Spiramide

Cat. No.: HY-100971

CAS No.: 510-74-7

Molecular Formula: $C_{22}H_{26}FN_3O_2$

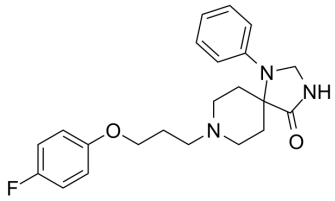
Molecular Weight: 383.46

Target: 5-HT Receptor; Dopamine Receptor

Pathway: GPCR/G Protein; Neuronal Signaling

Storage: -20°C, stored under nitrogen

* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 20 mg/mL (52.16 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6078 mL	13.0392 mL	26.0783 mL
	5 mM	0.5216 mL	2.6078 mL	5.2157 mL
	10 mM	0.2608 mL	1.3039 mL	2.6078 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 mg/mL (5.22 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2 mg/mL (5.22 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Spiramide (AMI-193) is a potent and selective antagonist of 5-HT_2 and dopamine D2 receptor, with K_is of 2 nM and 3 nM, respectively. Spiramide has >2000-fold selectivity for 5-HT_2 versus 5-HT_{1C} (K_i=4300 nM) receptors. Spiramide exhibits antipsychotic activity[1][2][3].

IC_{50} & Target

5-HT_2 Receptor 2 nM (Ki)	D_2 Receptor 3 nM (Ki)	5-HT_{1A} Receptor 50 nM (Ki)	D_1 Receptor 2530 nM (Ki)
5-HT_{1C} Receptor 4300 nM (Ki)			

In Vitro

Spiramide retains affinity for 5-HT_{1A} sites (K_i=50 nM) and also binds at dopamine D2 sites (K_i=3 nM), but possesses low

affinity for dopamine D1 sites (K_i=2530 nM)[1].

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

In Vivo

AMI-193 (0.003-0.01 mg/kg; i.m.) dose-dependently decreases response rate in monkeys under a fixed-interval (FI) schedule of stimulus termination[2].

AMI-193 (0.003-0.01 mg/kg; i.m.) attenuates the discriminative-stimulus effects of cocaine in drug-discrimination experiments[2].

AMI-193 (0.003-0.01 mg/kg; i.m.) reduces response rate under a second-order schedule of i.v. self-administration of cocaine (0.1 mg/infusion)[2].

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

Animal Model:	Adult male squirrel monkeys (850-1300 g)[2]
Dosage:	0.003, 0.01 mg/kg
Administration:	I.m. on Tuesday, Wednesday, and Thursday the following week
Result:	Decreased the response rate. The rate-decreasing effects were reversed by cocaine.

REFERENCES

[1]. Ismaiel AM, et, al. Antagonism of 1-(2,5-dimethoxy-4-methylphenyl)-2-aminopropane stimulus with a newly identified 5-HT2- versus 5-HT1C-selective antagonist. J Med Chem. 1993 Aug 20;36(17):2519-25.

[2]. Czoty PW, et, al. Behavioral effects of AMI-193, a 5-HT(2A)- and dopamine D(2)-receptor antagonist, in the squirrel monkey. Pharmacol Biochem Behav. 2000 Oct;67(2):257-64.

[3]. Kjellberg B, et, al. Partial restoration by a neuroleptic (spiramide) of items of grooming behaviour suppressed by amphetamine. Arch Int Pharmacodyn Ther. 1974 Jul;210(1):61-6.

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