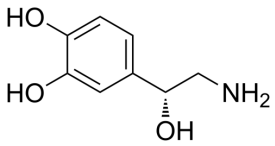
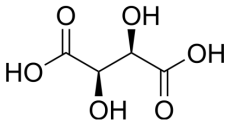
Norepinephrine tartrate





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:	Please store the product under the recommended conditions in the Certificate of Analysis.

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)

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.

See more customer validations on www.MedChemExpress.com

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