Harmine

Cat. No.: HY-N0737A
CAS No.: 442-51-3
Molecular Formula: C₁₃H₁₂N₂O
Molecular Weight: 212.25
Target: DYRK; 5-HT Receptor
Pathway: Protein Tyrosine Kinase/RTK; GPCR/G Protein; Neuronal Signaling
Storage: Powder
-20°C 3 years
4°C 2 years
In solvent
-80°C 1 year
-20°C 6 months

SOLVENT & SOLUBILITY

In Vitro
DMSO: ≥ 30 mg/mL (141.34 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

<table>
<thead>
<tr>
<th>Solvent Concentration</th>
<th>Mass 1 mg</th>
<th>Mass 5 mg</th>
<th>Mass 10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mM</td>
<td>4.7114 mL</td>
<td>23.5571 mL</td>
<td>47.1143 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td>0.9423 mL</td>
<td>4.7114 mL</td>
<td>9.4229 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td>0.4711 mL</td>
<td>2.3557 mL</td>
<td>4.7114 mL</td>
</tr>
</tbody>
</table>

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 (11.78 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (11.78 mM); Clear solution

BIOLOGICAL ACTIVITY

Description
Harmine is a natural dual-specificity tyrosine phosphorylation-regulated kinase (DYRK) inhibitor with anticancer and anti-inflammatory activities. Harmine has a high affinity of 5-HT₂A serotonin receptor, with an $K_i$ of 397 nM[1].

IC₅₀ & Target

| 5-HT₂A Receptor | IC₅₀ 397 nM (Kᵢ) | DYRK1A |

In Vitro
Harmine inhibits tau phosphorylation by DYRK1A by selected DANDYs, with an IC₅₀ of 190 nM[2]. Harmine negatively regulates homologous recombination (HR) by interfering Rad51 recruitment, resulting in severe cytotoxicity in hepatoma cells. Furthermore, NHEJ inhibitor Nu7441 markedly sensitizes Hep3B cells to the anti-proliferative effects of Harmine[3].
In Vivo

It is shown that brain water content is significantly increased in the TBI group. Treatment with Harmine significantly reduces the tissue water content at 1, 3 and 5 days, compared with the TBI group. Harmine treatment significantly reduces the escape latency at 3 and 5 days, compared with the TBI group. Post-TBI administration of Harmine significantly improves the motor function recovery of the rats at 1, 3 and 5 days following TBI, compared with the TBI group without Harmine treatment. The neuronal survival rate in the Harmine-treated group is significantly increased, compared with the TBI group. Administration of Harmine results in marked elevation in the expression of GLT-1, compared with the TBI group. The administration of Harmine significantly reduces the expression of caspase 3, compared with the TBI group[4].

PROTOCOL

Animal Administration [4]

Rats[4]

A total of 150 male Sprague-Dawley rats (age, 10-12 weeks; weighing, 280-320 g) are used in the present study. The rats are randomly divided into three groups: Sham-operated group (sham; n=15); the TBI group (TBI; n=35) and the TBI + Harmine-treated group (Harmine; n=35). Harmine is administered immediately following TBI (i.p, 30 mg/kg per day) for up to 5 days. The sham and TBI groups receive equal volumes of 0.9% saline solution (i.p.). The rats are grouped as follows for examination of behavioral recovery: Sham, n=3; TBI, n=7; and Harmine, n=7. Following TBI, the NSS is evaluated at 1, 3 and 5 days. Each rat is assessed by an observer who is blinded to the animal treatment[4].

CUSTOMER VALIDATION

- Int Immunopharmacol. 2023 May 5;119:110208.

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REFERENCES

[1]. Glennon RA, et al. Binding of beta-carbolines and related agents at serotonin (5-HT(2) and 5-HT(1A)), dopamine (D(2)) and benzodiazepine receptors. Drug Alcohol Depend. 2000 Aug 1;60(2):121-32.


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

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