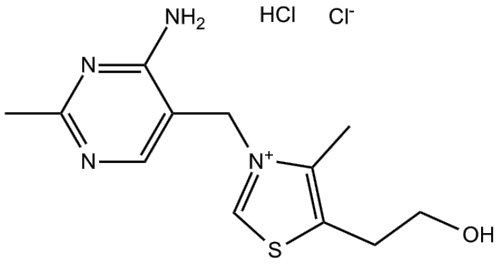
Product Data Sheet

Product Name: Vitamin B1 (Thiamine HCl) (Standard)
Cat. No.: GC14040

Chemical Properties

Cas. No.	67-03-8		
Chemical Name	2-[3-[(4-amino-2-methylpyrimidin-5-yl)methyl]-4-methyl-1,3-thiazol-3-ium-5-yl]ethanol;chloride;hydrochloride		
SMILES	CC1=C(SC=[N+]1CC2=CN=C(N=C2N)C)CCO.Cl.[Cl-]		
Formula	$C_{12}H_{17}N_4OS.HCl$	M.Wt	337.27
Solubility	≥ 10.9mg/mL in Water	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure



Protocol

Cell experiment [1]:

Cell lines C6 rat glioma cells (glioblastoma cell line)

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Preparation Method	C6 cells were cultured in Dulbecco's modified Eagle's medium (DMEM) containing 1% penicillin-streptomycin and 10% fetal bovine serum (FBS) at 37°C in a 5% CO_2 atmosphere. Thiamine and glutamate were dissolved in physiological saline and freshly prepared before use. Cells were treated with Thiamine (0.25–4mg/mL) for 1 hour before exposure to glutamate (10mM) for 24 hours.
Reaction Conditions	0.25–4mg/mL; 1h
Applications	Thiamine significantly increased cell viability against glutamate-induced cytotoxicity. Thiamine also reduced oxidative stress markers (MDA levels) and increased antioxidant enzyme levels (SOD and CAT). Thiamine decreased endoplasmic reticulum stress proteins (GRP78, ATF-4, CHOP) and caspase-3 levels, mitigating apoptosis and necroptosis.

Animal experiment [2]:

Animal models	SLC19A3-deficient mice
Preparation Method	Mice were maintained on a conventional diet (1.71mg/100g Thiamine) until 5–6 weeks of age, then switched to a Thiamine-restricted diet (0.60mg/100g Thiamine) for 2, 3, or 5 days, followed by a high-Thiamine diet (8.50mg/100g Thiamine). Mice were sacrificed at different time points for brain analysis.
Dosage form	0.60mg/100g, 1.71mg/100g, 8.50mg/100g; o.p.

Caution: Product has not been fully validated for medical applications. For research use only.
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Applications	High-dose Thiamine administration significantly prevented brain lesions and prolonged survival in SLC19A3-deficient mice. Thiamine deficiency caused acute neurodegeneration, which was largely preventable with prompt high-dose thiamine treatment

References:
[1] Ergül M, Ta?k?ran A?. Thiamine Protects Glioblastoma Cells against Glutamate Toxicity by Suppressing Oxidative/Endoplasmic Reticulum Stress. Chem Pharm Bull (Tokyo). 2021;69(9):832-839.
[2] Suzuki K, Yamada K, Fukuhara Y, et al. High-dose thiamine prevents brain lesions and prolongs survival of Slc19a3-deficient mice. PLoS One. 2017 Jun 30;12(6):e0180279.

Background

Vitamin B1 (Thiamine HCl) (Standard) is a water-soluble essential micronutrient[1]. Thiamine readily crosses the blood–brain barrier and is an important tool compound for studying energy metabolism, neuroinflammation, and aging-related metabolic dysfunction[2]. Vitamin B1 (Thiamine HCl) (Standard) is converted into its active coenzyme form, thiamine diphosphate (ThDP), by thiamine diphosphokinase in the liver, kidneys, and leukocytes[3]. ThDP acts as a cofactor for several key metabolic enzymes, including pyruvate dehydrogenase, α-ketoglutarate dehydrogenase, and transketolase, which are involved in regulating glycolysis, the tricarboxylic acid cycle, and the pentose phosphate pathway, thereby maintaining cellular energy homeostasis. Thiamine deficiency can lead to lactic acidosis, oxidative stress, and neuronal damage[4].

In vitro, treatment of rat embryonic hippocampal neurons with Vitamin B1 (Thiamine HCl) (Standard) (30–300μM) for 72 hours significantly increased the survival of hippocampal neurons in high cell density culture ($1\times10^5/cm^2$), with the maximal effect at 100μM[5]. Pre-treatment of C6 glioma cells with Vitamin B1 (Thiamine HCl) (Standard) (0.25–4mg/mL) for 1 hour, followed by exposure to glutamate (10mM) for 24 hours, significantly increased cell viability, reduced levels of the oxidative stress marker malondialdehyde (MDA), while increasing levels of superoxide dismutase (SOD) and catalase (CAT), decreased endoplasmic reticulum stress markers, and reduced the number of

apoptotic cells[6].

In vivo, a high-dose Vitamin B1 (Thiamine HCl) (Standard) diet (8.50mg/100g) was used to treat Slc19a3 knockout (Slc19a3 KO) mice after 2 or 3 days of a thiamine-restricted diet (0.60mg/100g). Thiamine significantly prolonged the survival of the mice and prevented acute neurodegeneration[7]. Thiamine (200mg/kg) was administered via intraperitoneal injection to Balb/c male mice. Chronic administration of Thiamine alone (once daily for 14 days) significantly increased the threshold for pentylenetetrazole-induced seizures[8].

References:
[1] Polegato BF, Pereira AG, Azevedo PS, et al. Role of Thiamin in Health and Disease. Nutr Clin Pract. 2019 Aug;34(4):558-564.
[2] Kerns JC, Gutierrez JL. Thiamin. Adv Nutr. 2017 Mar 15;8(2):395-397.
[3] Chan AHY, Ho TCS, Irfan R, et al. Design of thiamine analogues for inhibition of thiamine diphosphate (ThDP)-dependent enzymes: Systematic investigation through Scaffold-Hopping and C2-Functionalisation. Bioorg Chem. 2023 Sep;138:106602.
[4] Bettendorff L. Synthetic Thioesters of Thiamine: Promising Tools for Slowing Progression of Neurodegenerative Diseases. Int J Mol Sci. 2023 Jul 10;24(14):11296.
[5] Geng MY, Saito H, Katsuki H. The effects of thiamine and oxythiamine on the survival of cultured brain neurons. Jpn J Pharmacol. 1995 Jul;68(3):349-52.
[6] Ergül M, Taşkıran AŞ. Thiamine Protects Glioblastoma Cells against Glutamate Toxicity by Suppressing Oxidative/Endoplasmic Reticulum Stress. Chem Pharm Bull (Tokyo). 2021;69(9):832-839.
[7] Suzuki K, Yamada K, Fukuhara Y, et al. High-dose thiamine prevents brain lesions and prolongs survival of Slc19a3-deficient mice. PLoS One. 2017 Jun 30;12(6):e0180279.
[8] Mesdaghinia A, Alinejad M, Abed A, et al. Anticonvulsant effects of thiamine on pentylenetetrazole-induced seizure in mice. Nutr Neurosci. 2019 Mar;22(3):165-173.