# MDL 105519

**Cat. No.:** HY-15085

**CAS No.:** 161230-88-2

**Molecular Formula:** $C_{18}H_{11}Cl_2NO_4$

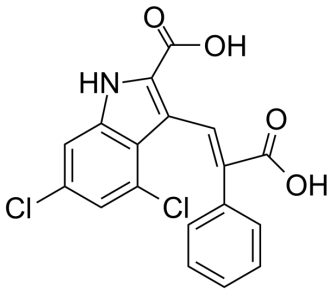
**Molecular Weight:** 376.19

**Target:** iGluR

**Pathway:** Membrane Transporter/Ion Channel; 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 : 17 mg/mL (45.19 mM; Need ultrasonic and warming)

| Preparing Stock Solutions | Solvent / Mass / Concentration | 1 mg | 5 mg | 10 mg |
|---|---|---|---|---|
| | 1 mM | 2.6582 mL | 13.2912 mL | 26.5823 mL |
| | 5 mM | 0.5316 mL | 2.6582 mL | 5.3165 mL |
| | 10 mM | 0.2658 mL | 1.3291 mL | 2.6582 mL |

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

## BIOLOGICAL ACTIVITY

**Description**

MDL 105519 is a potent and selective antagonist of glycine binding to the NMDA receptor.

**In Vitro**

MDL 105519 is a potent and selective ligand for the glycine recognition site that completely inhibit the binding of [$^3$H]glycine to rat brain membranes with a $K_i$ value of 10.9 nM. MDL 105519 is approximately 10,000-fold selective for the glycine recognition site relative to the other receptor types investigated. MDL 105519 inhibits NMDA-dependent responses, such as elevations of [$^3$H]TCP binding in brain membranes, cyclic GMP accumulation in brain slices, and alterations in cytosolic $Ca^{2+}$ and $Na^+$-$Ca^{2+}$ currents in cultured neurons. Inhibition is non-competitive with respect to NMDA and could be nullified with D-serine[1].

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

**In Vivo**

MDL 105519 is an NMDA receptor antagonist in vivo. Intravenously administration of MDL 105519 prevents harmaline-stimulated increases in cerebellar cyclic GMP content, providing biochemical evidence of NMDA receptor antagonism in vivo . This antagonism is associated with anticonvulsant activity in genetically based, chemically induced, and electrically mediated seizure models. Anxiolytic activity is observed in the rat separation-induced vocalization model, but muscle-relaxant activity is apparent at lower doses. Higher doses impair rotorod performance, but are without effect on mesolimbic

dopamine turnover or prepulse inhibition of the startle reflex[1].

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## PROTOCOL

**Animal Administration** [1]

Adult, male, CD rats are administered MK-801 (n=4, 2 mg/kg, i.p.) or MDL 105519 (n=4, 2 mg/kg, i.p.) and extracellular dopamine concentrations are measured using in vivo microdialysis[1].

Mice: Mice adult male CD-1 are injected with various doses of MDL 105519 (8, 16, 32, 64, 128 mg/kg) intraperitoneally and 30 min later are administered harmaline 50 mg/kg. Sixty minutes after the first injection, the mice are killed and cerebellar cGMP content is measured by radioimmunoassay[1].

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

## REFERENCES

[1]. Baron BM, et al. Pharmacological characterization of MDL 105,519, an NMDA receptor glycine site antagonist. Eur J Pharmacol. 1997 Apr 4;323(2-3):181-92.

**Caution: Product has not been fully validated for medical applications. For research use only.**

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