Carboxy-PTIO potassium

Cat. No.: HY-18734A

CAS No.: 148819-94-7

Molecular Formula: $C_{14}H_{16}KN_2O_4$

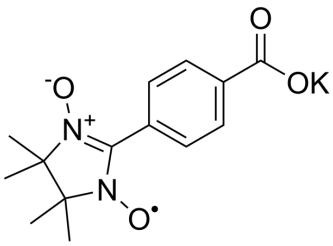
Molecular Weight: 315.39

Target: NO Synthase

Pathway: Immunology/Inflammation

Storage: -20°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

H_2O : 25 mg/mL (79.27 mM; ultrasonic and warming and heat to 60°C)

DMSO : 10 mg/mL (31.71 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.1707 mL	15.8534 mL	31.7068 mL
5 mM	0.6341 mL	3.1707 mL	6.3414 mL
10 mM	0.3171 mL	1.5853 mL	3.1707 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 50 mg/mL (158.53 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Carboxy-PTIO potassium is a potent nitric oxide (NO) scavenger that can make a quick reaction with NO to produce NO_2. Carboxy-PTIO potassium can prevent hypotension and endotoxic shock through the direct scavenging action against NO in lipopolysaccharide-stimulated rat model[1][2][3].

In Vitro

Carboxy-PTIO potassium (200 μM; 1 h prior to physalin A; 24 hours) significantly suppresses the stimulation of NO expression induced by Physalin A (HY-N9942) treatment, but no change is observed in Carboxy-PTIO treatment alone[1].

Carboxy-PTIO potassium (200 μM; 1 h prior to physalin A; 24 hours) reduces Physalin A-induced cleavage of procaspase-3 and PARP, down-regulated ICAD expression,diminishing DNA fragmentation in nuclei[1].

Carboxy-PTIO potassium (200 μM; 1 h prior to Physalin A; 24 hours) shows no effect on iNOS expression. However, decreased-mTOR and p-mTOR levels induced by Physalin A is reversed by Carboxy-PTIO with concomitant suppression of LC3 I to LC3 II conversions in A375-S2 cells [1].

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

Western Blot Analysis[1]

Cell Line:	A375-S2 cells
Concentration:	200 μM
Incubation Time:	1 h prior to physalin A; 24 hours
Result:	Diminished physalin A-induced procaspase-3 and PARP cleavage.

In Vivo

Carboxy-PTIO (intravenous?injection; 0.056-1.70 mg/kg/min; infused for 1 hr beginning 90 min after the LPS injection 90 min) treatment improves the hypotension, renal dysfunction and survival rate in Lps-treated rats. But it does not affect each parameter in normal rats[3].

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

Animal Model:	SD rats[3]
Dosage:	0.056-1.70 mg/kg/min
Administration:	Intravenous injection; 0.056-1.70 mg/kg/min; infused for 1 hr beginning 90 min after the LPS injection 90 min
Result:	Exhibited a potent therapeutic value in endotoxin shock through the direct scavenging action against NO.

CUSTOMER VALIDATION

- Antioxid Redox Signal. 2022 Oct 14.
- iScience. 2023 Jul 7.

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REFERENCES

[1]. Hao He, et al.Nitric oxide induces apoptosis and autophagy; autophagy down-regulates NO synthesis in physalin A-treated A375-S2 human melanoma cells.Food Chem Toxicol. 2014 Sep;71:128-35.

[2]. T Akaike, et al. Antagonistic action of imidazolineoxyl N-oxides against endothelium-derived relaxing factor/.NO through a radical reaction. Biochemistry. 1993 Jan 26;32(3):827-32.

[3]. M Yoshid, et al. Therapeutic effects of imidazolineoxyl N-oxide against endotoxin shock through its direct nitric oxide-scavenging activity. Biochem Biophys Res Commun. 1994 Jul 29;202(2):923-30.

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