SZABO SCANDIC

Part of Europa Biosite

Produktinformation

- Forschungsprodukte & Biochemikalien
- Zellkultur & Verbrauchsmaterial
- Diagnostik & molekulare Diagnostik
- Laborgeräte & Service

Weitere Information auf den folgenden Seiten!
See the following pages for more information!

Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

F. +43(0)1 489 3961-7

mail@szabo-scandic.com

www.szabo-scandic.com

linkedin.com/company/szaboscandic

Thioacetamide

Cat. No.: HY-Y0698

CAS No.: 62-55-5

Molecular Formula: C_2H_5NS

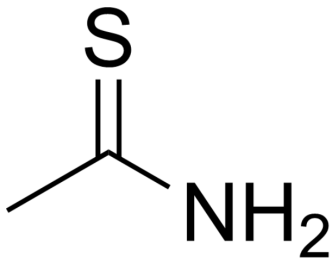
Molecular Weight: 75.13

Target: Necroptosis

Pathway: Apoptosis

Storage: 4°C, stored under nitrogen

* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (1331.03 mM; Need ultrasonic)

H_2O : 50 mg/mL (665.51 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	13.3103 mL	66.5513 mL	133.1026 mL
5 mM	2.6621 mL	13.3103 mL	26.6205 mL
10 mM	1.3310 mL	6.6551 mL	13.3103 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: ≥ 2.5 mg/mL (33.28 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (33.28 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (33.28 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Thioacetamide (TAA) is an indirect hepatotoxin and causes parenchymal cell necrosis. Thioacetamide requires metabolic activation by microsomal CYP2E1 to thioacetamide-S-oxide initially and then to thioacetamide-S-dioxide, which is a highly reactive metabolite, and its reactive metabolites covalently bind to proteins and lipids thereby causing oxidative stress and centrilobular necrosis. Thioacetamide can induce chronic liver fibrosis, encephalopathy and other events model[1][2][3][4].

In Vitro

Thioacetamide (TAA; 0-10000 μM; 24 h; WB-F344 cells) has cytotoxicity in a concentration-dependent manner[4].

Thioacetamide (TAA; 1000 and 10000 μM; 0-24 h; WB-F344 cells) has differentially-expressed genes in the early phases at low (1000 μM) and high (10000 μM) concentrations[4].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[4]

Cell Line:	WB-F344 cells
Concentration:	0-10000 μM
Incubation Time:	24 hours
Result:	Had 20% and 50% cell death at the 1000 and 10000 μM concentrations, respectively.

In Vivo

β-Amyloid (1-40) can be used in animal modelling to construct models of Alzheimer's disease.

1. Induction of liver tumors[5]

Background

Thioacetamide causes malignant transformation of hepatocytes by inducing DNA damage, cytotoxicity and inflammatory responses.

Specific Mmodeling Methods

Rats: Adult males • 180-200 gDosage: 200 mg/kg • intraperitoneal injection • twice a week for 16 weeks

Note

(1) Prepared by dissolving in 0.9% w/v NaCl for intraperitoneal injection[5].

(2) Inhibitors can be used after 13 weeks[5].

Modeling Indicators

Molecular changes: Increased activity of serum ALT, AST, ALP and GGT increased bilirubin concentration, and decreased serum albumin. Increased levels of Wnt3a and β-catenin, decreased levels of GSK 3β and increased levels of Notch1, Smo and Gli2 in liver tissue[5].

Tissue changes: Liver sections showed multiple cirrhotic nodules, abnormal arrangement of hepatic cords, central veins and portal veins separated by thick fibrous tissue, leukocyte infiltration, vascular congestion, obvious bile, and tubular hyperplasia. Strong blue-stained liver fibrosis was observed around cirrhotic or tumor nodules[5].

Correlated Product(s): /

Opposite Product(s): Saxagliptin (HY-10285)

2. Induction of liver fibrosis model[6]

Background

Thioacetamide is metabolized by the CYP2E1 enzyme system in the liver into active metabolites, which produce ROS, causing oxidative stress, leading to lipid peroxidation of liver cell membranes, damaging liver cells, and the damaged liver cells release pro-inflammatory factors, leading to liver disease.

Specific Mmodeling Methods

Rats: Adult male Wistar rats • 225-250 gAdministration: 150 mg/kg • intraperitoneal injection • twice a week for 12 weeks

Note

(1) 7.5% saline solution was injected intraperitoneally at 2 ml/kg and the model was established eight weeks later[6].

(2) 36 h after the last injection, blood was drawn by cardiac puncture[6].

Modeling Indicators

Molecular changes: Serum ALT, AST, ALP and GGT activities and total bilirubin levels increased significantly, and liver MDA and 4HNE contents increased[6].

Tissue changes: Obvious vacuolar degeneration, bile duct epithelial proliferation and wide fibrous septa appear [6].

Correlated Product(s): Pheneturide (HY-111177)Isoniazid (HY-B0329)

Opposite Product(s): Nilotinib (HY-10159)Imatinib (HY-15463)

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male ICR mice[2]
Dosage:	100 mg/kg
Administration:	Intraperitoneal injection; three times weekly for eight weeks
Result:	Induced chronic liver fibrosis in male ICR mice and resulted in lower body weight, serum cholesterol and triglycerides as well as increased liver size, ALT, AST and LDH values.

Animal Model:	Male C57BL/6 mice (20-25g, aged 8-12 weeks)[3]
Dosage:	200, 600, and 1,200 mg/kg
Administration:	Intraperitoneal injection; once
Result:	Altered the neuropsychiatric state, motor behavior and reflex and sensory functions. Increased in the glutamate release in the cerebral cortex of Hepatic encephalopathy (HE) mice.

REFERENCES

[1]. Wallace MC, et, al. Standard operating procedures in experimental liver research: thioacetamide model in mice and rats. Lab Anim. 2015 Apr;49(1 Suppl):21-9.

[2]. Chen IS, et, al. Hepatoprotection of silymarin against thioacetamide-induced chronic liver fibrosis. J Sci Food Agric. 2012 May;92(7):1441-7.

[3]. Miranda AS, et, al. A thioacetamide-induced hepatic encephalopathy model in C57BL/6 mice: a behavioral and neurochemical study. Arq Neuropsiquiatr. 2010 Aug;68(4):597-602.

[4]. Yeom HJ, et, al. Expression analysis of early response-related genes in rat liver epithelial cells exposed to thioacetamide in vitro. J Vet Med Sci. 2009 Jun;71(6):719-27.

[5]. Ahmed G Abd Elhameed, et al. Saxagliptin defers thioacetamide-induced hepatocarcinogenesis in rats: A novel suppressive impact on Wnt/Hedgehog/Notch1 signaling. Environ Toxicol Pharmacol

[6]. Mohamed E Shaker, et al. Nilotinib counteracts thioacetamide-induced hepatic oxidative stress and attenuates liver fibrosis progression. Fundam Clin Pharmacol. 2011 Apr;25(2):248-57.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA