## Amisulpride

| Cat. No.: | HY-14545 |
| CAS No.: | 71675-85-9 |
| Molecular Formula: | C₁₇H₂₇N₃O₄S |
| 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 6 months -20°C 1 month |

### SOLVENT & SOLUBILITY

| In Vitro | H₂O : 0.2 mg/mL (0.54 mM; Need ultrasonic) |

### BIOLOGICAL ACTIVITY

**Description**
Amisulpride is a **dopamine D₂/D₃ receptor** antagonist with Kᵢs of 2.8 and 3.2 nM for human dopamine D₂ and D₃, respectively.

**IC₅₀ & Target**
Kᵢ: 2.8 nM (D₂ receptor), 3.2 nM (D₃ receptor)

**In Vitro**
Amisulpride is an atypical dopamine D₂/D₃ receptor antagonist with Kᵢs of 2.8 and 3.2 nM for human dopamine D₂ and D₃, respectively. Amisulpride (100 nM) inhibits quinpirole-elicited [³H]thymidine incorporation with an IC₅₀ value of 22±3 nM (n=3). Amisulpride slightly but significantly increases [³H]dopamine release from slices of the rat striatum (S₂/S₁=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].

**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].

### PROTOCOL

| Product Data Sheet |

**Inhibitors**
- Agonists
- Screening Libraries

[¹] www.MedChemExpress.com
The functional effects of Amisulpride at the dopamine D3 receptor subtype are assessed. Briefly, the mitogenic response elicited in NG108-15 neuroblastoma-glioma cells stably transfected with human dopamine D3 receptor cDNA by the addition of 10 nM quinpirole in the presence of 1 μM forskolin is quantified by the incorporation of [3H]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.

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
