HTHQ

Cat. No.: HY-100768

CAS No.: 148081-72-5

Molecular Formula: $C_{15}H_{24}O_2$

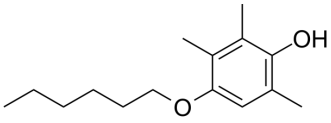
Molecular Weight: 236.35

Target: 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	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (423.10 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.2310 mL	21.1551 mL	42.3101 mL
5 mM	0.8462 mL	4.2310 mL	8.4620 mL
10 mM	0.4231 mL	2.1155 mL	4.2310 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 25 mg/mL (105.78 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (10.58 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (10.58 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

HTHQ (1-O-hexyl-2,3,5-trimethylhydroquinone) is a potent lipophilic phenolic antioxidant. HTHQ has considerable anti-oxidative activity by directly reacting with reactive oxygen species (ROS) and scavenging ROS to form more stable free radicals[1][2].

IC_{50} & Target

ROS[1]

In Vitro

HTHQ (0-100 μM; 24 hours; PC12 cells) treatment increases cell viabilities in a dose-dependent manner[1].

HTHQ (10 μM; 0.6-24 hours; PC12 cells) inhibits 3,4-L-Dihydroxyphenylalanine (L-DOPA) -induced phosphorylation of sustaines extracellular signal-regulated kinases (ERK1/2), p38 mitogen-activated protein kinase (MAPK), and c-Jun N-terminal kinase (JNK1/2). HTHQ also normalizes L-DOPA-reduced Bcl-2-associated death protein (Bad) phosphorylation and Bcl-2-associated X protein (Bax) expression, promoting cell survival[1].

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

Cell Viability Assay[1]

Cell Line:	PC12 cells
Concentration:	0 μM, 1 μM, 10 μM, and 100 μM
Incubation Time:	24 hours
Result:	Reduced cell viability at 24 hours caused by both 100 and 200 μM L-DOPA was significantly attenuated.

Western Blot Analysis[1]

Cell Line:	PC12 cells
Concentration:	10 μM
Incubation Time:	0.6-24 hours
Result:	Inhibited L-DOPA-induced phosphorylation of sustained extracellular signal-regulated kinases (ERK1/2), p38 mitogen-activated protein kinase (MAPK), and c-Jun N-terminal kinase (JNK1/2). And also normalized L-DOPA-reduced Bcl-2-associated death protein (Bad) phosphorylation and Bcl-2-associated X protein (Bax) expression.

In Vivo

HTHQ (50-200 mg/kg; oral administration; for 4 weeks; specific pathogen-free male Sprague Dawley rats) treatment significantly improves liver weight and serum chemistry level. HTHQ reduces hydroxyproline and malondialdehyde level in the liver. HTHQ treatment also reduces fibrotic septa. HTHQ administration shows reduced mRNA level of PDGF (Plateletderived growth factor) , α-SMA (α-smooth muscle actin) and TGF-β (transforming growth factor-β) than DMN-induced hepetic fibrosis animals in the liver tissue[2].

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

Animal Model:	48 specific pathogen-free male Sprague Dawley (SD) rats (6-week-old) with dimethylnitrosamine (DMN) [2]
Dosage:	50 mg/kg, 100 mg/kg, 200 mg/kg
Administration:	Oral administration; for 4 weeks
Result:	Improved against DMN-induced liver fibrosis in male SD rats.

CUSTOMER VALIDATION

- J Cell Mol Med. 2020 Sep;24(18):10468-10477.
- Mercer University. June, 2021.

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REFERENCES

[1]. Park HJ, et al. 1-O-Hexyl-2,3,5-Trimethylhydroquinone Ameliorates l-DOPA-Induced Cytotoxicity in PC12 Cells. Molecules. 2019 Mar 1;24(5). pii: E867.

[2]. Jung YR et al. Inhibitory Effect of 1-O-Hexyl-2,3,5-Trimethylhydroquinone on Dimethylnitrosamine-induced Liver Fibrosis in Male SD Rats. Toxicol Res. 2010 Sep;26(3):193-201.

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