Rasagiline mesylate

Cat. No.: HY-14605

CAS No.: 161735-79-1

Molecular Formula: $C_{13}H_{17}NO_3S$

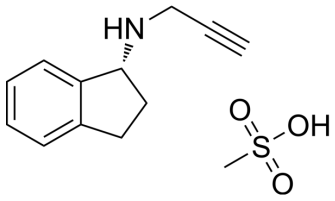
Molecular Weight: 267.34

Target: Monoamine Oxidase; Autophagy; Apoptosis

Pathway: Neuronal Signaling; Autophagy; Apoptosis

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 : 25 mg/mL (93.51 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.7406 mL	18.7028 mL	37.4056 mL
	5 mM	0.7481 mL	3.7406 mL	7.4811 mL
	10 mM	0.3741 mL	1.8703 mL	3.7406 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (374.06 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Rasagiline (R-AGN1135) mesylate is a highly potent selective irreversible mitochondrial monoamine oxidase (MAO) inhibitor with IC_{50}s of 4.43?nM and 412?nM for rat brain MAO B and A activity, respectively[1]. Rasagiline (mesylate) is a click chemistry reagent, itcontains an Alkyne group and can undergo copper-catalyzed azide-alkyne cycloaddition (CuAAc) with molecules containing Azide groups.

IC_{50} & Target

rMAO-B	rMAO-A
4.43 nM (IC_{50})	412 nM (IC_{50})

In Vitro

Rasagiline (0.25 nM; 96 hours) significantly increases the proliferation rates of SH-SY5Y and 1242-MG upon Dexamethasone (10 μM) treatment[2].

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

Cell Viability Assay[2]

Cell Line:	Neuroblastoma SH-SY5Y, and glioblastoma 1242-MG
Concentration:	0.25 nM
Incubation Time:	96 hours
Result:	Caused ~60% increase in the cell proliferation rate for SH-SY5Y cells treated with Dexamethasone. Caused ~35% increase in cell proliferation rate for 1242-MG cells treated with Dexamethasone.

In Vivo

Rasagiline is neuroprotective in a transgenic model of multiple system atrophy. Motor behavioural tests show improvements in motor deficits associated with 2.5 mg/kg Rasagiline therapy[3].

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

Animal Model:	(PLP)-α-synuclein transgenic mice over 6 months of age[3]
Dosage:	Low-(0.8 mg/kg b.w.) and high dose (2.5 mg/kg b. w.)
Administration:	Administered subcutaneously every 24 h for a total period of 4 weeks (from day 1 till day 28 of the experiment).
Result:	Low dose treatment did not show protective efficacy in striatum with number of neurons similar to placebo treated MSA mice. High dose was associated with about 15% rescue of DARPP-32 immunoreactive striatal neurons. Low dose treatment had no effect on nigral neuronal loss, but high dose completely protected nigral neurons with numbers comparable to healthy controls.

CUSTOMER VALIDATION

- Eur J Med Chem. 2023 Apr 28;255:115417.
- Front Cell Neurosci. 2018 Sep 11;12:309.
- Bioorg Chem. 2023 Jun 3, 106654.
- Oncotarget. 2018 Jan 30;9(15):12137-12153.

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REFERENCES

[1]. M B Youdim, et al. Rasagiline [N-propargyl-1R(+)-aminoindan], a selective and potent inhibitor of mitochondrial monoamine oxidase B. Br J Pharmacol. 2001 Jan;132(2):500-6.

[2]. Nadia Stefanova, et al. Rasagiline is neuroprotective in a transgenic model of multiple system atrophy. Exp Neurol. 2008 Apr;210(2):421-7.

[3]. Shawna Tazik, et al. Comparative neuroprotective effects of Rasagiline and aminoindan with selegiline on dexamethasone-induced brain cell apoptosis. Neurotox Res. 2009 Apr;15(3):284-90.

Caution: Product has not been fully validated for medical applications. For research use only.

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