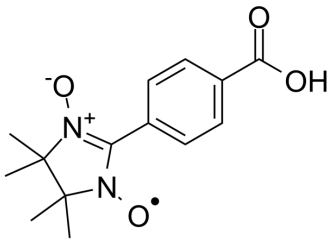
Carboxy-PTIO

Cat. No.:	HY-18734
CAS No.:	145757-47-7
Molecular Formula:	$C_{14}H_{17}N_2O_4$
Molecular Weight:	277.3
Target:	NO Synthase
Pathway:	Immunology/Inflammation
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Carboxy-PTIO is a potent nitric oxide (NO) scavenger that can make a quick reaction with NO to produce NO_2. Carboxy-PTIO 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 (200 μM; 1 h prior to physalin A; 24 hours) significantly suppresses the stimulation of NO expression induced by physalin A treatment, but no change is observed in Carboxy-PTIO treatment alone[1].
Carboxy-PTIO (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 (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.

Cell Viability Assay[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 naomal rats[3].

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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.

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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