Amisulpride

Cat. No.: HY-14545

CAS No.: 71675-85-9

Molecular Formula: $C_{17}H_{27}N_3O_4S$

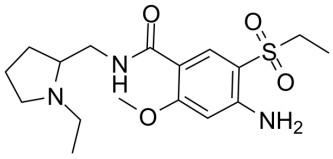
Molecular Weight: 369.48

Target: Dopamine Receptor

Pathway: GPCR/G Protein; Neuronal Signaling

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (135.33 mM; Need ultrasonic)

H_2O : 0.2 mg/mL (0.54 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.7065 mL	13.5325 mL	27.0651 mL
5 mM	0.5413 mL	2.7065 mL	5.4130 mL
10 mM	0.2707 mL	1.3533 mL	2.7065 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 (6.77 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.77 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: 2.5 mg/mL (6.77 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Amisulpride is a dopamine D_2/D_3 receptor antagonist with K_is of 2.8 and 3.2 nM for human dopamine D_2 and D_3, respectively.

IC_{50} & Target

Ki: 2.8 nM (D_2 receptor), 3.2 nM (D_3 receptor)[1]

In Vitro

Amisulpride is an atypical dopamine D_2/D_3 receptor antagonist with K_is of 2.8 and 3.2 nM for human dopamine D_2 and D_3,

	respectively. Amisulpride (100 nM) inhibits quinpirole-elicited [^{3}H]thymidine incorporation with an IC_{50} value of 22±3 nM (n=3). Amisulpride slightly but significantly increases [^{3}H]dopamine release from slices of the rat striatum (S_2/S_1=0.88±0.04 under control conditions, n=6; 1.04±0.08 in the presence of 100 nM Amisulpride,n=4; P<0.05) and opposes the inhibitory effects of 7-OH-DPAT in both brain areas[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Only the highest dose of Amisulpride (100 mg/kg) significantly reduces dopamine levels in the striatum or limbic system. Amisulpride significantly increases the synthesis of dopamine in the rat striatum and limbic system at doses of 20 and 100 mg/kg. Amisulpride (0.5 to 75 mg/kg) fails to provoke an additional increase in dopa accumulation in the striatum but slightly accelerates, at 75 mg/kg, dopamine synthesis in the limbic system. In comparison with vehicle-treated controls, Amisulpride (10 mg/kg) increases extracellular dopamine levels. The administration of Amisulpride (0.5 to 15 mg/kg s.c.) provokes a time- and dose-dependent increase in the stimulation-evoked dopamine release. Amisulpride decreases striatal ACh levels significantly at 30 and 100 mg/kg (87.5% and 56.3% of control levels, respectively)[1]. In both acute study, Amisulpride (70 mg/kg, p.o.) significantly increases the duration of swimming behavior [F(3,28)=45.90, p<0.01][2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	The functional effects of Amisulpride at the dopamine D_3 receptor subtype are assessed. Briefly, the mitogenic response elicited in NG108-15 neuroblastoma-glioma cells stably transfected with human dopamine D_3 receptor cDNA by the addition of 10 nM quinpirole in the presence of 1 μM forskolin is quantified by the incorporation of [^{3}H]thymidine. Antagonism of quinpirole-induced mitogenesis is measured in the presence of increasing (0.1 to 100 nM) concentrations of Amisulpride[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	A total of 64 male Swiss albino mice weighing between 20 to 30 g are used. The animals are fed with standard pellet diet and water ad libitum. The mice are divided in different groups (n=8 in each group) and drug administration is done as follows: Group 1 (control): distilled water (1 mL/kg) 23.5, 5 and 1 h before the test. Group 3 (Amisulpride): Amisulpride (70 mg/kg) 23.5, 5 and 1 h before the test[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Schoemaker H, et al. Neurochemical characteristics of amisulpride, an atypical dopamine D2/D3 receptor antagonist with both presynaptic and limbic selectivity. J Pharmacol Exp Ther. 1997 Jan;280(1):83-97.

[2]. Pawar GR, et al. Evaluation of antidepressant like property of amisulpride per se and its comparison with fluoxetine and olanzapine using forced swimming test in albino mice. Acta Pol Pharm. 2009 May-Jun;66(3):327-31.

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