Web: www.taiclone.com
Tel: +886-2-2735-9682
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Triciribine

Catalog No: tcsc0968

Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Size: 100mg

Specifications

CAS No:
35943-35-2

Formula:
$C_{13}H_{16}N_6O_4$

Pathway:
PI3K/Akt/mTOR;Cell Cycle/DNA Damage;Anti-infection

Target:
Akt;DNA/RNA Synthesis;HIV

Purity / Grade:
>98%

Solubility:
DMSO : ≥ 44 mg/mL (137.37 mM)

Alternative Names:
API-2;NSC 154020;TCN

Observed Molecular Weight:
320.3

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Product Description

Triciribine is a **DNA synthesis** inhibitor, also inhibits **Akt** and **HIV-1/2** with **IC_{50}** of 130 nM, and 0.02-0.46 μM, respectively.

IC50 & Target: DNA synthesis[1]

IC50: 130 nM (Akt)[2]

IC50: 0.02-0.46 μM (HIV-1/2)[3]

In Vitro: The nucleoside analog Triciribine (TCN) is a purine analog which is initially shown to inhibit DNA synthesis. Triciribine selectively inhibits the phosphorylation and activation of all three Akt isoforms. At a concentration of 10 μM Triciribine Akt phosphorylation is inhibited at both Thr308 and Ser473. Triciribine effectively inhibits the phosphorylation and consequently the catalytic activity of Akt in PC-3 cells[1]. The Akt inhibitor Triciribine (TCN) does not effectively inhibit the human cell line U87MG but inhibits other astrocytoma cell lines in a grade-dependent manner. The WHO II K1861-10 line is incompletely inhibited (69% maximum inhibition) with a GI_{50} value of 1.7 μM for Triciribine. 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 (IC_{50}=130 nM) in KR158 cells[2]. Triciribine (TCN) is a novel tricyclic compound with known antitumor activity. Using a syncytial plaque assay, Triciribine is active against HIV-1 at 0.01-0.02 μM. Using a microtiter XTT assay, Triciribine is active against a panel of HIV-1 and HIV-2 strains at IC_{50} values ranging from 0.02 to 0.46 μM[3].

In Vivo: Triciribine (TCBN) treatment, administered for 7 days after 14 days of hypoxia until 21 days of hypoxia is reached, reversed the vascular thickening as shown by immunohistochemistry and Western analyses. On the other hand, Rapamycin treatment did not prevent hypoxia-induced pulmonary alveolar haemorrhage and congestion. Triciribine partially inhibited progressive pruning of the vasculature, which supports our previous finding that Triciribine alleviates vessel occlusion in microcapillaries. In contrast, Rapamycin treatment did not significantly reverse the reduced vascular density due to chronic hypoxia and had no significant effect on pruning of small vessels[4].