Piperoxan hydrochloride

Cat. No.: HY-100850

CAS No.: 135-87-5

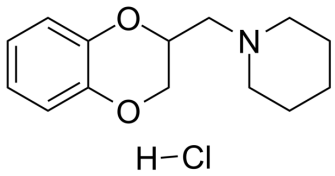
Molecular Formula: $C_{14}H_{20}ClNO_2$

Molecular Weight: 269.77

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 : 50 mg/mL (185.34 mM; Need ultrasonic)
DMSO : ≥ 31 mg/mL (114.91 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.7069 mL	18.5343 mL	37.0686 mL
5 mM	0.7414 mL	3.7069 mL	7.4137 mL
10 mM	0.3707 mL	1.8534 mL	3.7069 mL

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

In Vivo

1. Add each solvent one by one: PBS
Solubility: 25 mg/mL (92.67 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Piperoxan (Benodaine) hydrochloride is an α_2 adrenoceptor antagonist. Piperoxan hydrochloride is the first-generation antihistamine.

IC_{50} & Target

adrenoceptor[1]

In Vitro

When the medulla is superfused with α_2 adrenoceptor antagonist Piperoxane (50 μM; 5 min) while the pons is with artificial cerebrospinal fluid (ACSF), the three inactive preparations display rhythmic phrenic bursts at a low frequency (2-4 c/min), and the phrenic burst frequency of the 12 active ones significantly increases during the last 3 min of Piperoxane applications (163±12% of the previous mean frequency). In active medullary preparations, the effects of NA applications (25 μM; 5 min) are compared when the preparations sre superfused either by ACSF (n=8) or by the α2 adrenoceptor antagonist Piperoxane (50 μM; PIP-ACSF; n=5). NA applications either alone (NA-ACSF) or with Piperoxane (PIP-ACSF+NA) significantly increases the phrenic burst frequency. However, the blockage of the medullary α2 adrenoceptors by Piperoxane potentiates a phrenic

burst frequency increase: during the fifth minute of NA applications, the phrenic burst frequency reached 171±11% of the mean control value when ACSF is applied alone and 234±21% of the mean control value when PIP-ACSF is applied in control condition[1].

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

PROTOCOL

Kinase Assay [1]

The mouse neonates (P0-P3) are ether-anesthetized and decerebrated; the brain stems and the cervical spinal cords are dissected out and placed ventral sides up in a 2 mL chamber superfused with artificial cerebrospinal fluid (ACSF) at 27±0.25°C (mean±SD), renewed at a rate of 2 mL/min. The ACSF [containing (in mM) 129 NaCl, 3.35 KCl, 1.26 $CaCl_2$, 1.15 $MgCl_2$, 21 $NaHCO_3$, 0.58 NaH_2PO_4, and 30 glucose] is oxygenated and equilibrated (pH 7.4 at 27°C) by bubbling carbogene (95% O_2-5% CO_2). In the pharmacological experiments, this is replaced by another ACSF in which bioreactive substances are dissolved: noradrenaline at 25 µM (NA-ACSF) or α2 adrenoceptor antagonists, either Piperoxane at 50 µM (PIP-ACSF) or yohimbine at 50 µM (YO-ACSF). In some of the experiments, a patch-clamp microelectrode (1 µm diameter tip) is lowered within the ventral pons into the A5 nucleus where a solution of either ACSF or NA (1 mM) is pressure-ejected. The ejected volume is estimated 20 nL for a pressure pulse lasting 2 s[1].

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Animal Administration [2]

Mice[2]

Male Balb-C mice are used, weighing between 20 and 25 g. In mice pretreated with the α-adrenoceptor antagonist Piperoxan, or with naloxone, both at a dose of 3×10^{-5} mol /kg s.c. given 15 min before the acetic acid, the antinociceptive action of (-)-isoprenaline is only slightly antagonized. Dose-ratios of 1.45 and 1.7, are produced by these two antagonists.

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

REFERENCES

[1]. Viemari JC, et al. Nasal trigeminal inputs release the A5 inhibition received by the respiratory rhythm generator of the mouse neonate. J Neurophysiol. 2004 Feb;91(2):746-58.

[2]. Bentley GA, et al. The antinociceptive action of some beta-adrenoceptor agonists in mice. Br J Pharmacol. 1986 Jul;88(3):515-21.

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