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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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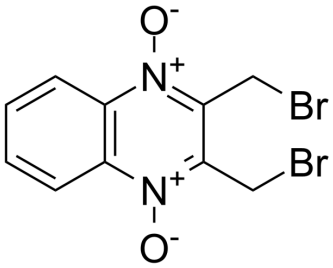
Conoidin A

Cat. No.:	HY-116090
CAS No.:	18080-67-6
Molecular Formula:	$C_{10}H_8Br_2N_2O_2$
Molecular Weight:	347.99
Target:	Parasite
Pathway:	Anti-infection
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 : 14.29 mg/mL (41.06 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.8736 mL	14.3682 mL	28.7365 mL
	5 mM	0.5747 mL	2.8736 mL	5.7473 mL
	10 mM	0.2874 mL	1.4368 mL	2.8736 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: ≥ 1.43 mg/mL (4.11 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Conoidin A is a cell permeable inhibitor of ***T. gondii*** enzyme peroxiredoxin II (TgPrxII) with nematicidal properties. Conoidin A covalently binds to the peroxidatic Cys47 of TgPrxII, irreversibly inhibiting its hyperperoxidation activity with an IC_{50} of 23 μM. Conoidin A also inhibits hyperoxidation of mammalian PrxI and PrxII (but not PrxIII)[1][2]. Conoidin A has antioxidant, neuroprotective effects and can be used for the research of ischaemic heart disease[3].

IC_{50} & Target

Toxoplasma

In Vitro

Peroxiredoxins are a widely conserved family of enzymes that function in antioxidant defense and signal transduction. And the changes in PrxII expression are associated with a variety of human diseases, including cancer[1].
Conoidin A binds to the peroxidatic cysteine of TgPrxII, inhibiting its enzymatic activity in vitro. Conoidin A also shown to alkylate or crosslink catalytic cysteines of wild type AcePrx-1 in Ancylostoma ceylanicum and human PrxII and PrxIV with similar efficiency. But it is ineffective to mitochondrial hPrxIII[2].

	Conoidin A (5 μM) can inhibit the glucose oxidase-mediated hyperoxidation of mammalian peroxiredoxin I and II[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Conoidin A (intraperitoneal injection; 5 mg/kg; for three successive days before MI/R injury) blocks the effect of Luteolin (HY-N0162) on the ST□segment elevation. Furthermore, an increase in the infarct size presented of the MI/R group can be reduced by Luteolin. But pre□treatment with conoidin A abolishs the effect of Luteolin. Pre□treatment with conoidin A also prevents Luteolin-reduced activities of CK□MB, AST and LDH in vivo[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Rat myocardial I/R model[3]
Dosage:	5 mg/kg
Administration:	Intraperitoneal injection; 5 mg/kg; for three successive days before MI/R injury
Result:	Significantly reversed the antioxidative effect of Luteolin. Impaired the protective effects of luteolin.

REFERENCES

[1]. Jeralyn D Haraldsen, et al. IDENTIFICATION OF CONOIDIN A AS A COVALENT INHIBITOR OF PEROXIREDOXIN II. Org Biomol Chem. 2009;7:3040-3048.

[2]. Gu Liu, et al. Optimisation of conoidin A, a peroxiredoxin inhibitor. ChemMedChem. 2010 Jan;5(1):41-5.

[3]. Bo Wei, et al. Luteolin ameliorates rat myocardial ischaemia-reperfusion injury through activation of peroxiredoxin II. Br J Pharmacol

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