



## **Clonidine (hydrochloride)**

Catalog No: tcsc2509

## **Available Sizes**

Size: 100mg

Size: 500mg



## **Specifications**

CAS No:

4205-91-8

Formula:

 $C_9H_{10}CI_3N_3$ 

**Pathway:** 

GPCR/G Protein

**Target:** 

Adrenergic Receptor

**Purity / Grade:** 

>98%

**Solubility:** 

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

**Observed Molecular Weight:** 

266.55

## **Product Description**

Clonidine hydrochloride is an agonist of  $\alpha 2$ -adrenoceptor and potent antihypertensive agent.

In Vitro: Clonidine (0.01, 0.1 or 1  $\mu$ M) significantly induces CGRP ( $\alpha$  and  $\beta$ ) mRNA expression in a dose-dependent manner in endothelial cells. Clonidine treatment (1  $\mu$ M) for 24 h significantly increases the NO level in endothelial cells. NO pathway modulates CGRP production induced by clonidine<sup>[2]</sup>.

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) 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 [4].

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