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Produktinformation

- Forschungsprodukte & Biochemikalien
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- Diagnostik & molekulare Diagnostik
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See the following pages for more information!

Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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OR-1896

Cat. No.: HY-135746

CAS No.: 220246-81-1

Molecular Formula: $C_{13}H_{15}N_3O_2$

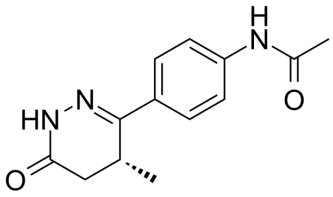
Molecular Weight: 245.28

Target: Phosphodiesterase (PDE); Potassium Channel; Drug Metabolite; Apoptosis

Pathway: Metabolic Enzyme/Protease; Membrane Transporter/Ion Channel; Apoptosis

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 : 62.5 mg/mL (254.81 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.0770 mL	20.3849 mL	40.7697 mL
5 mM	0.8154 mL	4.0770 mL	8.1539 mL
10 mM	0.4077 mL	2.0385 mL	4.0770 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: ≥ 2.08 mg/mL (8.48 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (8.48 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (8.48 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

OR-1896 is an active long-lived metabolite of Levosimendan. OR-1896 is a highly selective phosphodiesterase (PDE) III isoform inhibitor and a powerful vasodilator. OR-1896 can open ATP-sensitive K^+ channels and has Ca^{2+}-sensitizing effect. OR-1896 mitigates cardiomyocyte apoptosis, cardiac remodeling and myocardial inflammation[1].

IC_{50} & Target			
	PDE3/PDE ⊠	K+ Channel	Drug Metabolite

In Vitro

There are many evidences has accumulated and revealed a variety of beneficial pleiotropic effects OR-1896. OR-1896 evokes

	prominent vasodilatory responses, activation of ATP-sensitive sarcolemmal K^+ channels of smooth muscle cells appears as a powerful vasodilator mechanism. Additionally, activation of ATP-sensitive K^+ channels in the mitochondria potentially extends the range of cellular actions towards the modulation of mitochondrial ATP production and implicates a pharmacological mechanism for cardioprotection[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	During the metabolism of Levosimendan approximately 5% of the drug is converted to the metabolite OR-1855 in the large intestine, and then acetylated in the liver to form the active metabolite OR-1896. Binding to plasma proteins is 98% for Levosimendan but only 40% for OR-1896. Unlike Levosimendan, which has an elimination half-life of 1-1.5 h, the half-life of OR-1896 is about 75 to 80 h allowing cardiovascular effects to persist up to 7 to 9 days after discontinuation of a 24-hour infusion of levosimendan. The pharmacokinetic of the parent drug is unaltered in subjects with severe renal impairment or with moderate hepatic impairment, whereas the elimination of its metabolites (OR-1896) can be prolonged[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Pharmaceuticals. 2023 May 30, 16(6), 815.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Papp, Z, et al., Levosimendan: molecular mechanisms and clinical implications: consensus of experts on the mechanisms of action of levosimendan. Int J Cardiol. 2012 Aug 23;159(2):82-7.

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