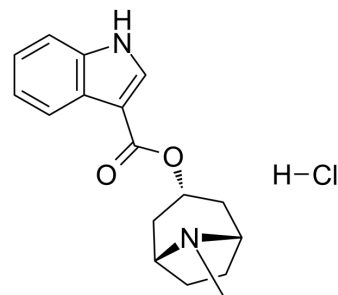


## Tropisetron Hydrochloride

Cat. No.:	HY-B0020
CAS No.:	105826-92-4
Molecular Formula:	C <sub>17</sub> H <sub>21</sub> ClN <sub>2</sub> O <sub>2</sub>
Molecular Weight:	320.81
Target:	5-HT 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

DMSO : 70 mg/mL (218.20 mM; Need ultrasonic)  
 H<sub>2</sub>O : ≥ 50 mg/mL (155.86 mM)  
 \* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	3.1171 mL	15.5855 mL	31.1711 mL
	5 mM	0.6234 mL	3.1171 mL	6.2342 mL
	10 mM	0.3117 mL	1.5586 mL	3.1171 mL

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

#### In Vivo

1. Add each solvent one by one: PBS  
 Solubility: 120 mg/mL (374.05 mM); Clear solution; Need ultrasonic

### BIOLOGICAL ACTIVITY

#### Description

Tropisetron Hydrochloride (SDZ-ICS-930) is a selective 5-HT<sub>3</sub> receptor antagonist and α<sub>7</sub>-nicotinic receptor agonist with an IC<sub>50</sub> of 70.1 ± 0.9 nM for 5-HT<sub>3</sub> receptor. IC<sub>50</sub> value: 70.1 ± 0.9 nM Target: 5-HT<sub>3</sub> receptor; α<sub>7</sub>-nicotinic receptor in vitro: Retinal ganglion cells (RGCs) pretreated with 100 nM tropisetron before glutamate increased cell survival to an average of 105% compared to controls. Inhibition studies using the alpha<sub>7</sub> nAChR antagonist, MLA (10 nM), support the hypothesis that tropisetron is an effective neuroprotective agent against glutamate-induced excitotoxicity; mediated by α<sub>7</sub> nAChR activation. Tropisetron had no discernible effects on pAkt levels but significantly decreased p38 MAPK levels associated with excitotoxicity from an average of 15 ng/ml to 6 ng/ml [2]. Tropisetron, but not granisetron, significantly inhibits the phosphatase activity of calcineurin, over-expresses the CB(1) receptors at both transcriptional and protein levels, and reduces cAMP content in cerebellar granule neurons (CGNs) [4]. in vivo: Animals were treated intracerebroventricularly with tropisetron, mCPBG (selective 5-HT<sub>3</sub> receptor agonist) or mCPBG plus tropisetron on days 1, 3, 5 and 7. Tropisetron significantly diminished the elevated levels of these markers and reversed the cognitive deficit. Interestingly, tropisetron was also found to be a potent inhibitor of calcineurin phosphatase activity [1]. tropisetron (5mg/kg/day) plus mCPBG

(10mg/kg/day), and granisetron (5mg/kg/day) intraperitoneally on days 3-35 post-immunization. Treatment with tropisetron and granisetron markedly suppressed the clinical symptoms of EAE ( $p < 0.001$ ) and reduced leukocyte infiltration as well as demyelination in the spinal cord ( $p < 0.05$ ) [3].

**IC<sub>50</sub> & Target**

5-HT<sub>3</sub> Receptor  
70.1 nM (IC<sub>50</sub>)

**CUSTOMER VALIDATION**

- Acta Pharmacol Sin. 2024 May 3.
- Int J Neuropsychopharmacol. 2019 Sep 1;22(9):574-584.

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**REFERENCES**

- [1]. Rahimian R, et al. Tropisetron attenuates amyloid-beta-induced inflammatory and apoptotic responses in rats. Eur J Clin Invest. 2013 Oct;43(10):1039-51.
- [2]. Swartz MM, et al. Tropisetron as a neuroprotective agent against glutamate-induced excitotoxicity and mechanisms of action. Neuropharmacology. 2013 Oct;73:111-21.
- [3]. Aminian A, et al. Tropisetron diminishes demyelination and disease severity in an animal model of multiple sclerosis. Neuroscience. 2013 Jun 15;248C:299-306.
- [4]. Rahimian R, et al. Tropisetron upregulates cannabinoid CB1 receptors in cerebellar granule cells: possible involvement of calcineurin. Brain Res. 2011 Oct 12;1417:1-8.

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

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