Pyrrolidinedithiocarbamate ammonium

Cat. No.: HY-18738
CAS No.: 5108-96-3
Molecular Formula: C₅H₁₂N₂S₂
Molecular Weight: 164.29
Target: NF-κB
Pathway: NF-κB
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: ≥ 42 mg/mL (255.65 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>6.0868 mL</td>
<td>30.4340 mL</td>
<td>60.8680 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td>1.2174 mL</td>
<td>6.0868 mL</td>
<td>12.1736 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td>0.6087 mL</td>
<td>3.0434 mL</td>
<td>6.0868 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 >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (12.66 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (12.66 mM); Clear solution

BIOLOGICAL ACTIVITY

Description
Pyrrolidinedithiocarbamate ammonium is a selective NF-κB inhibitor.

IC₅₀ & Target
Target: NF-κB

In Vitro
Pretreatment of cells with pyrrolidinedithiocarbamate (3-1000 μM) dose-dependently attenuate IL-8 production.
Furthermore, pyrrolidinedithiocarbamate (100 μM) suppresses the accumulation of IL-8 mRNA.
Pyrrolidinedithiocarbamate inhibits the activation of NF-κB, because pyrrolidinedithiocarbamate suppresses both NF-κB DNA binding and NF-κB-dependent transcriptional activity. NF-κB inhibition with pyrrolidinedithiocarbamate
decrease IL-8 production by intestinal epithelial cells[1].

**In Vivo**

The DSS+pyrrolidinedithiocarbamate ammonium-treated groupII exhibits suppression of shortening of intestinal length and reduction of DAI score. Activated NF-κB level and IL-1β and TNF-α levels are significantly lower in DSS+pyrrolidinedithiocarbamate ammonium-treated groupII. These findings suggest that suppression of NF-κB activity by pyrrolidinedithiocarbamate ammonium can delay the healing of mucosal tissue defects (erosions or ulcers) arising from inflammation, but that it can strongly suppress the expression of inflammatory cytokines (IL-1β and TNF-α), resulting in significant alleviation of colitis. Pyrrolidinedithiocarbamate ammonium is useful for the treatment of ulcerative colitis[2].

**PROTOCOL**

**Cell Assay [1]**

The human colon cancer cell line HT-29 is obtained and cells are grown in modified McCoy’s 5A medium supplemented with 10% fetal bovine serum. To study the effect of pyrrolidinedithiocarbamate ammonium on IL-8 production, HT-29 cells in 96-well plates are induced with 20 ng/mL of IL-1β for 18 h. Various concentrations (3–1000 μM) of pyrrolidinedithiocarbamate or its vehicle (culture medium) are added to the cells 30 min prior to IL-1β stimulation. The concentration of IL-8 in the supernatant is determined using solid-phase enzyme-linked immunosorbent assay[1].

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

**Animal Administration**

**Animal Administration:** [2]Pyrrolidinedithiocarbamate is administered intraperitoneally to mice at dose levels of 100 and 50 mg/kg. Mice are divided into a DSS-untreated group (normal group), DSS-treated control group, DSS+pyrrolidinedithiocarbamate-treated group (low-dose group), and DSS+pyrrolidinedithiocarbamate-treated groupII (high-dose group). In each group, the disease activity index score (DAI score), intestinal length, histological score, and the levels of activated NF-κB and inflammatory cytokines (IL-1β and TNF-α) in tissue are measured[2].

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

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**REFERENCES**

