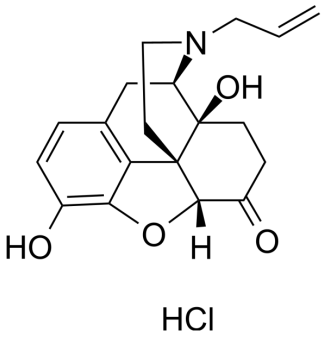
Naloxone hydrochloride

Cat. No.:	HY-17417
CAS No.:	357-08-4
Molecular Formula:	$C_{19}H_{22}ClNO_4$
Molecular Weight:	363.84
Target:	Opioid 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 : 62.5 mg/mL (171.78 mM; Need ultrasonic)
DMSO : ≥ 30 mg/mL (82.45 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.7485 mL	13.7423 mL	27.4846 mL
5 mM	0.5497 mL	2.7485 mL	5.4969 mL
10 mM	0.2748 mL	1.3742 mL	2.7485 mL

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

In Vivo

1. Naloxone is made up in a vehicle of 2% dimethylformamide (aqueous)[4].

BIOLOGICAL ACTIVITY

Description

Naloxone hydrochloride is an antagonist of Opioid receptor. Naloxone hydrochloride alleviates opioid-overdose-induced respiratory depression. Naloxone hydrochloride may cause pulmonary edema and cardiac arrhythmias[1].

In Vivo

Naloxone (2.0 mg/kg with constant infusion of 1.7 mg/kg/h) causes a significant improvement in neurobehavioral outcome which persists up to 4 weeks postinjury in rat. Naloxone treatment causes a modest and nonsignificant increase in mean arterial blood pressure (MAP)[1]. Naloxone (0.4 mg/kg) causes memory facilitation and antagonizes the amnestic effect of ACTH and epinephrine in rat[2]. Naloxone treatment diminishes the strength of the initial tetanus in a dose-related manner in cats. Naloxone (5 or 10 mg/kg, i.v.) causes subsequent doses of morphine to produce less PTP depression but has no effect on maximal twitch depression[3].

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

PROTOCOL

Animal Administration [2]

Young adult Wistar rats are used in the assay: females for experiments l and 3 (age 50-70 days, weight 100-185 g) and males for experiments 2 and 4 (age 51-69 days, weight 140-200 g). The same apparatus is used for both: a 50×25×25 cm box made of plywood with a frontal glass wall, whose floor consists of 1 mm-caliber bronze bars spaced l0 mm apart. In task l, a 5 cm high, 25×25 cm wooden platform is introduced into the box so as to cover the left half of the grid. Rats are held gently by their bodies and loared onto the platform facing the rear left corner, at which time a quartz chronometer is activated. Time is counted until the animals stepped down from the platform and placed their four paws on the grid, upon which a 0.5 mA, 60 Hz footshock is continuously delivered until the animals return to the platform. In task 2 the platform is only 7 cm wide and covered only the leftmost seven bars of the grid; the footshock is of only 0.3 mA and is delivered in 0.4 msec pulses once every 2 sec until the animals climb back onto the platform. Immediately after training in each task the animals are withdrawn from the box injected i.p. with one of the following: saline (1.0 mL/kg), $ACTH_{1-24}$ (0.2 or 2.0 μg/kg), epinephrine HCl (5.0 or 50.0 μg/kg), human β-endorphin (0.1 or 1.0 μg/kg), naloxone HCl (0.4 mg/kg), or ACTH or epinephrine given together with either β-endorphin or naloxone. All drugs are dissolved in saline to an injection volume of 1.0 mL/kg. Twenty-four hr after training the animals again are placed on the platform (the wide one for task 1, experiments 1 and 2, and the narrow one for task 2, experiments 3 and 4), and their latency to stepdown onto the grid again is measured as in the training session. A ceiling of 180 sec is imposed on this measure.

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CUSTOMER VALIDATION

- Cell Rep. 2020 Mar 17;30(11):3625-3631.e6.
- Eur J Pain. 2017 May;21(5):804-814.

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REFERENCES

[1]. McIntosh TK, et al. Beneficial effect of the nonselective opiate antagonist naloxone hydrochloride and the thyrotropin-releasing hormone (TRH) analog YM-14673 on long-term neurobehavioral outcome following experimental brain injury in the rat. J Neurotrau

[2]. Izquierdo I, et al. Effect of ACTH, epinephrine, beta-endorphin, naloxone, and of the combination of naloxone or beta-endorphinwith ACTH or epinephrine on memory consolidation. Psychoneuroendocrinology. 1983;8(1):81-7.

[3]. Soteropoulos GC, et al. Neuromuscular effects of morphine and naloxone. J Pharmacol Exp Ther. 1973 Jan;184(1):136-42.

[4]. Sun L, et al. Endocannabinoid activation of CB1 receptors contributes to long-lasting reversal of neuropathic pain by repetitive spinal cord stimulation. Eur J Pain. 2017 May;21(5):804-814.

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