产品名称：Triciribine
产品别名：Triciribine

生物活性：

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.

IC_{50} & Target [1][2][3]	DNA synthesis	Akt	HIV-1	HIV-2
		130 nM (IC_{50}, Cell Assay)	0.02-0.46 μM (IC_{50})	0.02-0.46 μM (IC_{50})

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 IC50 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].

Solvent&Solubility

In Vitro:

DMSO : 100 mg/mL (**312.21 mM;** Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	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	1.5610 mL	3.1221 mL

*请根据产品在不同溶剂中的溶解度选择合适的溶剂配制储备液；一旦配成溶液，请分装保存，避免反复冻融造成的产品失效。

储备液的保存方式和期限：-80°C, 6 months; -20°C, 1 month。-80°C 储存时，请在 6 个月内使用，-20°C 储存时，请在 1 个月内使用。

In Vivo:

请根据您的实验动物和给药方式选择适当的溶解方案。以下溶解方案都请先按照 **In Vitro** 方式配制澄清的储备液，再依次添加助溶剂：

——为保证实验结果的可靠性，澄清的储备液可以根据储存条件，适当保存；体内实验的工作液，建议您现用现配，当天使用； 以下溶剂前显示的百分比是指该溶剂在您配制终溶液中的体积占比；如在配制过程中出

	现沉淀、析出现象，可以通过加热和/或超声的方式助溶 1.请依序添加每种溶剂： 10% DMSO→40% PEG300 →5% Tween-80 → 45% saline Solubility: ≥ 2.5 mg/mL (7.81 mM); Clear solution 此方案可获得 ≥ 2.5 mg/mL (7.81 mM，饱和度未知) 的澄清溶液。 以 1 mL 工作液为例，取 100 μL 25.0 mg/mL 的澄清 DMSO 储备液加到 400 μL PEG300 中，混合均匀向上述体系中加入 50 μL Tween-80，混合均匀；然后继续加入 450 μL 生理盐水定容至 1 mL。 2.请依序添加每种溶剂： 10% DMSO→ 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.5 mg/mL (7.81 mM); Clear solution 此方案可获得 ≥ 2.5 mg/mL (7.81 mM，饱和度未知) 的澄清溶液。 以 1 mL 工作液为例，取 100 μL 25.0 mg/mL 的澄清 DMSO 储备液加到 900 μL 20% 的 SBE-β-CD 生理盐水水溶液中，混合均匀。 3.请依序添加每种溶剂： 10% DMSO →90% corn oil Solubility: ≥ 2.5 mg/mL (7.81 mM); Clear solution 此方案可获得 ≥ 2.5 mg/mL (7.81 mM，饱和度未知) 的澄清溶液，此方案不适用于实验周期在半个月以上的实验。 以 1 mL 工作液为例，取 100 μL 25.0 mg/mL 的澄清 DMSO 储备液加到 900 μL 玉米油中，混合均匀。
References	[1]. Dieterle A, et al. The Akt inhibitor triciribine sensitizes prostate carcinoma cells to TRAIL-induced apoptosis. Int J Cancer. 2009 Aug 15;125(4):932-41. [2]. Gürsel DB, et al. Control of proliferation in astrocytoma cells by the receptor tyrosine kinase/PI3K/AKT signaling axis and the use of PI-103 and TCN as potential anti-astrocytoma therapies. Neuro Oncol. 2011 Jun;13(6):610-21 [3]. Kucera LS, et al. Activity of triciribine and triciribine-5'-monophosphate against human immunodeficiency virus types 1 and 2. AIDS Res Hum Retroviruses. 1993 Apr;9(4):307-14. [4]. Abdalla M, et al. The Akt inhibitor, triciribine, ameliorates chronic hypoxia-induced vascular pruning and TGFβ-induced pulmonary fibrosis. Br J Pharmacol. 2015 Aug;172(16):4173-88.

实验参考：

Cell Assay	The human SF295 and U87MG GBM lines and the mouse K1861-10, KR158, and KR130G astrocytoma lines are plated at a density of 2500 cells/100 μL complete media in 96-well plates. Mouse primary astrocytes are plated at 5000 cells/100 μL. Cells are treated in triplicate with serial dilutions of inhibitors (The inhibitors tested are PI-103, Triciribine and Rapamycin) ranging from μM to pM. Cell proliferation is measured after 3 days using the Alamar Blue assay on a Novostar plate reader. Values for 50% inhibitory concentration (IC_{50}) and 50% growth inhibitory concentration (GI_{50}) are calculated using standard procedures in GraphPad Prism v4 and Microsoft Excel[2].
Animal Administration	Mice[4] $Akt1^{+/+}$ and $Akt1^{-/-}$ mice are subjected to normoxia or hypoxia (10% O_2) for 7 and 14 days (n=2-6 mice per group). Noteworthy, high mortality is observed in $Akt1^{-/-}$ mice exposed to hypoxia longer than 14-16 days. For pharmacological inhibition studies, $Akt1^{+/+}$ mice, subjected to normoxia or chronic hypoxia for 14 days, received daily i.p. injection of saline, Triciribine (0.5 mg/kg per day) or Rapamycin (1.5 mg/kg per day) for 7 days, and the total continuous exposure to hypoxia or normoxia is 21 days (n=6-8 mice per group). Pharmacological inhibitors are administered daily while

	the mice are maintained in the hypoxia chamber to minimize exposure to air and spontaneous reversal of pulmonary remodelling.
References	[1]. Dieterle A, et al. The Akt inhibitor triciribine sensitizes prostate carcinoma cells to TRAIL-induced apoptosis. Int J Cancer. 2009 Aug 15;125(4):932-41. [2]. Gürsel DB, et al. Control of proliferation in astrocytoma cells by the receptor tyrosine kinase/PI3K/AKT signaling axis and the use of PI-103 and TCN as potential anti-astrocytoma therapies. Neuro Oncol. 2011 Jun;13(6):610-21 [3]. Kucera LS, et al. Activity of triciribine and triciribine-5'-monophosphate against human immunodeficiency virus types 1 and 2. AIDS Res Hum Retroviruses. 1993 Apr;9(4):307-14. [4]. Abdalla M, et al. The Akt inhibitor, triciribine, ameliorates chronic hypoxia-induced vascular pruning and TGFβ-induced pulmonary fibrosis. Br J Pharmacol. 2015 Aug;172(16):4173-88.