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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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Cariporide

Cat. No.: HY-19693

CAS No.: 159138-80-4

Molecular Formula: $C_{12}H_{17}N_3O_3S$

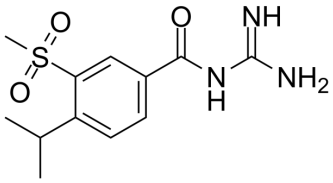
Molecular Weight: 283.35

Target: Na+/H+ Exchanger (NHE)

Pathway: Membrane Transporter/Ion Channel

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (352.92 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.5292 mL	17.6460 mL	35.2920 mL
5 mM	0.7058 mL	3.5292 mL	7.0584 mL
10 mM	0.3529 mL	1.7646 mL	3.5292 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 (8.82 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.82 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.82 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Cariporide (HOE-642) is a selective Na^+/H^+ exchange inhibitor.

In Vitro

Cariporide significantly suppresses markers of cell death, such as TUNEL positivity and caspase-3 cleavage, at 8 or 16 hours. Cariporide remarkably suppresses cytosolic Na^+ and Ca^{2+} accumulation. Cariporide prevents mitochondrial membrane potential loss induced by $H^{2+}O^{2+}$[1]. Cariporide (HOE-642) ameliorates myocardial ischemia/reperfusion injury, by the well-established reduction of cytosolic Ca^{2+} in cardiac myocytes through inhibition of Na^+/H^+ exchange[2]. Cariporide (HOE-642), has inhibitory effects on the degranulation of human platelets, the formation of platelet–leukocyte-aggregates, and the

	activation of the GPIIb/IIIa receptor (PAC-1)[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Intravenous administration of cariporide significantly decreases brain Na^+ uptake and reduces cerebral edema, brain swelling, and infarct volume[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	Neonatal rat cardiomyocytes are randomly separated into groups: (1) control group, (2) incubation with 100 μM hydrogen peroxide, or (3) pretreatment with 10 μM cariporide for 20 minutes followed by 100 μM hydrogen peroxide. Caspase-3 activity is measured by detection of the cleavage of a colorimetric caspase-3 substrate, N-acetyl-Asp-Glu-Val-Asp-p-nitroaniline, using an assay kit[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [4]	Rats: Cariporide and/or bumetanide are administered intravenously (15 or 30 mg/kg in 2 to 4 doses, respectively, of 7.5 mg/kg) starting at 20 minutes before initiation of pMCAO. For neurologic outcome experiments, some rats are given cariporide and/or bumetanide by a single intraperitoneal injection[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Acta Pharm Sin B. 5 September 2022.
- JCI Insight. 2023 Aug 29;e170928.
- Hum Reprod. 2024 Feb 14:deae020.
- Am J Physiol Heart Circ Physiol. 2023 Dec 15.
- FASEB J. 2019 Jun;33(6):7202-7212.

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REFERENCES

[1]. Teshima Y, et al. Cariporide (HOE642), a selective Na+-H+ exchange inhibitor, inhibits the mitochondrial death pathway. Circulation. 2003 Nov 4;108(18):2275-81.

[2]. Chang HB, et al. Na(+)/H(+) exchanger in the regulation of platelet activation and paradoxical effects of cariporide. Exp Neurol. 2015 Oct;272:11-6.

[3]. O'Donnell ME, et al. Intravenous HOE-642 reduces brain edema and Na uptake in the rat permanent middle cerebral artery occlusion model of stroke: evidence for participation of the blood-brain barrier Na/H exchanger. J Cereb Blood Flow Metab. 2013 Feb;33(2):225-34.

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