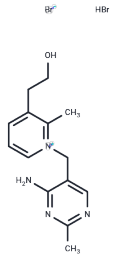
Data Sheet (Cat.No.T36947)

TargetMol

Pyrithiamine hydrobromide

Chemical Properties



CAS No. : 534-64-5

Formula: C14H20Br2N4O

Molecular Weight: 420.14

Storage: Keep away from moisture,Keep away from direct sunlight,Store at low temperature
Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	Pyrithiamine hydrobromide, an inhibitor of thiamine metabolism that acts as a substrate for thiamine pyrophosphate kinase, causes neurologic symptoms similar to wernickke - korsakoff syndrome in animals.
Targets(IC50)	Others
In vivo	In adult rats, intraperitoneal administration of Pyrithiamine hydrobromide (0.5 mg/kg, twice daily for 14 days) effectively induced thiamine deficiency, leading to behavioral abnormalities (e.g., ataxia, seizures) and neuronal degeneration in the thalamus and hippocampus. This confirms its utility as an anti-thiamine agent for creating neurodegenerative disease models[1].

Solubility Information

Solubility	H2O: 80 mg/mL (190.41 mM),Sonication is recommended. PBS (pH 7.2): 5 mg/mL (11.9 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.3802 mL	11.9008 mL	23.8016 mL
5 mM	0.476 mL	2.3802 mL	4.7603 mL
10 mM	0.238 mL	1.1901 mL	2.3802 mL
50 mM	0.0476 mL	0.238 mL	0.476 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.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Lee H, Tarter J, Holburn GE, Price RR, Weinstein DD, Martin PR. In vivo localized proton NMR spectroscopy of thiamine-deficient rat brain. Magn Reson Med. 1995 Sep;34(3):313-8.
Woolley, D.W. An enzymatic study of the mode of action of pyrithiamine (neopyrithiamine). J. Biol. Chem. 191(1), 43-54 (1951).
Vetreno, R.P., Anzalone, S.J., and Savage, L.M. Impaired, spared, and enhanced ACh efflux across the hippocampus and striatum in diencephalic amnesia is dependent on task demands. Neurobiol. Learn Mem. 90(1), 237-244 (2008).
Zahr, N.M., Sullivan, E.V., Rohlfing, T., et al. Concomitants of alcoholism: Differential effects of thiamine deficiency, liver damage, and food deprivation on the rat brain in vivo. Psychopharmacology (Berl) 233(14), 2675-2686 (2016).