Propranolol hydrochloride

**Cat. No.:** HY-B0573  
**CAS No.:** 318-98-9  
**Molecular Formula:** C₁₆H₂₂ClNO₂  
**Molecular Weight:** 295.8

**Target:** Adrenergic Receptor; Autophagy  
**Pathway:** GPCR/G Protein; Autophagy

**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: ≥ 150 mg/mL (507.10 mM)  
H₂O: 33.33 mg/mL (112.68 mM; Need ultrasonic)  
* “≥” means soluble, but saturation unknown.*

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Solvent Concentration</th>
<th>Mass (1 mg)</th>
<th>Mass (5 mg)</th>
<th>Mass (10 mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 mM</td>
<td>3.3807 mL</td>
<td>16.9033 mL</td>
<td>33.8066 mL</td>
</tr>
<tr>
<td></td>
<td>5 mM</td>
<td>0.6761 mL</td>
<td>3.3807 mL</td>
<td>6.7613 mL</td>
</tr>
<tr>
<td></td>
<td>10 mM</td>
<td>0.3381 mL</td>
<td>1.6903 mL</td>
<td>3.3807 mL</td>
</tr>
</tbody>
</table>

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

### BIOLOGICAL ACTIVITY

**Description**  
Propranolol hydrochloride is a nonselective β-adrenergic receptor (βAR) antagonist with an IC₅₀ of 12 nM.

**IC₅₀ & Target**  
IC₅₀: 12 nM (βAR)[¹]

**In Vitro**  
In cultured endothelial or tumor cells, propranolol has been shown to both reduce cAMP levels and simultaneously activate the mitogen-activated protein kinase (MAPK) pathway downstream of βAR inhibition[²]. It displays high affinity for 5-HT₁B receptors (Kᵢ = 17 nM), and milder affinity for SHT₁D receptors (Kᵢ = 10.2 μM)[³].

**In Vivo**  
Chronic administration of propranolol increased the beta(1)-adrenoceptors but decreased the beta(2)-adrenoceptors without changing total amount of plasma membrane beta-adrenoceptors[⁴].
Male Wistar rats weighing 250–300 g are used in the study. Propranolol is dissolved with tap water, and given ad lib. The daily consumption of propranolol is estimated to be 40 mg/kg based on a mean intake of 35 mL/day of water for a 250 g rat. The treatment period of β-adrenoceptor antagonists is changed from 1 to 3 or 6 weeks and the effects are examined[4].

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

REFERENCES


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