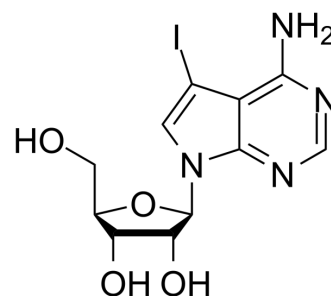


## 5-Iodotubercidin

<b>Cat. No.:</b>	HY-15424		
<b>CAS No.:</b>	24386-93-4		
<b>Molecular Formula:</b>	C <sub>11</sub> H <sub>13</sub> IN <sub>4</sub> O <sub>4</sub>		
<b>Molecular Weight:</b>	392		
<b>Target:</b>	Adenosine Kinase		
<b>Pathway:</b>	Metabolic Enzyme/Protease; Neuronal Signaling		
<b>Storage:</b>	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	1 year
		-20°C	6 months



### SOLVENT & SOLUBILITY

#### In Vitro

DMSO : 25 mg/mL (63.78 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	2.5510 mL	12.7551 mL	25.5102 mL
	5 mM	0.5102 mL	2.5510 mL	5.1020 mL
	10 mM	0.2551 mL	1.2755 mL	2.5510 mL

Please refer to the solubility information to select the appropriate solvent.

#### In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline  
Solubility: ≥ 2.5 mg/mL (6.38 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)  
Solubility: ≥ 2.5 mg/mL (6.38 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil  
Solubility: ≥ 2.5 mg/mL (6.38 mM); Clear solution
- Add each solvent one by one: 5% DMSO >> 40% PEG300 >> 5% Tween-80 >> 50% saline  
Solubility: ≥ 2.5 mg/mL (6.38 mM); Clear solution
- Add each solvent one by one: 5% DMSO >> 95% (20% SBE-β-CD in saline)  
Solubility: ≥ 2.5 mg/mL (6.38 mM); Clear solution
- Add each solvent one by one: 1% DMSO >> 99% saline  
Solubility: ≥ 0.5 mg/mL (1.28 mM); Clear solution

### BIOLOGICAL ACTIVITY

#### Description

5-Iodotubercidin (NSC 113939), an ATP mimetic, is a potent adenosine kinase inhibitor with an IC<sub>50</sub> 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 <sup>[1][2][3]</sup> .
<b>IC<sub>50</sub> &amp; Target</b>	IC <sub>50</sub> : 26 nM (adenosine kinase)
<b>In Vitro</b>	5-Iodotubercidin (NSC 113939) inhibits CK1, insulin receptor tyrosine kinase, phosphorylase kinase, PKA, CK2 and PKC, and the IC <sub>50</sub> values are 0.4, 3.5, 5-10, 5-10, 10.9 and 27.7 μM respectively <sup>[1]</sup> . 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 <sup>[4]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.
<b>In Vivo</b>	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 <sup>[2]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## PROTOCOL

<b>Kinase Assay</b> <sup>[2]</sup>	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 <sub>2</sub> , 1.0 mM ATP, 1.0 μM [U- <sup>14</sup> 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- <sup>14</sup> 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 [ <sup>14</sup> C]AMP formed in the presence of 5% DMSO. The concentration of inhibitor required to inhibit 50% of the AK activity (IC <sub>50</sub> ) is determined graphically from plots of inhibitor concentration versus percent (%) control enzyme activity. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
<b>Cell Assay</b> <sup>[4]</sup>	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 assay. For transfection, FuGENE 6 Transfection Agent is used at a 3:1 ratio with plasmid DNA. Cells are analyzed 24-48 h after transfection. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
<b>Animal Administration</b> <sup>[2]</sup>	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 <sub>50</sub> 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. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## CUSTOMER VALIDATION

- Cell Stem Cell. 2023 Apr 6;30(4):450-459.e9.
- Sci Transl Med. 2020 May 6;12(542):eaba0769.
- Cell Rep. 2023 May 23;42(6):112547.
- Front Mol Neurosci. 2016 Jun 3;9:42.
- Sci Rep. 2017 Feb 21;7:42885.

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## 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.

**Caution: Product has not been fully validated for medical applications. For research use only.**

Tel: 609-228-6898

Fax: 609-228-5909

E-mail: [tech@MedChemExpress.com](mailto:tech@MedChemExpress.com)

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA