Norepinephrine bitartrate monohydrate

Cat. No.: HY-13715B

CAS No.: 108341-18-0

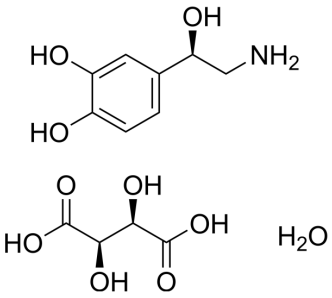
Molecular Formula: $C_{12}H_{19}NO_{10}$

Molecular Weight: 337.28

Target: Adrenergic Receptor; Autophagy; Endogenous Metabolite

Pathway: GPCR/G Protein; Neuronal Signaling; Autophagy; Metabolic Enzyme/Protease

Storage: 4°C, protect from light, stored under nitrogen
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light, stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 50 mg/mL (148.24 mM; Need ultrasonic)
DMSO : ≥ 30 mg/mL (88.95 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.9649 mL	14.8245 mL	29.6490 mL
5 mM	0.5930 mL	2.9649 mL	5.9298 mL
10 mM	0.2965 mL	1.4824 mL	2.9649 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 150 mg/mL (444.73 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (6.17 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (6.17 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (6.17 mM); Clear solution

BIOLOGICAL ACTIVITY

Description Norepinephrine (Levarterenol; L-Noradrenaline) bitartrate monohydrate is a potent adrenergic receptor (AR) agonist. Norepinephrine activates α_1, α_2, β_1 receptors[1][2][3][4].

IC_{50} & Target				
	α1-adrenergic receptor	α2-adrenergic receptor	Beta-1 adrenergic receptor	Microbial Metabolite

	Human Endogenous Metabolite
In Vitro	Norepinephrine (Levarterenol; L-Noradrenaline) bitartrate monohydrate is generally considered to be a β_1-subtype selective adrenergic agonist over β_2-adrenoceptor. Norepinephrine(NE) bitartrate monohydrate also has direct activity at the β_2-adrenoceptor in higher concentrations[2]. 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 μM) 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[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [3]	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 μM), CGP (10 nM), or Norepinephrine (NE) and CGP[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Mol Immunol. 2023 Jan 5.
- ACS Nano. 2022 Aug 23;16(8):12553-12568.
- Nat Commun. 2022 Jul 25;13(1):4278.
- Cell Rep Med. 2023 May 24;101061.
- Theranostics. 2022 Jun 6;12(10):4718-4733.

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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.

[3]. Brian P Ramos, et al. Adrenergic pharmacology and cognition: focus on the prefrontal cortex. Pharmacol Ther. 2007 Mar;113(3):523-36.

[4]. Xinyu Xu, et al. Binding pathway determines norepinephrine selectivity for the human β 1 AR over β 2 AR. Cell Res. 2021 May;31(5):569-579.

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

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