Tinoridine hydrochloride

Cat. No.: HY-111354

CAS No.: 25913-34-2

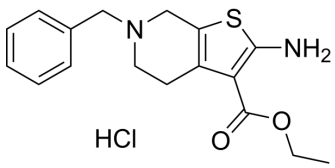
Molecular Formula: $C_{17}H_{21}ClN_2O_2S$

Molecular Weight: 352.88

Target: Glutathione Peroxidase

Pathway: Metabolic Enzyme/Protease

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 16.67 mg/mL (47.24 mM; Need ultrasonic)
H_2O : 2.5 mg/mL (7.08 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.8338 mL	14.1691 mL	28.3382 mL
5 mM	0.5668 mL	2.8338 mL	5.6676 mL
10 mM	0.2834 mL	1.4169 mL	2.8338 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 1.67 mg/mL (4.73 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1.67 mg/mL (4.73 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Tinoridine hydrochloride is a nonsteroidal anti-inflammatory agent and also has potent radical scavenger and antiperoxidative activity.

In Vitro

Tinoridine reduces a stable free radical, diphenyl-p-picrylhydrazyl, in the molar ratio of about 1:2, indicating its free radical scavenging ability. Tinoridine inhibits the lipid peroxidation in rat liver microsomes induced by xanthine-xanthine oxidase system in the presence of ADP and Fe^{2+}, in which hydroxyl radical is formed. Tinoridine is demonstrated to be oxidized in the course of the lipid peroxidation by following the fluorescence derived from the oxidation product of tinoridine. It is also oxidized by the xanthine-xanthine oxidase system in the presence of Fe^{2+}, but its oxidation is slow in the absence of Fe^{2+} and almost completely inhibited by catalase. Tinoridine is also oxidized by H_2O_2-Fe^{2+} system producing OH (Fenton reaction), but it does not affect the reduction of cytochrome c caused by superoxide radical[1].

	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	CCl_4 aministration produces a marked decrease in the concentrations of liver microsomal cytochrome P-450 and G6Pase, indicating that hepatic endoplasmic reticulum function is disrupted. Prior treatment of the animals with tinoridine (100 mg/kg) significantly reduces the CCl_4-induced alterations in the enzyme activities, and a rapid recovery toward the normal values is observed[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [2]	CCl_4 aministration produces a marked decrease in the concentrations of liver microsomal cytochrome P-450 and G6Pase, indicating that hepatic endoplasmic reticulum function is disrupted. Prior treatment of the animals with tinoridine (100 mg/kg) significantly reduces the CCl_4-induced alterations in the enzyme activities, and a rapid recovery toward the normal values is observed[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Rat: Male Wistar rats (180-220 g) are used in the experiments. Drugs (Tinoridine) are given orally as a suspension in 0.5% methylcellulose solution 1 hr before CCl_4, administration. Control animals receive an equivalent amount of the vehicle. CCl_4 is administered ip at a dose of 0.25 ml/kg as a 5% (v/v) solution in olive oil. The animals are killed by carotid excision at different times after CCl_4 administration; the livers are rapidly removed, weighed and processed for biochemical or histologic analysis[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. O Shimada, et al. Hydroxyl radical scavenging action of tinoridine. Agents Actions. 1986 Nov;19(3-4):208-14.

[2]. Yasuda H, et al. The protective effect of tinoridine against carbon tetrachloride hepatotoxicity. Toxicol Appl Pharmacol. 1980 Mar 15;52(3):407-13.

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