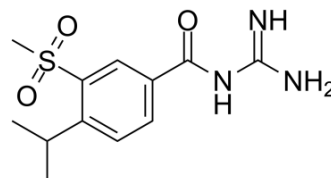


Cariporide

Cat. No.:	HY-19693		
CAS No.:	159138-80-4		
Molecular Formula:	C ₁₂ H ₁₇ N ₃ O ₃ S		
Molecular Weight:	283.35		
Target:	Sodium Channel		
Pathway:	Membrane Transporter/Ion Channel		
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 : ≥ 100 mg/mL (352.92 mM)
 * "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent Concentration	Mass		
		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

- 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
- 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
- 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 Ca²⁺ accumulation. Cariporide prevents mitochondrial membrane potential loss induced by H₂O₂^[1]. Cariporide (HOE-642) ameliorates myocardial ischemia/reperfusion injury, by the well-established reduction of cytosolic Ca²⁺ 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

	<p>activation of the GPIIb/IIIa receptor (PAC-1)^[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
In Vivo	<p>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.</p>

PROTOCOL

Cell Assay ^[1]	<p>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.</p>
Animal Administration ^[4]	<p>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.</p>

CUSTOMER VALIDATION

- 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.

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

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