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Lieferung & Zahlungsart

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Zuschläge

- Mindermengenzuschlag
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Clonidine hydrochloride

Cat. No.: HY-B0409A

CAS No.: 4205-91-8

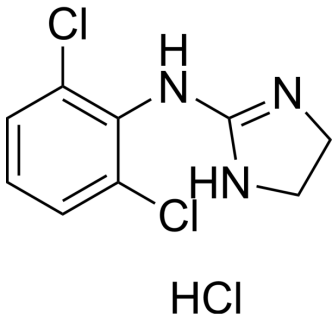
Molecular Formula: $C_9H_{10}Cl_3N_3$

Molecular Weight: 266.55

Target: Adrenergic Receptor

Pathway: GPCR/G Protein; Neuronal Signaling

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_2O : 33.33 mg/mL (125.04 mM; Need ultrasonic)
DMSO : 7.6 mg/mL (28.51 mM; Need ultrasonic and warming)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.7516 mL	18.7582 mL	37.5164 mL
5 mM	0.7503 mL	3.7516 mL	7.5033 mL
10 mM	0.3752 mL	1.8758 mL	3.7516 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (375.16 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Clonidine hydrochloride is an agonist of α2-adrenoceptor and potent antihypertensive agent.

IC_{50} & Target

α adrenergic receptor

In Vitro

Clonidine (0.01, 0.1 or 1 μM) significantly induces CGRP (α and β) mRNA expression in a dose-dependent manner in endothelial cells. Clonidine treatment (1 μM) for 24 h significantly increases the NO level in endothelial cells. NO pathway modulates CGRP production induced by clonidine[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Clonidine (50 μg/kg, i.p.) induces a significant decrease in body temperature of rat lasting 3 hr, with the maximum at 1 hr after administration. An intracerebroventricular pretreatment of rats with neutral doses of phentolamine 15 min before clonidine considerably antagonizes the clonidine-induced hypothermia[1]. Clonidine (0.003-0.05 mg/kg, i.p.) potently suppresses dopamine efflux in the prefrontal cortex induced by PCP. Pretreatment with the alpha-2A receptor antagonist

(BRL-44408) prevents clonidine from suppressing PCP-induced dopamine overflow in the prefrontal cortex[3]. In DMSO-pretreated SO rats, clonidine (0.6 μg i.c.) has no effect on blood pressure. However, after central adenosine A1R blockade (DPCPX) in SO rats, clonidine significantly ($P < 0.05$, one-way ANOVA) reduces blood pressure. In contrast, in DMSO-pretreated ABD rats, clonidine (0.6 μg i.c.) causes significant reduction in blood pressure; importantly, central A1R blockade (DPCPX pretreatment) does not influence ($P > 0.05$, one-way ANOVA) clonidine-evoked reduction in blood pressure in ABD rats. In DPCPX-pretreated SO rats and along with the appearance of the hypotensive response, clonidine causes a significant ($P < 0.05$) increase in the RVLM pERK1/2 level compared with basal or clonidine treatment in DMSO-pretreated SO rats. In vehicle (DMSO)-pretreated ABD rats, clonidine significantly ($P < 0.05$) enhances RVLM pERK1/2, and this response is not affected by DPCPX pretreatment[4].

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PROTOCOL

Animal Administration [3]

On the day of the experiment, the flow rate is increased to 2 μL/min approximately 2 h before beginning the collection of baseline samples. Dialysates are collected every 20 min; after 4 baseline samples are collected, animals are pretreated with an intra-peritoneal (i.p.) injection of either 0.9% saline (the vehicle), clonidine (0.0033, 0.01 or 0.05 mg/kg) or guanfacine (0.05 or 0.5 mg/kg), before receiving an injection of PCP (2.5 mg/kg, i.p.) 20 min later. In a separate study, BRL (1.0 mg/kg) is administered 20 min prior to clonidine. In addition, for some control experiments, the animals only receive one injection of saline, clonidine (0.01 or 0.05 mg/kg), guanfacine (0.5 mg/kg) or BRL (1.0 mg/kg).

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

CUSTOMER VALIDATION

- Cell Rep. 2019 Dec 3;29(10):2929-2935.e4
- Neurosci Bull. 2022 Apr;38(4):386-402.
- medRxiv. 2024 May 31.

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REFERENCES

[1]. Bugajski J, et al. The involvement of central alpha-adrenergic and histamine H2-receptors in the hypothermia induced by clonidine in the rat. Neuropharmacology. 1980 Jan;19(1):9-15.

[2]. Zhang YM, et al. Clonidine induces calcitonin gene-related peptide expression via nitric oxide pathway in endothelial cells. Peptides. 2009 Sep;30(9):1746-52.

[3]. Jentsch JD, et al. Clonidine and guanfacine attenuate phencyclidine-induced dopamine overflow in rat prefrontal cortex: mediating influence of the alpha-2A adrenoceptor subtype. Brain Res. 2008 Dec 30;1246:41-6.

[4]. Nassar N, et al. Brainstem adenosine A1 receptor signaling masks phosphorylated extracellular signal-regulated kinase 1/2-dependent hypotensive action of clonidine in conscious normotensive rats. J Pharmacol Exp Ther. 2009 Jan;328(1):83-9.

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