Imipramine hydrochloride

Cat. No.: HY-B1490
CAS No.: 113-52-0
Molecular Formula: C₁₉H₂₅ClN₂
Molecular Weight: 316.87
Target: Serotonin Transporter
Pathway: 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**

H₂O : ≥ 34 mg/mL (107.30 mM)

*“≥” means soluble, but saturation unknown.

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Solvent Mass</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mM, 1.5779 mL</td>
<td>3.1559 mL</td>
<td>15.7793 mL</td>
<td>31.5587 mL</td>
<td></td>
</tr>
<tr>
<td>5 mM, 0.6312 mL</td>
<td>3.1559 mL</td>
<td>6.3117 mL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 mM, 0.3156 mL</td>
<td>1.5779 mL</td>
<td>3.1559 mL</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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

BIOLOGICAL ACTIVITY

**Description**

Imipramine hydrochloride inhibits serotonin transporter with an IC₅₀ value of 32 nM in vitro.

**IC₅₀ & Target**

IC₅₀: 32 nM (serotonin)[¹]

**In Vitro**

Depression-like behavior is often complicated by chronic pain. Antidepressants including imipramine are widely used to treat chronic pain, but the mechanisms are not fully understood[²]. Imipramine (IC₅₀=32 nM) and desipramine (IC₅₀=160 nM) are found to be potent inhibitors of the human placental serotonin transporter[¹].

**In Vivo**

Administration of imipramine reverses social avoidance behavior, significantly increasing the interaction time. 24 days of imipramine treatment in RSD mice significantly decreases stress-induced mRNA levels for IL-6 in brain microglia[³]. Chronic mild stress induces a long-term altered gene expression profile in the prefrontal cortex that is partially reverted by imipramine treatment (10mg/kg, i.p.)[⁴]. Chronic imipramine administration alters the amino acid dynamics in the brain. In the striatum, the concentrations of asparagine, glutamine and methionine are significantly
increased by chronic imipramine administration. In the thalamus and hypothalamus, chronic imipramine administration significantly decreased the valine concentration\[5\]. Imipramine reduces pain-related negative emotion without influencing pain and that this effect is diminished by denervation of 5-HT neurons and by anti-BDNF treatment. Imipramine also normalizes derangement of ERK/CREB coupling, which leads to induction of BDNF. This suggests a possible interaction between 5-HT and BDNF\[2\]. Imipramine treatment counteracts the corticosterone administration-induced increase in the reactivity of rat CA3 hippocampal circuitry to the activation of the 5-HT receptor\[6\].

### PROTOCOL

**Animal Administration**\[3\][5]

**Rats:** The Wistar (WIS) and Wistar Kyoto (WKY) rats are divided into four groups: (1) a control WIS rat group, (2) an imipramine-treated WIS rat group, (3) a control WKY rat group and (4) an imipramine-treated WKY rat group. Distilled water (10 mL/kg) or imipramine solution (10 mg/10 mL/kg) is orally administered for 28 days except on the day of the open field test, when nothing is administered in order to avoid the acute effect of single administration on the open field test\[5\].

**Mice:** C57BL/6 mice subjected to repeated social defeat (RSD), home cage control (HCC) are randomly selected into four groups: RSD/imipramine, RSD/vehicle, HCC/imipramine, and HCC/vehicle. Mice in the RSD/imipramine received daily intraperitoneal (i.p.) injections of imipramine (20 mg/kg) for 24 days after the 6 cycles of RSD. HCC/imipramine received daily i.p. imipramine at the same dose while RSD/vehicle and HCC/vehicle groups received i.p. injections of vehicle (sodium chloride, 0.9%) for 24 days at the same time point\[3\].

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

### REFERENCES


