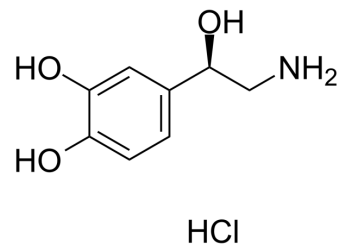


Norepinephrine hydrochloride

Cat. No.:	HY-13715A
CAS No.:	329-56-6
Molecular Formula:	C ₈ H ₁₂ ClNO ₃
Molecular Weight:	205.64
Target:	Adrenergic Receptor; Autophagy; Endogenous Metabolite
Pathway:	GPCR/G Protein; Neuronal Signaling; Autophagy; Metabolic Enzyme/Protease
Storage:	4°C, sealed storage, away from moisture * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro	H ₂ O : 100 mg/mL (486.29 mM; Need ultrasonic)				
	DMSO : 44 mg/mL (213.97 mM; Need ultrasonic)				
		Solvent Concentration	Mass		
	Preparing Stock Solutions	1 mM	1 mg	5 mg	10 mg
		5 mM	4.8629 mL	24.3143 mL	48.6287 mL
10 mM		0.9726 mL	4.8629 mL	9.7257 mL	
	10 mM	0.4863 mL	2.4314 mL	4.8629 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 (10.11 mM); Clear solution 2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.08 mg/mL (10.11 mM); Clear solution 3. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.08 mg/mL (10.11 mM); Clear solution				

BIOLOGICAL ACTIVITY

Description	Norepinephrine hydrochloride (Levarterenol hydrochloride) is a β ₁ -selective adrenergic receptor agonist with EC ₅₀ of 5.37 μM ^[1] .	
IC₅₀ & Target	β ₁ adrenoceptor 5.37 μM (EC ₅₀)	Human Endogenous Metabolite
In Vitro	Norepinephrine (NE) bitartrate monohydrate is generally considered to be a β ₁ -subtype selective adrenergic agonist. Norepinephrine(NE) also has direct activity at the β ₂ -adrenoceptor in higher concentrations ^[1] . Adipocytes from the inguinal	

fat pad (iWA) or the interscapular fat pad (BA) are isolated from neonatal wild-type C57BL/6J mice and cultured. To examine the effect of activating AT2 upon β -adrenergic signaling, cAMP production is first assessed in response to Norepinephrine (NE, 10 μ M) with or without CGP (10 nM) co-treatment. Norepinephrine (NE) increases cAMP as expected in iWA, and CGP does not alter this effect. Norepinephrine (NE) is also known to induce lipolysis, and liberated fatty acids are required to functionally activate UCP1 protein and to stimulate heat production. CREB phosphorylation at Ser133 is increased after Norepinephrine (NE) treatment and significantly attenuated with CGP co-treatment in mouse iWA^[2].

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

PROTOCOL

Cell Assay ^[2]

Subcutaneous preadipocytes derived from a 38-year old non-diabetic female donor are immortalized with TERT and HPV E6/E7. For the current studies, a stable diploid clone (referred to as clone B) with consistent differentiation capacity is isolated by ring cloning. Cells are grown in preadipocyte PGM2 media. Once cells are confluent, differentiation is induced by incubation in differentiation media consisting of dexamethasone, IBMX, indomethacin, and additional insulin. Cells are differentiated for 10 days. Prior to treatment, media is replaced with PGM2 media for one day and then switched to serum-free media overnight for treatments. Adipocytes are treated for 6 hours with vehicle, Norepinephrine (NE, 10 μ M), CGP (10 nM), or Norepinephrine (NE) and CGP^[2].

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

CUSTOMER VALIDATION

- Cardiovasc Res. 2018 Feb 1;114(2):300-311.
- Cell Rep. 2019 Dec 3;29(10):2936-2943.e4.
- Br J Pharmacol. 2020 Aug;177(15):3389-3402.
- Cell Death Dis. 2020 Aug 18;11(8):644.
- FASEB J. 2020 Nov;34(11):14892-14904.

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REFERENCES

[1]. MacGregor DA, et al. Relative efficacy and potency of beta-adrenoceptor agonists for generating cAMP in human lymphocytes. Chest. 1996 Jan;109(1):194-200.

[2]. Littlejohn NK, et al. Suppression of Resting Metabolism by the Angiotensin AT2 Receptor. Cell Rep. 2016 Aug 9;16(6):1548-60.

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

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