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SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

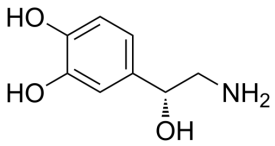
F. +43(0)1 489 3961-7

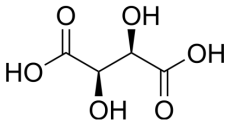
mail@szabo-scandic.com

www.szabo-scandic.com

linkedin.com/company/szaboscandic

Norepinephrine tartrate





Cat. No.:	HY-13715C
CAS No.:	51-40-1
Molecular Formula:	$C_{12}H_{17}NO_9$
Molecular Weight:	319.26
Target:	Endogenous Metabolite; Adrenergic Receptor; Autophagy
Pathway:	Metabolic Enzyme/Protease; GPCR/G Protein; Neuronal Signaling; Autophagy
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)

BIOLOGICAL ACTIVITY

Description

Norepinephrine (Levarterenol; L-Noradrenaline) tartrate is a potent adrenergic receptor (AR) agonist. Norepinephrine tartrate 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) tartrate is generally considered to be a β_1-subtype selective adrenergic agonist over β_2-adrenoceptor. Norepinephrine(NE) tartrate 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.

RT-PCR[2]

Cell Line:	Subcutaneous preadipocytes Adipocytes.
Concentration:	10 μM.
Incubation Time:	6 hours.
Result:	AT2 activation suppressed Norepinephrine induced UCP1 in white adipocytes (iWA)

In Vivo

Induction of cardiomyopathy[5][6]

Background

Norepinephrine is a potent growth factor for cardiomyocytes, and long-term infusion of sub-hypertensive doses of Norepinephrine in animals can cause an increase in myocardial mass and left ventricular wall thickness. Norepinephrine activates the raf-1 kinase/MAP kinase cascade through α1 and β-adrenergic stimulation, and the signaling pathways from these two receptors work synergistically to induce cardiomyocyte hypertrophy.

Specific Mmodeling Methods

Rat: Sprague-Dawley rats • adult (6 months old) • Male &bull.
Administration: Each rat was continuously injected with 100 μg/kg/h (Norepinephrine; HY-13715) or 200 μg/kg/h (Norepinephrine; HY-13715) through an osmotic minipump.

Note

Modeling Record

Molecular changes: Significantly increased Dnmt activity and the expressions of Dnmt1, 3a, and 3b in the left ventricle.
Significantly increased the mRNA expressions of fetal genes ANP, BNP, and βMHC in the left ventricle.
Significantly increased ROS production in the left ventricle and increased global genomic DNA methylation and gene-specific CpG methylation at the Egr-1 binding site of the left ventricular PKCε promoter region in a concentration-dependent manner.
Increased lactate dehydrogenase release in a concentration-dependent manner.
Significantly reduced the left ventricular developed pressure and dP/dtmax, inducing the upregulation of myotrophin and downregulation of four-and-a-half LIM domains protein 2 (FHL2).
Phenotype observations: Increased the myocardial infarction area and sustained the elevation of blood pressure.
Induced cardiac hypertrophy and reduced cardiac contractility.
Increased the left ventricular weight.

Correlated Product(s): 5-Aza-2'-deoxycytidine (HY-A0004), Prazosin (HY-B0193), Propranolol (HY-B0573B)

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. 2024 May 7;15(1):3834.
- Nat Commun. 2022 Jul 25;13(1):4278.
- Cell Rep Med. 2023 May 24;101061.

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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.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA