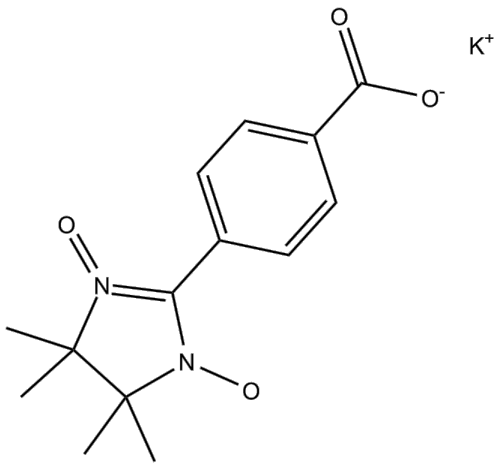
Product Data Sheet

Product Name: Carboxy-PTIO, potassium salt
Cat. No.: GC16211

Chemical Properties

Cas. No.	148819-94-7		
Chemical Name	2-(4-carboxyphenyl)-4,5-dihydro-4,4,5,5-tetramethyl-1H-imidazolyl-1-oxy-3-oxide, mono potassium salt		
SMILES	CC(C1(C)C)(N(=C(N1[O])C2=CC=C(C([O-])=O)C=C2)=O)C.[K+]		
Formula	$C_{14}H_{16}KN_2O_4$	M.Wt	315.38
Solubility	>1.4 mg/ml in DMSO, >3.3 mg/ml in DMF, >1.6mg/ml in Ethanol	Storage	Desiccate at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure



Protocol

Cell experiment [1]:

Cell lines A375-S2 cells

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Preparation Method	A375-S2 cells were pre-treated with 1400W (200μM) or Carboxy-PTIO potassium salt (200μM) for 1 h prior to 15μM physalin A treatment for 24 h.
Reaction Conditions	200μM; 1 h
Applications	The inhibitor of nitric oxide synthase 1400W and NO scavenger Carboxy-PTIO potassium salt significantly suppressed the stimulation of NO expression induced by physalin A treatment.

Animal experiment [2]:

Animal models	C57BL/10J mice
Preparation Method	animals was assigned to receive Carboxy-PTIO potassium salt (0.3-1.2 mg/kg; i.p.) or the same volume of saline at the time of MCA occlusion. The right femoral artery was cannulated for measuring arterial blood gases,glucose, hematocrit, and blood pressure before and 45 min after the induction of cerebral ischemia.
Dosage form	0.3-1.2 mg/kg；i.p.
Applications	Mice treated with Carboxy-PTIO potassium salt had dose-specifically reduced brain infarction, significantly by 27-30%, even when therapy was delayed up to 4 h after the ischemic insult.

Caution: Product has not been fully validated for medical applications. For research use only.
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References:

[1] He H, Feng Y S, Zang L H, et al. Nitric oxide induces apoptosis and autophagy; autophagy down-regulates NO synthesis in physalin A-treated A375-S2 human melanoma cells[J]. Food and chemical toxicology, 2014, 71: 128-135.
[2] Lee E J, Hung Y C, Chen H Y, et al. Delayed treatment with carboxy-PTIO permits a 4-h therapeutic window of opportunity and prevents against ischemia-induced energy depletion following permanent focal cerebral ischemia in mice[J]. Neurochemical research, 2009, 34: 1157-1166.

Background

Carboxy-PTIO potassium salt is a nitric oxide (NO) scavenger[1]. Carboxy-PTIO potassium salt can react stoichiometrically with NO and can be used for EPR detection of NO[2].

In vitro, pretreatment of A375-S2 cells with Carboxy-PTIO potassium salt (200μM) for 1h significantly inhibited physalin A-stimulated NO expression, reduced procaspase-3 and PARP cleavage, reversed the decrease in mTOR and p-mTOR levels, and also inhibited in the cells Conversion of LC3 I to LC3 II [3]. Pretreating HUVEC cells with Carboxy-PTIO potassium salt (100μM) for 6 hours significantly enhanced the inhibitory effect of IL-1β on PGIS [4]. Carboxy-PTIO potassium salt (0.5μM, 1μM) pretreated MDA-MB-231 cells for 1 hour, which significantly reduced the inhibitory activity of compounds 3a-f on cells [5].

In vivo, intravenous injection of Carboxy-PTIO potassium salt (0.3-1.2 mg/kg) in mice with cerebral ischemic injury resulted in a dose-dependent reduction in the incidence of cerebral infarction by 27% even when treatment was delayed for up to 4 hours. -30%[6]. Carboxy-PTIO potassium salt (0.056-1.70mg/kg/min) was injected intravenously for 60 minutes to treat endotoxic shock rats, which significantly improved the symptoms of hypotension and renal insufficiency in rats and avoided the shock state [7].

References:
[1]Pfeiffer S, Leopold E, Hemmens B, et al. Interference of carboxy-PTIO with nitric oxide-and peroxynitrite-mediated reactions[J]. Free Radical Biology and Medicine, 1997, 22(5): 787-794.
[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.

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[3] He H, Feng Y S, Zang L H, et al. Nitric oxide induces apoptosis and autophagy; autophagy down-regulates NO synthesis in physalin A-treated A375-S2 human melanoma cells[J]. Food and chemical toxicology, 2014, 71: 128-135.
[4] Camacho M, LÓpez-Belmonte J, Vila L. Rate of vasoconstrictor prostanoids released by endothelial cells depends on cyclooxygenase-2 expression and prostaglandin I synthase activity[J]. Circulation research, 1998, 83(4): 353-365.
[5] Kang F, Zhu J, Wu J, et al. O 2-3-Aminopropyl diazeniumdiolates suppress the progression of highly metastatic triple-negative breast cancer by inhibition of microvesicle formation via nitric oxide-based epigenetic regulation[J]. Chemical Science, 2018, 9(34): 6893-6898.
[6]Lee E J, Hung Y C, Chen H Y, et al. Delayed treatment with carboxy-PTIO permits a 4-h therapeutic window of opportunity and prevents against ischemia-induced energy depletion following permanent focal cerebral ischemia in mice[J]. Neurochemical research, 2009, 34: 1157-1166.
[7]Yoshida M, Akaike T, Wada Y, et al. Therapeutic effects of imidazolineoxyl N-oxide against endotoxin shock through its direct nitric oxide-scavenging activity[J]. Biochemical and biophysical research communications, 1994, 202(2): 923-930.

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