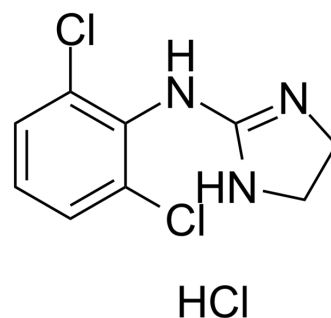


## Clonidine hydrochloride

Cat. No.:	HY-B0409A
CAS No.:	4205-91-8
Molecular Formula:	C <sub>9</sub> H <sub>10</sub> Cl <sub>3</sub> N <sub>3</sub>
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 <sub>2</sub> O : 33.33 mg/mL (125.04 mM; Need ultrasonic)																	
	DMSO : 7.6 mg/mL (28.51 mM; Need ultrasonic and warming)																	
Preparing Stock Solutions	<table border="1"> <thead> <tr> <th rowspan="2">Solvent Concentration</th> <th rowspan="2">Mass</th> <th>1 mg</th> <th>5 mg</th> <th>10 mg</th> </tr> </thead> <tbody> <tr> <td>1 mM</td> <td>3.7516 mL</td> <td>18.7582 mL</td> <td>37.5164 mL</td> </tr> <tr> <td>5 mM</td> <td>0.7503 mL</td> <td>3.7516 mL</td> <td>7.5033 mL</td> </tr> <tr> <td>10 mM</td> <td>0.3752 mL</td> <td>1.8758 mL</td> <td>3.7516 mL</td> </tr> </tbody> </table>	Solvent Concentration	Mass	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
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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 α <sub>2</sub> -adrenoceptor and potent antihypertensive agent.
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 <sup>[2]</sup> . 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 <sup>[1]</sup> . 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 <sup>[3]</sup> . 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  $\mu\text{g}$  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<sup>[4]</sup>.

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## PROTOCOL

### Animal Administration <sup>[3]</sup>

On the day of the experiment, the flow rate is increased to 2  $\mu\text{L}/\text{min}$  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.

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

Tel: 609-228-6898

Fax: 609-228-5909

E-mail: [tech@MedChemExpress.com](mailto:tech@MedChemExpress.com)

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