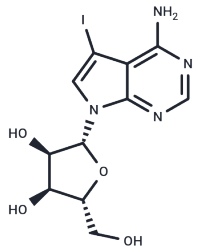
Data Sheet (Cat.No.T6745)

TargetMol
YOUR TARGET MOLECULES

5-Iodotubercidin

Chemical Properties



CAS No. : 24386-93-4

Formula: C11H13IN4O4

Molecular Weight: 392.15

Appearance: no data available

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year

Biological Description

Description	5-Iodotubercidin (NSC-113939) is a potent adenosine kinase inhibitor with IC50 of 26 nM. It inhibits nucleoside transporter, CK1, insulin receptor tyrosine kinase, phosphorylase kinase, PKA, CK2 and PKC.
Targets(IC50)	Adenosine Receptor
In vitro	5-Iodotubercidin(Itu) is a genotoxic drug that activates the Atm-p53 pathway and has anti-tumor activity. Itu is unique compared to other nucleoside analogs in the way that it induces G2 arrest in a p53 -dependent manner. Moreover, at higher doses, Itu might activate p53-independent pathways, which may cooperate with p53 to kill cells and inhibit tumor growth. Incorporation of Itu metabolite into DNA causes DNA breaks, which triggers the DNA damage response. Itu might be a potential chemotherapeutic drug with distinct working mechanisms[2].
In vivo	5-Iodotubercidin is a potent inhibitor of adenosine kinase in the rat brain. 5-iodotubercidin has potent hypotensive, muscle relaxant, and hypothermic actions in mice, and that these effects could be blocked by theophylline, an adenosine receptor antagonist. 5-iodotubercidin (1 mg/kg, i.p.) administered prior to an ischemic episode, fails to provide a degree of cerebroprotection, assessed either by locomotor or histopathological indices Even though 5-iodotubercidin administration is known to enhance extracellular adenosine levels in brain slices[1].
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 MgCl2, 1.0 mM ATP, 1.0 µM [U-14C]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-14C]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 [14C]AMP formed in the presence of 5% DMSO. The concentration of inhibitor required to inhibit

	50% of the AK activity (IC50) is determined graphically from plots of inhibitor concentration versus percent (%) control enzyme activity.
Cell Research	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.
Animal Research	Animal Models: Male Mongolian gerbilsFormulation: salineDosages: 1, 2.5 and 5 mg/kgAdministration: i.p.

Solubility Information

Solubility	DMSO: 19.6 mg/mL (50 mM), (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.550 mL	12.7502 mL	25.5004 mL
5 mM	0.510 mL	2.550 mL	5.1001 mL
10 mM	0.255 mL	1.275 mL	2.550 mL
50 mM	0.051 mL	0.255 mL	0.510 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Reference

Phillis JW, et al. Life Sci. 1993, 53(6):497-502.
Zhang X, et al. PLoS One. 2013, 8(5):e62527.