Pargyline hydrochloride

Cat. No.: HY-A0091

CAS No.: 306-07-0

Molecular Formula: $C_{11}H_{14}ClN$

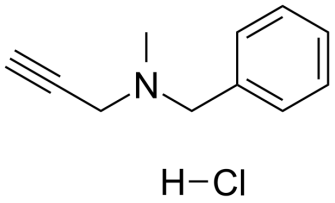
Molecular Weight: 195.69

Target: Monoamine Oxidase

Pathway: 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

H_2O : 100 mg/mL (511.01 mM; Need ultrasonic)

DMSO : ≥ 32 mg/mL (163.52 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	5.1101 mL	25.5506 mL	51.1012 mL
5 mM	1.0220 mL	5.1101 mL	10.2202 mL
10 mM	0.5110 mL	2.5551 mL	5.1101 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (12.78 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (12.78 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (12.78 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Pargyline hydrochloride is an irreversible monoamine oxidase (MAO) inhibitor with K_is of 13 μM and 0.5 μM for MAO-A and MAO-B, respectively. Pargyline hydrochloride has antihypertensive and anticancer activities[1][2][3].

IC_{50} & Target

MAO-B	MAO-A
0.5 μM (Ki)	13 μM (Ki)

In Vitro

Pargyline (0.5-2 mM; 24-120 hours; LNCaP-LN3 cells) treatment inhibits the proliferation of prostate cancer cells in a time- and dose-dependent manner[2].
Pargyline (0.5-2 mM; 24-48 hours; LNCaP-LN3 cells) treatment decreases S phase and increases the G1 phase in the cells in a dose-dependent manner[2].
Pargyline (0.5 mM; 24 hours; LNCaP-LN3 cells) treatment increases the apoptotic cells[2].
Pargyline (2 mM; 48 hours; LNCaP-LN3 cells) treatment induces an increase of cytochrome c and a decrease of caspase-3 in the cells, but does not lead to a change of BCL-2 expression[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[2]

Cell Line:	LNCaP-LN3 cells
Concentration:	0.5 mM, 1 mM, 1.5 mM or 2 mM
Incubation Time:	24 hours, 48 hours, 72 hours, 96 hours or 120 hours
Result:	Inhibited the proliferation of prostate cancer cells in a time- and dose-dependent manner.

Cell Cycle Analysis[2]

Cell Line:	LNCaP-LN3 cells
Concentration:	0.5 mM, 2 mM
Incubation Time:	24 hours, 48 hours
Result:	The S phase ratio of the cells was decreased, while their G1 phase ratio was increased.

Apoptosis Analysis[2]

Cell Line:	LNCaP-LN3 cells
Concentration:	0.5 mM
Incubation Time:	24 hours
Result:	Increased the apoptotic cells.

Western Blot Analysis[2]

Cell Line:	LNCaP-LN3 cells
Concentration:	2 mM
Incubation Time:	48 hours
Result:	Induced an increase of cytochrome c and a decrease of caspase-3.

In Vivo

Pargyline (10 mg/kg; iv) treatment induces a moderate (about 20 mm Hg) but persistent (48 h) decrease of systolic blood pressure in unanesthetized adult spontaneously hypertensive rats (SHR) but not in normotensive rats[3].
A low dose of Pargyline (200 μg; icv) injected directly into the brain lowered arterial pressure. The hypotensive action of Pargylline in SHR appears to be the consequence of Norepinephrine accumulating at an inhibitory α-adrenoceptor in brain [3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Parkinson Dis. 2020;10(2):523-542.
- Neural Regen Res. 2021;16:1660-70.

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REFERENCES

[1]. C J Fowler, et al. The nature of the inhibition of rat liver monoamine oxidase types A and B by the acetylenic inhibitors clorgyline, l-deprenyl and pargyline. Biochem Pharmacol. 1982 Nov 15;31(22):3555-61.

[2]. Fuentes JA, et al. Central mediation of the antihypertensive effect of pargyline in spontaneously hypertensive rats. Eur J Pharmacol. 1979 Jul 15;57(1):21-7.

[3]. Hyung Tae Lee, et al. Effects of the monoamine oxidase inhibitors pargyline and tranylcypromine on cellular proliferation in human prostate cancer cells. Oncol Rep. 2013 Oct;30(4):1587-92.

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