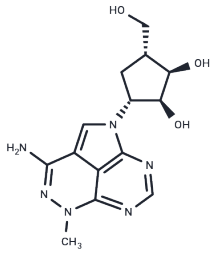
Data Sheet (Cat.No.T6065)

TargetMol

Triciribine

Chemical Properties



CAS No. : 35943-35-2

Formula: C13H16N6O4

Molecular Weight: 320.3

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	Triciribine (NSC-154020) is a DNA synthesis inhibitor, and it also inhibits Akt and HIV-1/2.
Targets(IC50)	Akt,HIV Protease,DNA/RNA Synthesis
In vitro	Triciribine exhibits maximum growth inhibition around 1-10 μM and inhibits phosphorylation of Akt, as well as downstream p70S6K, to basal levels at 100 μM (IC50 = 130 nM). Triciribine shows particular promise for inhibiting growth in Nf1 and Trp53 mutant astrocytoma cells in a grade-dependent manner. The WHO II K1861-10 line is inhibited, incompletely (69% maximum inhibition), with a GI50 value of 1.7 μM for Triciribine, whereas higher-grade tumor lines (KR158, KR130, and SF295) are inhibited to a greater extent (>80% maximum inhibition) at lower GI50 values (0.4–1.1 mM). Importantly, Triciribine is much less effective at inhibiting primary astrocytes (GI5013.6 mM), suggesting that this inhibitor may show specificity for tumor cells. [1] Triciribine inihibits HIV-1with an IC50 of 20 nM. Greater than 90% inhibition is achieved at 0.1 μM and complete inhibition of syncytia formation is achieved at 5 μM. Associated cell toxicity in the same cell line for Triciribine is 46 μM, resulting in selectivity indices of 2250. Triciribine markedly inhibits HIV-1-induced p24 core antigen production, reverse transcriptase, and infectious virus production in a dose-dependent manner using HIV-1 acutedly infected CEM-SS, H9, and persistently infected H9III B and U1 cells. [2] Triciribine inhibits Akt phosphorylation at Thr308 and Ser473 and Akt activity in the human prostate cancer cell line PC-3. Triciribine sensitizes PC-3 cells to TRAIL- and anti-CD95-induced apoptosis, whereas the cells remain resistant to DNA damaging chemotherapeutics. [3] Triciribine is highly selective for Akt and does not inhibit the activation of phosphatidylinositol 3-kinase, phosphoinositide-dependent kinase-1, protein kinase C, serum and glucocorticoid-inducible kinase, protein kinase A, signal transducer and activators of transcription 3, extracellular signal-regulated kinase-1/2, or c-Jun NH2-terminal kinase. [4]
In vivo	1 mg/kg/day i.p. treated Triciribine inhibits OVCAR3, OVCAR8 and PANC1 tumor growth, which overexpressing Akt, by 90%, 88% and 80% in nude mice, respectively. However, Triciribine has little effect on the growth of OVCAR5 and COLO357 cells. [4]
Kinase Assay	Akt Phosphorylation Changes Assay: Cells are grown to 80%–90% confluency and stimulated for 5–10 minutes with 1–10 ng/mL of epidermal growth factor or platelet derived growth factor (PDGF)-AA with or without 10–20 mM of U0126 or LY-294002.

Kinase Assay	Protein lysates (5-20 μg) are separated by 12%-15% SDS PAGE and analyzed by Western blot for Akt, phosphorylated Akt (phospho-Ser 473), MAPK, and phosphorylated MAPK (p44/42 phospho-Thr202/Tyr204) antibodies (1:1000).
Cell Research	Triciribine is evaluated for cytotoxicity by seeding CEM-SS cells at a density of 1 × 104 cells/well in growth medium, using a 96-well flat-bottom plate. Serial fivefold dilutions of Triciribine are prepared in growth medium and added to the wells as a second overlay. After a 48-hours incubation at 37 °C, the cells are pulse labeled with [3H]dThd (1 μCi per well, specific activity 20 Ci/mmol) for 6 hours and the cells are harvested to measure total DNA synthesis.(Only for Reference)

Solubility Information

Solubility	DMSO: 106 mg/mL (330.94 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 (6.24 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	3.1221 mL	15.6104 mL	31.2207 mL
5 mM	0.6244 mL	3.1221 mL	6.2441 mL
10 mM	0.3122 mL	1.561 mL	3.1221 mL
50 mM	0.0624 mL	0.3122 mL	0.6244 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

Gursel DB, et al, Nero Oncol, 2011, 13(6), 610-621.
Wang G, Xu J, Ma H, et al. Phenolipid JE improves metabolic profile and inhibits gluconeogenesis via modulating AKT-mediated insulin signaling in STZ-induced diabetic mice. Pharmacological Research. 2022: 106569.
Kucera LS, et al, AIDS Res Hum Retroviruses, 1993, 9(4), 307-314.
Dieterle A, et al, Int J Cancer , 2009, 125(4), 932-941.
Yang L, et al, Cancer Res, 2004, 64(13), 4394-4399.