# Product Data Sheet

Product Name: (±)-Epibatidine
Cat. No.: GC14410

## Chemical Properties

| | | | |
|---|---|---|---|
| Cas. No. | 148152-66-3 | | |
| Chemical Name | (1S,2S,4R)-2-(6-chloropyridin-3-yl)-7-azabicyclo[2.2.1]heptane | | |
| SMILES | ClC(N=C1)=CC=C1[C@H]2[C@H](CC3)N[C@H]3C2 | | |
| Formula | $C_{11}H_{13}ClN_2$ | M.Wt | 208.69 |
| Solubility | <24.55mg/ml in ethanol | Storage | Store at -20°C |
| General tips | For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months. | | |
| Shipping Condition | Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request. | | |
| Structure | (±)-Epibatidine | | |

## Protocol


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**Animal experiment:**

Rats[1]Voiding is studied in urethane-anesthetized (1.2 g/kg sc) or awake female Sprague-Dawley rats (250-300 g). In all experiments, control cystometrograms (CMGs) are recorded for ~2 h before intracerebroventricular and intravenous injection of vehicle or (±)-Epibatidine solutions. Dose-response curves are constructed by administering increasing doses of (±)-Epibatidine [0.001-1 μg in 1 μL intracerebroventricularly (icv); 0.001-1 μg in 200 μL iv] at 30-min to 2-h intervals. (±)-Epibatidine is administered ~30 min after vehicle (aCSF, 1 μL icv; or saline solution, 200 μL iv). Chlorisondamine (10 μg, 1 μL icv) is injected 10-30 min before (±)-Epibatidine via the intracerebroventricular route in some experiments to block the effect of the agonist. The intravesical pressure to induce micturition (PT), MVP, and intercontraction interval (ICI; the interval between voids or reflex bladder contractions) are measured and converted into percent change from control values[1].


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References:

[1]. Lee SJ, et al. Effect of (+/-)-epibatidine, a nicotinic agonist, on the central pathways controlling voidingfunction in the rat. Am J Physiol Regul Integr Comp Physiol. 2003 Jul;285(1):R84-90.

## Background

(+)-AJ 76 hydrochloride is an antagonist of dopamine autoreceptor with pKi values of 6.95, 6.67, 6.37, 6.21 and 6.07 for hD3, hD4, hD2S, hD2L and rD2 receptors, respectively.

Dopamine receptor is a G protein-coupled receptor and mainly exists in the vertebrate central nervous system (CNS). Dopamine receptor is a receptor for dopamine and plays a critical role in memory, learning, pleasure, cognition, motivation and fine motor control.

(+)-AJ 76 hydrochloride is a dopamine receptor antagonist. In rats, AJ76 stimulated locomotor activity and increased the levels of 3,4-dihydroxyphenylacetic acid (DOPAC) and HVA in brain, which were dopamine metabolites [1]. In rats injected with cocaine, (+)-A J76 increased the locomotor stimulation during the first 30 min. However, (+)-AJ76 inhibited the later more intense locomotor stimulation and cocaine-induced stereotypies [2]. In vivo, (+)-AJ76 induced dopamine release mainly through interaction with

dopamine receptors in the terminal regions of the A9 and A10 dopaminergic fibers. However, (+)-AJ76 increased the level of DOPAC via the somatodendritic autoreceptors [3].

References:

[1]. Kullingsjö H, Carlsson A, Svensson K. Effects of repeated administration of the preferential dopamine autoreceptor antagonist, (+)-AJ76, on locomotor activity and brain DA metabolism in the rat. Eur J Pharmacol, 1991, 205(3): 241-246.
[2]. Piercey MF, Lum JT, Hoffmann WE, et al. Antagonism of cocaine's pharmacological effects by the stimulant dopaminergic antagonists, (+)-AJ76 and (+)-UH232. Brain Res, 1992; 588(2): 217-222.
[3]. Waters N, Hansson L, Löfberg L, et al. Intracerebral infusion of (+)-AJ76 and (+)-UH232: effects on dopamine release and metabolism in vivo. Eur J Pharmacol, 1994, 251(2-3): 181-190.


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