N-tert-Butyl-α-phenylnitrone

Cat. No.: HY-128463

CAS No.: 3376-24-7

Molecular Formula: $C_{11}H_{15}NO$

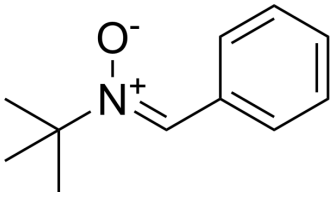
Molecular Weight: 177.24

Target: COX; Reactive Oxygen Species

Pathway: Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (564.21 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	5.6421 mL	28.2103 mL	56.4207 mL
5 mM	1.1284 mL	5.6421 mL	11.2841 mL
10 mM	0.5642 mL	2.8210 mL	5.6421 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 (14.11 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (14.11 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (14.11 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

N-tert-Butyl-α-phenylnitrone is a nitrone-based free radical scavenger that forms nitroxide spin adducts. N-tert-Butyl-α-phenylnitrone inhibits COX2 catalytic activity. N-tert-Butyl-α-phenylnitrone has potent ROS scavenging, anti-inflammatory, neuroprotective, anti-aging and anti-diabetic activities, and can penetrate the blood-brain barrier[1][2][3][4].

IC_{50} & Target		
	COX-2	Reactive oxygen species (ROS)

In Vitro

N-tert-Butyl-α-phenylnitrone (PBN) (25-100 μM) treatment leads to a significant decrease in 2,2'-azobis (2-amidinopropane)

dihydrochloride (AAPH)-induced intracellular ROS accumulation. N-tert-Butyl-α-phenylnitrone also attenuates AAPH-induced cytotoxicity, matrix degradation, and apoptosis. N-tert-Butyl-α-phenylnitrone suppresses AAPH-induced activation of ERK/MAPK pathway. N-tert-Butyl-α-phenylnitrone has the potenial for intervertebral disc degeneration (IDD) research[1].

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

In Vivo

N-tert-Butyl-α-phenylnitrone (PBN; 100 mg/kg; intraperitoneal injection; twice a day; C57Bl/6 mice) treatment not only abolishes the LPS-induced lipid peroxidation, nitrotyrosine residue levels, and GSH depletion, but also decreases the incidence of external malformations[2].

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

Animal Model:	C57Bl/6 mice induced by lipopolysaccharide (LPS)[2]
Dosage:	100 mg/kg
Administration:	Intraperitoneal injection; twice a day (on gestational day 8)
Result:	Abolished LPS-induced lipid peroxidation, nitrotyrosine residues, and GSH depletion.

REFERENCES

[1]. Zhenggang Zhou, et al. PBN Protects NP Cells From AAPH-induced Degenerative Changes by Inhibiting the ERK1/2 Pathway. Connect Tissue Res. 2020 Mar 30;1-10.

[2]. Lei Zhao, et al. Reactive Oxygen Species Contribute to Lipopolysaccharide-Induced Teratogenesis in Mice. Toxicol Sci. 2008 May;103(1):149-57.

[3]. Y Kotake, et al. Inhibition of NF-kappaB, iNOS mRNA, COX2 mRNA, and COX Catalytic Activity by phenyl-N-tert-butylnitrone (PBN). Biochim Biophys Acta. 1998 Nov 19;1448(1):77-84.

[4]. R A Floyd. Antioxidants, Oxidative Stress, and Degenerative Neurological Disorders. Proc Soc Exp Biol Med. 1999 Dec;222(3):236-45.

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