

Epothilone B

Catalog No: tcsc0714



Available Sizes

Size: 2mg

Size: 5mg

Size: 10mg

Size: 50mg



Specifications

CAS No:

152044-54-7

Formula:

$C_{27}H_{41}NO_6S$

Pathway:

Cell Cycle/DNA Damage;Cytoskeleton

Target:

Microtubule/Tubulin;Microtubule/Tubulin

Purity / Grade:

>98%

Solubility:

DMSO : ≥ 125 mg/mL (246.22 mM)

Alternative Names:

EPO 906;Patupilone

Observed Molecular Weight:

507.68

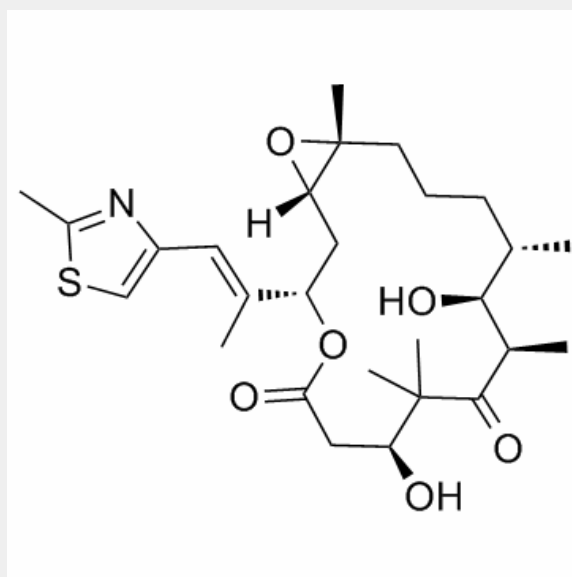
Product Description

Epothilone B is a **microtubule** (MT) targeting agent with **EC_{0.01}** of 1.8 μM.

IC50 & Target: EC0.01: 1.8 μM (Microtubule/Tubulin)^[1]

In Vitro: Epothilone B inhibits HCT116 cells with IC₅₀ of 0.8 nM in HCT-116 cell line cytotoxicity assay^[1]. Epothilone B (Patupilone) is a microtubule (MT) targeting agent. As shown by MTT cell proliferation assay, after 72 h of treatment Epothilone B efficiently inhibits cell growth with an IC₅₀ of 6 nM, while concentrations ≤1 nM are not cytotoxic. Epothilone B significantly inhibits transwell cell migration at the non-cytotoxic concentration of 1 nM, and the effect is more evident at 10 nM^[2]. Epothilone B (Patupilone) is a novel, non-taxane-related and nonneurotoxic microtubule-stabilizing agent in human medulloblastoma cell lines. Epothilone B reduces the proliferative activity in the D341 cell line, with an IC₅₀ of 0.53 nM; in the D425Med cell line, with an IC₅₀ of 0.37 nM; and in the DAOY cell line, with an IC₅₀ of 0.19 nM. In the D341Med cell line, the effect of Epothilone B on clonogenic survival is at dose range of Epothilone B similar to the level of proliferative activity and viability (IC₅₀, 0.50-0.75 nM). However, the clonogenicity of D425Med and DAOY cells is already strongly reduced at a 10-fold lower concentration of Epothilone B (IC₅₀, 30 pM). These results overall demonstrate that Epothilone B is highly potent against different medulloblastoma cell lines^[3].

In Vivo: Treatment with Epothilone B (Patupilone) or ionizing radiation alone results in a partial tumor growth suppression over 10 days, whereas combined treatment exerts a strong supra-additive tumor growth control, with complete tumor regression in the follow-up period (P[3].



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