Proglumide

Cat. No.: HY-B1330
CAS No.: 6620-60-6
Molecular Formula: C₁₈H₂₆N₂O₄
Molecular Weight: 334.41
Target: Cholecystokinin 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
DMSO: ≥ 65 mg/mL (194.37 mM)
* “≥” means soluble, but saturation unknown.

<table>
<thead>
<tr>
<th>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>2.9903 mL</td>
<td>14.9517 mL</td>
<td>29.9034 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td>0.5981 mL</td>
<td>2.9903 mL</td>
<td>5.9807 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td>0.2990 mL</td>
<td>1.4952 mL</td>
<td>2.9903 mL</td>
</tr>
</tbody>
</table>

Preparing Stock Solutions

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: ≥ 2.17 mg/mL (6.49 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.17 mg/mL (6.49 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.17 mg/mL (6.49 mM); Clear solution

BIOLOGICAL ACTIVITY

Description
Proglumide is a nonpeptide and orally active cholecystokinin (CCK)-A/B receptors antagonist. Proglumide selective blocks CCK’s effects in the central nervous system (CNS). Proglumide has ability to inhibit gastric secretion and to protect the gastroduodenal mucosa. Proglumide also has antiepileptic and antioxidant activities.[1][2][3][4][5].

IC₅₀ & Target
Cholecystokinin (CCK)-A/B receptors[1][2]
In Vitro

In an in vitro study, Proglumide at concentrations between 0.3-10 mM inhibits CCK-stimulated amylase release dose-dependently, while Proglumide does not influence the basal amylase release at concentrations between 0-3 mM. Dose-response curves to CCK for amylase release shifted to the right with increase in Proglumide concentration. This inhibition by Proglumide is reversible. In addition, the effect of Proglumide is selective for CCK and its related peptide[2]. The incubation of HT29 cells with Proglumide significantly reduces the [3H]-thymidine incorporation to HT29 cells in a dose-dependent manner, with an IC50 of 6.5 mM. Proglumide reduces in a dose-dependent manner the percentage of necrosis with a parallel increase of apoptosis up to 70%[3].

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

In Vivo

Proglumide (250-750 mg/kg; intraperitoneal injection; adult male Sprague Dawley rats) treatment is significantly effective in ameliorating the seizure activities, cognitive dysfunctions, and cerebral oxidative stress[1].

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

<table>
<thead>
<tr>
<th>Animal Model:</th>
<th>Adult male Sprague Dawley rats (200-250 g; 2 months old) are induced status epilepticus (SE)[1]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dosage:</td>
<td>250 mg/kg, 500 mg/kg, and 750 mg/kg</td>
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<tr>
<td>Administration:</td>
<td>Intraperitoneal injection</td>
</tr>
<tr>
<td>Result:</td>
<td>Dose-dependently and significantly increased the latencies to seizure and SE. Significantly and dose-dependently attenuated Li-PC (SE) induced increase in thiobarbituric acid (TBARS) and catalase (CAT), attenuated Li-Pc induced decrease in SOD, and attenuated depletion of GSH and glutathione-S transferase (GST) in the hippocampus and striatum.</td>
</tr>
</tbody>
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REFERENCES


