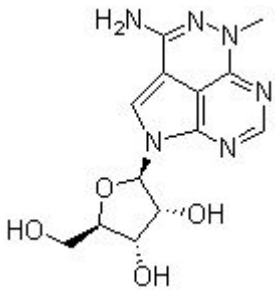
Biochempartner
瀚香生物科技

Product Introduction

Triciribine

Triciribine is a **DNA synthesis** inhibitor, also inhibits **Akt** and **HIV-1** with **IC50** of 130 nM and 20 nM, respectively; does not inhibit PI3K/PDK1; 5000-fold less active in cells lacking adenosine kinase. Phase 1/2.

Technical Data:

Molecular Weight (MW):	320.3	
Formula:	$C_{13}H_{16}N_6O_4$	
Solubility (25°C) * <1 mg/ml means slightly soluble or insoluble:	DMSO 64 mg/mL	
	Water <1 mg/mL	
	Ethanol <1 mg/mL	
Purity:	>98%	
Storage:	3 years -20℃Powder	
	6 months-80℃in DMSO	
CAS No.:	35943-35-2	

Biological Activity

Triciribine exhibits maximum growth inhibition around 1-10 μM and inhibits phosphorylation of Akt, as well as downstream p70S6K, to basal levels at 100μM (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

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(>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 0.1μM and complete inhibition of syncytia formation is achieved at 5μM. 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]

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]

References

[1] Gursel DB, et al, Nero Oncol, 2011, 13(6), 610-621.

[2] Kucera LS, et al, AIDS Res Hum Retroviruses, 1993, 9(4), 307-314.

[3] Dieterle A, et al, Int J Cancer , 2009, 125(4), 932-941.

[4] Yang L, et al, Cancer Res, 2004, 64(13), 4394-4399.

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