TBHQ

Cat. No.: HY-100489

CAS No.: 1948-33-0

Molecular Formula: $C_{10}H_{14}O_2$

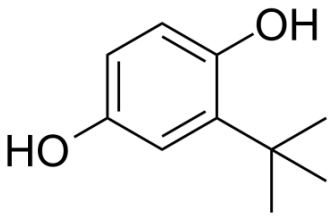
Molecular Weight: 166.22

Target: Keap1-Nrf2; ERK; Autophagy; Apoptosis; Ferroptosis

Pathway: NF-κB; MAPK/ERK Pathway; Stem Cell/Wnt; Autophagy; Apoptosis

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 : ≥ 56.66 mg/mL (340.87 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	6.0161 mL	30.0806 mL	60.1612 mL
5 mM	1.2032 mL	6.0161 mL	12.0322 mL
10 mM	0.6016 mL	3.0081 mL	6.0161 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 (15.04 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (15.04 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (15.04 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

TBHQ (tert-Butylhydroquinone) is a widely used Nrf2 activator, protects against Doxorubicin (DOX)-induced cardiotoxicity through activation of Nrf2[1]. TBHQ (tert-Butylhydroquinone) is also an ERK activator; rescues Dehydrocorydaline (DHC)-induced cell proliferation inhibitionin melanoma[2].

IC_{50} & Target			
	Nrf2	ERK	Autophagy

In Vitro

TBHQ (t-butylhydroquinone; tBHQ; 0-100 μM; 48 hours; H9c2 cells) alone does not affect H9c2 cells viability. Pre-incubation of the H9c2 cells with various concentrations of tBHQ for 24 hours enhances cell viability which is decreased due to exposure to ethanol in a dose-dependent manner. Treatment with tBHQ markedly enhances the viability of H9c2 cardiomyocytes exposed to ethanol[3].

TBHQ (5 μM; 15 min; H9c2 cells) treatment significantly reduces the amount of apoptotic cells exposed to ethanol[3].

TBHQ (5 μM; H9c2 cells) pre-treatment markedly inhibites the ethanol-induced increase in caspase-3 and Bax expression, and enhances Bcl-2 expression[3].

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

Cell Viability Assay[3]

Cell Line:	H9c2 cells
Concentration:	0 μM, 0.625 μM, 1.25 μM, 2.5 μM, 5 μM, 10 μM, 20 μM, 50 μM and 100 μM
Incubation Time:	48 hours
Result:	Enhanced the viability of H9c2 cardiomyocytes exposed to ethanol.

Apoptosis Analysis[3]

Cell Line:	H9c2 cells
Concentration:	5 μM
Incubation Time:	
Result:	Lowered the amount of apoptotic cells exposed to ethanol.

Western Blot Analysis[3]

Cell Line:	H9c2 cells
Concentration:	5 μM
Incubation Time:	
Result:	Inhibited the ethanol-induced increase in caspase-3 and Bax expression, and enhanced Bcl-2 expression.

In Vivo

TBHQ treatment (50 mg/kg; Intraperitoneal injection; three injections at intervals of 8 h that began 1-h post ICH; CD-1 mice) augments the DNA-Binding activity of Nrf2, attenuates oxidative brain damage and acute neurological deficits afterintracerebral hemorrhage (ICH), attenuates microglial activation with concomitant reduction in the release of proinflammatory cytokine interleukin-1β (IL-1β). TBHQ has the efficacy of post-injury administration in attenuating acute neurological injury after ICH[4].

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

Animal Model:	Male CD-1 mice (8-10 weeks old)[4]
Dosage:	50 mg/kg
Administration:	Intraperitoneal injection; three injections at intervals of 8 hours that began 1h post ICH.
Result:	The treatment augmented the DNA-binding activity of Nrf2, attenuated brain oxidative damage, attenuated the microglial activation and the expression of IL-1β.

CUSTOMER VALIDATION

- Proc Natl Acad Sci U S A. 2019 Feb 19;116(8):2996-3005.
- Theranostics. 2021 Jan 1;11(2):861-877.
- J Neuroinflammation. 2019 Oct 4;16(1):185.
- Oxid Med Cell Longev. 2020 May 13;2020:3145182.
- Biochem Pharmacol. 2020 Mar;173:113673.

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REFERENCES

[1]. Lin-Feng Wang, et al. Tert-butylhydroquinone ameliorates doxorubicin-induced cardiotoxicity by activating Nrf2 and inducing the expression of its target genes. Am J Transl Res. 2015; 7(10): 1724–1735.

[2]. Hu H, et al. Dehydrocorydaline inhibits cell proliferation, migration and invasion via suppressing MEK1/2-ERK1/2 cascade in melanoma.Onco Targets Ther. 2019 Jul 2;12:5163-5175.

[3]. XIAOJING SHI, et al. Tert-butylhydroquinone attenuates the ethanol-induced apoptosis of and activates the Nrf2 antioxidant defense pathway in H9c2 cardiomyocytes.Int J Mol Med. 2016 Jul; 38(1): 123–130.

[4]. Sukumari-Ramesh S, et al. Post-Injury Administration of Tert-butylhydroquinone Attenuates Acute Neurological Injury AfterIntracerebral Hemorrhage in Mice.J Mol Neurosci. 2016 Apr;58(4):525-31.

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