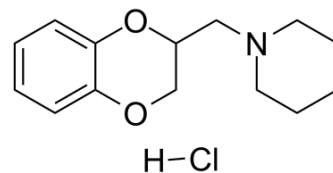


Piperoxan hydrochloride

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:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

H₂O : 50 mg/mL (185.34 mM; Need ultrasonic)

DMSO : ≥ 31 mg/mL (114.91 mM)

* "≥" means soluble, but saturation unknown.

	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
Preparing Stock Solutions	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.

BIOLOGICAL ACTIVITY

Description

Piperoxan (Benodaine) hydrochloride is an α₂ adrenoceptor antagonist. Piperoxan hydrochloride is the first-generation antihistamine.

IC₅₀ & Target

adrenoceptor^[1]

In Vitro

When the medulla is superfused with α₂ 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 are 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 CaCl₂, 1.15 MgCl₂, 21 NaHCO₃, 0.58 NaH₂PO₄, and 30 g/lucose] is oxygenated and equilibrated (pH 7.4 at 27°C) by bubbling carbogene (95% O₂-5% CO₂). In the pharmacological experiments, this is replaced by another ACSF in which bioactive 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].

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

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.

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

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