产品名称：5-碘代杀结核菌素
产品别名：5-Iodotubercidin；NSC 113939; 5-ITu

生物活性：

Description	5-Iodotubercidin (NSC 113939), an ATP mimetic, is a potent adenosine kinase inhibitor with an IC50 of 26 nM. 5-Iodotubercidin (NSC 113939) initiates glycogen synthesis in isolated hepatocytes by causing inactivation of phosphorylase and activation of glycogen synthase. 5-Iodotubercidin (NSC 113939) also inhibits CK1, insulin receptor tyrosine kinase, phosphorylase kinase, PKA, CK2, PKC and Haspin[1][2][3].
IC_{50} & Target	IC50: 26 nM (adenosine kinase)
In Vitro	5-Iodotubercidin (NSC 113939) inhibits CK1, insulin receptor tyrosine kinase, phosphorylase kinase, PKA, CK2 and PKC, and the IC50 values are 0.4, 3.5, 5-10, 5-10, 10.9 and 27.7 μM respectively[1]. 5-Iodotubercidin (20 μM) causes an important decrease in ATP concentration, and a concomitant smaller increase in AMP concentration. 5-Iodotubercidin decreases the activity of ACC and the rates of synthesis of fatty acids and cholesterol. In line with the iodotubercidin-mediated inhibition of ACC, 5-iodotubercidin induces a marked decrease in the intracellular concentration of malonyl-CoA[4].
In Vivo	5-Iodotubercidin (1 mL/kg, i.p.) is in agreement with activity observed against bicuculline-induced seizures following local administration of the AKI into the prepiriform cortex[2].

Solvent&Solubility

In Vitro:

DMSO : ≥ 49 mg/mL (124.95 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5500 mL	12.7502 mL	25.5004 mL
	5 mM	0.5100 mL	2.5500 mL	5.1001 mL
	10 mM	0.2550 mL	1.2750 mL	2.5500 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 (6.38 mM); Clear solution

此方案可获得 ≥ 2.5 mg/mL (6.38 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 (6.38 mM); Clear solution

此方案可获得 ≥ 2.5 mg/mL (6.38 mM，饱和度未知) 的澄清溶液。

源叶生物

	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 (6.38 mM); Clear solution 此方案可获得 ≥ 2.5 mg/mL (6.38 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]. Massillon D, et al. Identification of the glycogenic compound 5-iodotubercidin as a general protein kinase inhibitor. Biochem J. 1994 Apr 1;299 (Pt 1):123-8. [2]. Ugarkar BG, et al. Adenosine kinase inhibitors. 1. Synthesis, enzyme inhibition, and antiseizure activity of 5-iodotubercidin analogues. J Med Chem. 2000 Jul 27;43(15):2883-93. [3]. García-Villafranca J, et al. Effects of 5-iodotubercidin on hepatic fatty acid metabolism mediated by the inhibition of acetyl-CoA carboxylase. Biochem Pharmacol. 2002 Jun 1;63(11):1997-2000. [4]. De Antoni A, et al. A small-molecule inhibitor of Haspin alters the kinetochore functions of Aurora B. J Cell Biol. 2012 Oct 15;199(2):269-84. [5]. Acharya MM, et al. Adenosine Kinase Inhibition Protects against Cranial Radiation-Induced Cognitive Dysfunction. Front Mol Neurosci. 2016 Jun 3;9:42.

实验参考：

Cell Assay	HeLa cells are grown in DME supplemented with 10% fetal bovine serum (FBS) and 2 mM l-glutamine. Nocodazole is used at a concentration of 3.3 μM unless differently specified. Thymidine (2.5 mM) is used in the asssay. For transfection, FuGENE 6 Transfection Agent is used at a 3:1 ratio with plasmid DNA. Cells are analyzed 24-48 h after transfection. [4]
Animal Administration	Male SA rats (100-150 g) are maintained on a 12:12 light:dark cycle in temperaturecontrolled facilities with free access to food and water. One hour prior to seizure testing, the animals are injected intraperitoneally (1 mL/kg) with DMSO vehicle or with test compound dissolved in DMSO. At the time of the test, an electrolyte solution (2% lidocaine in 0.9% sodium chloride) is applied to the eyes. Maximal electroshock seizures are induced by administering a 60-Hz current of 150 mA for 0.2 s via corneal electrodes, using a Wahlquist Model H stimulator. The endpoint measured is suppression of hindlimb tonic extension (HTE) and expressed as percentage of animals in which the response is inhibited. At this supramaximal stimulation level, virtually 100% of control (vehicle-treated) animals show HTE. ED_{50}values are calculated from a dose-response curve using probit analysis. The N for the screening doses is 6-8; doseresponse determinations are conducted with at least 5 animals/dose. [2]
Kinase Assay	AK activity is measured in a radiochemical assay. The final reaction volume is 100 μL and contained 70 mM Tris-maleate (pH 7.0), 0.1% (w/v) bovine serum albumin, 1.0 mM $MgCl_2$, 1.0 mM ATP, 1.0 μM [U-^{14}C]adenosine (400-600 mCi/mmol) and various inhibitor concentrations. Inhibitors are prepared as 10 mM stock solutions in DMSO. The final DMSO concentration in the assay is 5% (v/v). Eleven different concentration of the test solutions ranging from 0.001 to 10.0 μM are utilized to determine a dose response curve of the inhibition of the enzyme. Reactions are started by adding the appropriate amount of purified human recombinant AK and incubated for 20 min at 37°C. The reactions are terminated by addition of the potent AKI GP3269. A 30-μL aliquot of each reaction is

	spotted on DEAE cellulose filter paper (cut in squares of appr 1×1 cm) and air-dried for 30 min. The dry filters are then washed for 3 min in deionized water to remove residual [U-^{14}C]adenosine, rinsed with ethanol and dried at 90°C for 20 min. The filter papers are counted in 5.5 mL of Ready Safe liquid scintillation cocktail using a Beckman LS3801 scintillation counter. Control AK activity is determined from the amount of [^{14}C]AMP formed in the presence of 5% DMSO. The concentration of inhibitor required to inhibit 50% of the AK activity (IC_{50}) is determined graphically from plots of inhibitor concentration versus percent (%) control enzyme activity. [2]
References	[1]. Massillon D, et al. Identification of the glycogenic compound 5-iodotubercidin as a general protein kinase inhibitor. Biochem J. 1994 Apr 1;299 (Pt 1):123-8. [2]. Ugarkar BG, et al. Adenosine kinase inhibitors. 1. Synthesis, enzyme inhibition, and antiseizure activity of 5-iodotubercidin analogues. J Med Chem. 2000 Jul 27;43(15):2883-93. [3]. García-Villafranca J, et al. Effects of 5-iodotubercidin on hepatic fatty acid metabolism mediated by the inhibition of acetyl-CoA carboxylase. Biochem Pharmacol. 2002 Jun 1;63(11):1997-2000. [4]. De Antoni A, et al. A small-molecule inhibitor of Haspin alters the kinetochore functions of Aurora B. J Cell Biol. 2012 Oct 15;199(2):269-84. [5]. Acharya MM, et al. Adenosine Kinase Inhibition Protects against Cranial Radiation-Induced Cognitive Dysfunction. Front Mol Neurosci. 2016 Jun 3;9:42.

源叶生物