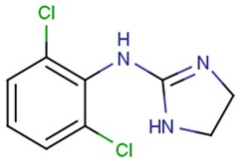
Data Sheet (Cat.No.T7045)

TargetMol
A DRUG SCREENING EXPERT

Clonidine

Chemical Properties



CAS No.:	4205-90-7
Formula:	C9H9Cl2N3
Molecular Weight:	230.09
Appearance:	N/A
Storage:	0-4℃ for short term (days to weeks), or -20℃ for long term (months).

Biological Description

Description	Clonidine is a centrally active alpha-adrenergic agonist used predominantly as an antihypertensive agent, usually in combination with other agents. Despite wide-scale use for many years, clonidine has not been linked definitively to either serum aminotransferase elevations or clinically apparent liver injury.
Targets(IC_{50})	α2-adrenergic receptor: None
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].
In vivo	Clonidine (3-50 μg/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]. 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]. 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 a 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 a vehicle (DMSO)-pretreated ABD rats, clonidine significantly ($P < 0.05$) enhances RVLM pERK1/2, and this response is not affected by DPCPX pretreatment [4].

Solubility Information

Solubility	DMSO: 15 mg/mL Water: 25 mg/mL (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	4.346 mL	21.731 mL	43.461 mL
5 mM	0.869 mL	4.346 mL	8.692 mL
10 mM	0.435 mL	2.173 mL	4.346 mL
50 mM	0.087 mL	0.435 mL	0.869 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. The storage conditions and period of the stock solution: - 80 ℃ for 6 months; - 20 ℃ for 1 month. Please use it as soon as possible.

Reference

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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