Zuclopenthixol

Cat. No.: HY-A0163
CAS No.: 53772-83-1
Molecular Formula: C₂₂H₂₅ClN₂OS
Molecular Weight: 400.96
Target: Dopamine Receptor
Pathway: GPCR/G Protein; Neuronal Signaling
Storage:
- Powder -20°C 3 years
- Powder 4°C 2 years
- Powder -80°C 6 months
- Powder -20°C 1 month
- In solvent 4°C 2 years
- In solvent -80°C 6 months

SOLVENT & SOLUBILITY

In Vitro
DMSO: ≥ 150 mg/mL (374.10 mM)
* “≥” means soluble, but saturation unknown.

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Solvent Concentration</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 mM</td>
<td>2.4940 mL</td>
<td>12.4701 mL</td>
<td>24.9401 mL</td>
</tr>
<tr>
<td></td>
<td>5 mM</td>
<td>0.4988 mL</td>
<td>2.4940 mL</td>
<td>4.9880 mL</td>
</tr>
<tr>
<td></td>
<td>10 mM</td>
<td>0.2494 mL</td>
<td>1.2470 mL</td>
<td>2.4940 mL</td>
</tr>
</tbody>
</table>

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

BIOLOGICAL ACTIVITY

Description
Zuclopenthixol is a thioxanthene derivative which acts as a mixed dopamine D1/D2 receptor antagonist.

IC₅₀ & Target
D1/D2 receptor[1].

In Vivo
After acute treatment, Zuclopenthixol (0.2 and 0.4 mg/kg)-treated animals exhibit ethopharmacological profiles characterized by a decrease in offensive behaviors without impairment of motor activity (0.2 mg/kg). In contrast, the antiaggressive action of the highest dose used (0.4 mg/kg) is accompanied by a marked increase of immobility. After subchronic treatment, no tolerance to Zuclopenthixol antiaggressive or motor activity is observed[1].

Administration of Zuclopenthixol (0.7 and 1.4 mg/kg) significantly elevate MDA level compared to respective controls. Nevertheless, there is no difference between the two dose levels with respect to their effect on rat brain MDA level. Post hoc pairwise comparisons between the means of groups (n=12) receiving different dose levels of Zuclopenthixol reveal that administration of 1.4 mg/kg of Zuclopenthixol significantly reduces GSH level compared to both vehicle-treated and Zuclopenthixol (0.7 mg/kg)-treated animals (P<0.001). Nevertheless, the lower dose of the drug does not
affect rat brain GSH level. Animals receiving 0.7 or 1.4 mg/kg of Zuclopenthixol exhibits significantly higher GSH levels than SCO treated animals. Administration of 0.7 mg/kg of Zuclopenthixol significantly elevated GSHPx activity compared to vehicle treated animals.[2]

**PROTOCOL**

**Animal Administration**

**Mice**[1]

Zuclopenthixol (0.025-0.4 mg/kg) is administered acutely or subchronically for 10 days, on agonistic behavior elicited by isolation in male mice. Individually housed mice are exposed to anosmic “standard opponents” 30 min after the drug administration, and encounters are videotaped and evaluated using an ethologically based analysis[1].

**Rats**[2]

Male albino rats of Wistar strain weighing 200-250 g are used. They are kept in a temperature of 23-25°C with alternating 12-hour light and dark cycles and allowed free access to food and water. Animals are divided into six groups (n=6). Two groups receive two dose levels of Zuclopenthixol (0.7 and 1.4 mg/kg i.p.) 60 min and SCO (1.4 mg/kg i.p.) 30 min before decapitation. A third group of rats is injected with saline, with the same content of ethanol (20% v/v) and vegetable oil (2.8% v/v) in the test solution, 60 min and then SCO (1.4 mg/kg i.p.) 30 min before decapitation. The forth and fifth groups of rats receive two dose levels of Zuclopenthixol (0.7 and 1.4 mg/kg i.p.) 60 min and saline 30 min before decapitation. A control group of six animals is given saline, with the same content of ethanol (20% v/v) and vegetable oil (2.8% v/v) in the test solution, 60 min and then saline 30 min before decapitation and is run concurrently with drug-treated groups[2].

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

**REFERENCES**
