
50 mM	0.0295 mL	0.1477 mL	0.2953 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Pavillard V, et al. Cancer Chemother Pharmacol. 2002, 49(4), 329-335.
Tobin P, et al. Br J Clin Pharmacol. 2006, 62(1), 122-129.
Shingyoji M, et al. Cancer Sci. 2004, 95(6), 537-540.
van Ark-Otte J, et al. Br J Cancer. 1998, 77(12), 2171-2176.
Jansen WJ, et al. Int J Cancer. 1997, 70(3):335-340.