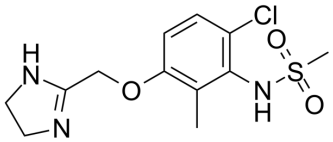
Dabuzalgron

Cat. No.:	HY-117071
CAS No.:	219311-44-1
Molecular Formula:	$C_{12}H_{16}ClN_3O_3S$
Molecular Weight:	317.79
Target:	Adrenergic Receptor; Apoptosis
Pathway:	GPCR/G Protein; Neuronal Signaling; Apoptosis
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 : 26 mg/mL (81.82 mM; Need ultrasonic)

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	3.1467 mL	15.7337 mL	31.4673 mL
5 mM	0.6293 mL	3.1467 mL	6.2935 mL
10 mM	0.3147 mL	1.5734 mL	3.1467 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 (6.55 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (6.55 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (6.55 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Dabuzalgron (Ro 115-1240) is an orally active and selective α-1A adrenergic receptor agonist for the treatment of urinary incontinence. Dabuzalgron protects against Doxorubicin-induced cardiotoxicity by preserving mitochondrial function[1].

IC_{50} & Target

α-1A adrenergic receptor[1]

In Vitro

Dabuzalgron treatment increases ERK phosphorylation in a dose-dependent fashion with an EC_{50} of 4.8 μM. ERK1/2 activation contributes to the cardioprotective effects of Dabuzalgron[1].

Dabuzalgron (10 μM; 4 hours) protects NRVMs from cell death due to Doxorubicin (DOX)[1].
Activation of the α1A-AR with Dabuzalgron (10 μM; 4 hours) mitigates the detrimental effects of DOX on mitochondrial membrane potential and abrogates the activation of important elements of the apoptotic response to mitochondrial damage[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	Neonatal rat ventricular myocytes (NRVMs)
Concentration:	0.1 μM, 1 μM, 10 μM and 100 μM
Incubation Time:	15 minutes
Result:	Increased ERK phosphorylation in a dose-dependent fashion with an EC_{50} of 4.8 μM.

In Vivo

Dabuzalgron (10 μg/kg; oral gavage; twice daily; for 7 days; C57Bl6J wild-type or α1A-AR knockout mice) treatment protects against DOX cardiotoxicity by activating the α1A-AR. Dabuzalgron protects against the reduction in transcripts related to mitochondrial function, up-regulates PGC1α, preserves ATP content, and reduces oxidative stress in the hearts of mice treated with DOX[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male C57Bl6J wild-type (WT) or α1A-AR knockout (AKO) mice (8-12-week-old) injected with Doxorubicin (DOX)[1]
Dosage:	10 μg/kg
Administration:	Oral gavage; twice daily; for 7 days
Result:	Preserved contractile function and reduced fibrosis after DOX administration. AKO mice treated with DOX had worse survival and more profoundly impaired contractile function than WT mice. Protected against the reduction in transcripts related to mitochondrial function, preserved ATP content, and reduced oxidative stress in the hearts of mice treated with DOX.

REFERENCES

[1]. Beak J, et al. An Oral Selective Alpha-1A Adrenergic Receptor Agonist Prevents Doxorubicin Cardiotoxicity. JACC Basic Transl Sci. 2017 Feb;2(1):39-53.

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