Granisetron

Cat. No.: HY-B0071

CAS No.: 109889-09-0

Molecular Formula: $C_{18}H_{24}N_4O$

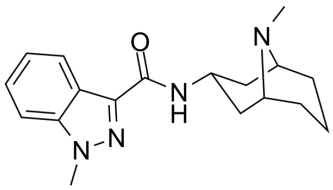
Molecular Weight: 312.41

Target: 5-HT Receptor

Pathway: GPCR/G Protein; 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

DMSO : 25 mg/mL (80.02 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.2009 mL	16.0046 mL	32.0092 mL
	5 mM	0.6402 mL	3.2009 mL	6.4018 mL
	10 mM	0.3201 mL	1.6005 mL	3.2009 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 (8.00 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.00 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.00 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Granisetron (BRL 43694) is a serotonin 5-HT3 receptor antagonist used as an antiemetic to treat nausea and vomiting following chemotherapy.

IC_{50} & Target

$5\text{-}HT_3$ Receptor
17 μM (IC_{50})

In Vitro

In rat forestomach GR reduced 5-HT-evoked contractions at IC50 17 /- 6 uM. In isolated rabbit heart, GR 0.003-0.03 nM dose-

	dependently reduced s-HT tachycardia; at high levels GR reduced submaximal and maximal responses to 5-HT[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Leukocyte accumulation was dose-dependently inhibited by granisetron both at 6 and 72 h after induction of inflammation. Granisetron increased PGE(2) level at a lower dose (50 microg/pouch) but higher doses (100 and 200 microg/pouch) inhibited the release. At the same time, TNFalpha production was decreased by the lower dose and increased by higher doses of granisetron in a reciprocal fashion[2]. The GTDS displayed non-inferiority to oral granisetron: complete control was achieved by 60% of patients in the GTDS group, and 65% in the oral granisetron group (treatment difference, -5%; 95% confidence interval, -13-3). Both treatments were well tolerated, the most common adverse event being constipation[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Sanger GJ, Nelson DR. Selective and functional 5-hydroxytryptamine3 receptor antagonism by BRL 43694 (granisetron). Eur J Pharmacol. 1989 Jan 10;159(2):113-24.

[2]. Maleki-Dizaji N, Eteraf-Oskouei T, Fakhrjou A, The effects of 5HT3 receptor antagonist granisetron on inflammatory parameters and angiogenesis in the air-pouch model of inflammation. Int Immunopharmacol. 2010 Sep;10(9):1010-6.

[3]. Boccia RV, Gordan LN, Clark G, Efficacy and tolerability of transdermal granisetron for the control of chemotherapy-induced nausea and vomiting associated with moderately and highly emetogenic multi-day chemotherapy: a randomized, double-blind, phase III

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