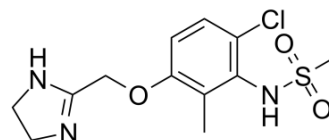


Dabuzalgron

Cat. No.:	HY-117071		
CAS No.:	219311-44-1		
Molecular Formula:	C ₁₂ H ₁₆ ClN ₃ O ₃ S		
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)			
		Solvent Concentration	Mass	
			1 mg	5 mg
	Preparing Stock Solutions	1 mM	3.1467 mL	15.7337 mL
		5 mM	3.1467 mL	6.2935 mL
		10 mM	0.3147 mL	1.5734 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 μ M; 4 hours) protects NRVMs from cell death due to Doxorubicin (DOX)^[1].

Activation of the α 1A-AR with Dabuzalgron (10 μ M; 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 μ M, 1 μ M, 10 μ M and 100 μ M
Incubation Time:	15 minutes
Result:	Increased ERK phosphorylation in a dose-dependent fashion with an EC ₅₀ of 4.8 μ M.

In Vivo

Dabuzalgron (10 μ g/kg; oral gavage; twice daily; for 7 days; C57Bl6J wild-type or α 1A-AR knockout mice) treatment protects against DOX cardiotoxicity by activating the α 1A-AR. Dabuzalgron protects against the reduction in transcripts related to mitochondrial function, up-regulates PGC1 α , 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 α 1A-AR knockout (AKO) mice (8-12-week-old) injected with Doxorubicin (DOX) ^[1]
Dosage:	10 μ g/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 α 1A-Adrenergic Receptor Agonist Prevents Doxorubicin Cardiotoxicity. JACC Basic Transl Sci. 2017 Feb;2(1):39-53.

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

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