

Product Data Sheet

Product Name: Pentylenetetrazole

Cat. No.: GC12357

Chemical Properties

Cas. No. 54-95-5

Chemical Name 6,7,8,9-tetrahydro-5H-tetrazolo[1,5-a]azepine

SMILES N12N=NN=C1CCCCC2

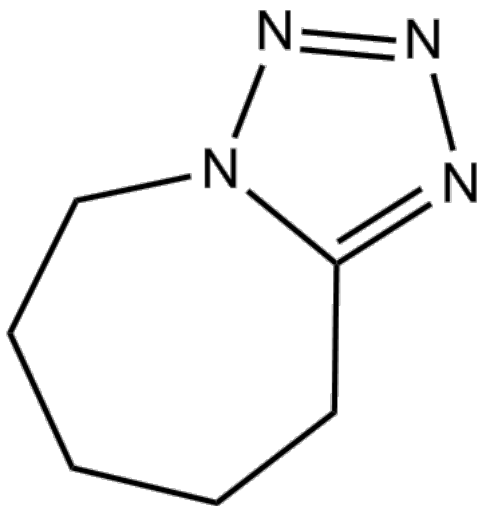
Formula $C_6H_{10}N_4$ M.Wt 138.17

Solubility DMF: 30 mg/ml, DMSO: 30 mg/ml, Ethanol: 30 mg/ml, PBS (pH 7.2): 3 mg/ml
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

Animal experiment [1]:

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Animal models Male Balb-c mice

Male Balb-c mice (3–6 months old) were housed in small groups in a room with controlled light/dark cycle (12/12h) at 22°C. They were separated in two groups and treated as follows: In group I (single convulsive dose), animals received i.p. 60mg/kg Pentylenetetrazole dissolved in 0.9% saline and were decapitated 15min, 30min and 24h after tonic clonic seizure. In group II (controls), animals received i.p. the same volume of saline and were decapitated 30min after administration of saline. The brain was quickly dissected and the hippocampus was removed and washed with ice-cold phosphate-EDTA buffer [10mM phosphate buffer, pH 7.2, containing 1mM EDTA and 1mM of the antioxidant BHA (in final 0.15% ethanol)]. Then, the tissue was homogenized in a glass-glass PotterElvehjem homogenizer and was used for the subsequent assays.

Dosage form 60mg/kg; i.p.; 15min, 30min and 24h

Pentylenetetrazole significantly decreased protein thiols (PSH), cysteine (CSH), and non-protein mixed disulfides (NPSSC), while increasing protein symmetric disulfides (PSSP), non-protein symmetric disulfides (NPSSR), lipid peroxidation, and protein oxidation levels in the mouse hippocampus.

References:

[1] Patsoukis N, Zervoudakis G, Panagopoulos NT, Georgiou CD, Angelatou F, Matsokis NA. Thiol redox state (TRS) and oxidative stress in the mouse hippocampus after pentylenetetrazol-induced epileptic seizure. *Neurosci Lett.* 2004;357(2):83-86.

Background

Pentylenetetrazole is a non-selective antagonist of the γ-aminobutyric acid (GABA) A receptor [1]. The GABAA receptor, a major inhibitory neurotransmitter receptor widely present in the central nervous

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system, mediates the fast inhibitory effects of GABA by increasing the permeability of the cell membrane to chloride ions, causing hyperpolarization and thus inhibiting neuronal excitability^[2]. Pentylentetrazole is often used to study the mechanism of epileptic seizures, screen antiepileptic drugs, and investigate oxidative stress and neuroinflammatory responses^{[3][4]}.

In vivo, Pentylentetrazole (35mg/kg; i.p.; every other day for 14 times) induced epileptic seizures, increased levels of oxidative stress markers (e.g., malondialdehyde, MDA), decreased the activities of antioxidant enzymes (e.g., glutathione, GSH; superoxide dismutase, SOD; catalase, CAT), elevated the levels of inflammatory cytokines (e.g., IL-1 β , IL-6, TNF- α), and led to neuroinflammatory responses and neuronal damage in mice model^[5]. Intraperitoneal injection of Balb-c mice with Pentylentetrazole (60mg/kg; i.p.; 24h) significantly decreased protein thiols (PSH), cysteine (CSH), and non-protein mixed disulfides (NPSSC), while increasing protein symmetric disulfides (PSSP), non-protein symmetric disulfides (NPSSR), lipid peroxidation, and protein oxidation levels in the mouse hippocampus^[6]. Pentylentetrazole (35mg/kg; i.p.; three times a week) induced kindling in rats, significantly increased the hippocampal concentrations of alanine, arginine, glutamate, aspartate, and taurine, while decreasing GABA levels, leading to an over fourfold increase in the Glu/GABA ratio^[7].

References:

- [1] Huang RQ, Bell-Horner CL, Dibas MI, Covey DF, Drewe JA, Dillon GH. Pentylentetrazole-induced inhibition of recombinant gamma-aminobutyric acid type A (GABA(A)) receptors: mechanism and site of action. *J Pharmacol Exp Ther.* 2001;298(3):986-995.
- [2] Lambert JJ, Belelli D, Hill-Venning C, Peters JA. Neurosteroids and GABAA receptor function. *Trends Pharmacol Sci.* 1995;16(9):295-303.
- [3] Shimada T, Yamagata K. Pentylentetrazole-Induced Kindling Mouse Model. *J Vis Exp.* 2018; (136):56573.
- [4] Monteiro ÁB, Alves AF, Ribeiro Portela AC, et al. Pentylentetrazole: A review. *Neurochem Int.* 2024;180:105841.
- [5] Younis NS, Almostafa MM, Mohamed ME. Geraniol Ameliorates Pentylentetrazol-Induced Epilepsy, Neuroinflammation, and Oxidative Stress via Modulating the GABAergic Tract: In vitro and in vivo studies. *Drug Des Devel Ther.* 2024;18:5655-5672.
- [6] Patsoukis N, Zervoudakis G, Panagopoulos NT, Georgiou CD, Angelatou F, Matsokis NA. Thiol redox state (TRS) and oxidative stress in the mouse hippocampus after pentylentetrazol-induced

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epileptic seizure. *Neurosci Lett.* 2004;357(2):83-86.

[7] Szyndler J, Maciejak P, Turzyńska D, et al. Changes in the concentration of amino acids in the hippocampus of pentylenetetrazole-kindled rats. *Neurosci Lett.* 2008;439(3):245-249.

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