Harmine

**Product Data Sheet**

**Harmine**

Cat. No.: HY-N0737A

CAS No.: 442-51-3

Molecular Formula: C₁₃H₁₂N₂O

Molecular Weight: 212.25

Target: 5-HT Receptor; RAD51; DYRK

Pathway: GPCR/G Protein; Neuronal Signaling; Cell Cycle/DNA Damage Protein Tyrosine Kinase/RTK

Storage:
- Powder: -20°C 3 years
- 4°C: 2 years
- In solvent: -80°C 6 months
- -20°C: 1 month

**Solvent & Solubility**

**In Vitro**

DMSO: 10 mg/mL (47.11 mM; Need ultrasonic and warming)

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Solvent Concentration</th>
<th>Mass for 1 mg</th>
<th>Mass for 5 mg</th>
<th>Mass for 10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 mM</td>
<td>4.7114 mL</td>
<td>23.5571 mL</td>
<td>47.1143 mL</td>
</tr>
<tr>
<td></td>
<td>5 mM</td>
<td>0.9423 mL</td>
<td>4.7114 mL</td>
<td>9.4229 mL</td>
</tr>
<tr>
<td></td>
<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.

**BIOLOGICAL ACTIVITY**

**Description**
Harmine, an inhibitor of DYRK1A and 5-HT₂A, with a Kᵢ of 397 nM for 5-HT₂A, could also negatively regulate HR by interfering Rad51 recruitment.

**IC₅₀ & Target**
5-HT₂A Receptor, Kᵢ: 397 nM; DYRK1A: RAD51

**In Vitro**
Harmine is an inhibitor of 5-HT₂A, with an Kᵢ of 397 nM[1]. Harmine inhibits tau phosphorylation by DYRK1A by selected DANDYs, with an IC₅₀ of 190 nM[2]. Harmine negatively regulates 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 GLT1, 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]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION


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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.

