Prim-O-glucosylcimifugin

Cat. No.: HY-N0635  
CAS No.: 80681-45-4  
Molecular Formula: C₂₂H₂₈O₁₁  
Molecular Weight: 468.45  
Target: NO Synthase; COX  
Pathway: Immunology/Inflammation  
Storage: 4°C, protect from light  
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)

**SOLVENT & SOLUBILITY**

In Vitro  
DMSO : ≥ 150 mg/mL (320.20 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>Concentration</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 mM</td>
<td>2.1347 mL</td>
<td>10.6735 mL</td>
<td>21.3470 mL</td>
<td></td>
</tr>
<tr>
<td>5 mM</td>
<td>0.4269 mL</td>
<td>2.1347 mL</td>
<td>4.2694 mL</td>
<td></td>
</tr>
<tr>
<td>10 mM</td>
<td>0.2135 mL</td>
<td>1.0673 mL</td>
<td>2.1347 mL</td>
<td></td>
</tr>
</tbody>
</table>

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

**BIOLOGICAL ACTIVITY**

Description  
Prim-O-glucosylcimifugin exerts anti-inflammatory effects through the inhibition of iNOS and COX-2 expression by regulating JAK2/STAT3 signaling.

<table>
<thead>
<tr>
<th>IC₅₀ &amp; Target</th>
<th>iNOS</th>
<th>COX-2</th>
</tr>
</thead>
<tbody>
<tr>
<td>In Vitro</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Prim-O-glucosylcimifugin (POG) is the highest content chromone and one of the major active constituents in Radix Saposhnikoviae (RS), Prim-O-glucosylcimifugin exerts anti-inflammatory effects in RAW 264.7 macrophages through the inhibition of iNOS and COX-2 expression by inhibiting JAK2/STAT3 signaling. The cytotoxicity of Prim-O-glucosylcimifugin is measured to LPS-activated Raw 264.7 macrophages. Raw 264.7 macrophages are treated with LPS (1 μg/mL) and increasing concentrations of Prim-O-glucosylcimifugin (15, 50, and 100 μg/mL) for 24 h and cell viability is evaluated by CCK-8 assay. Cell viability is not significantly affected after 24 h and exposure to 15-100 μg/mL Prim-O-glucosylcimifugin as compared with DMSO-treated cells (control). To investigate the anti-inflammatory effect of Prim-O-glucosylcimifugin, whether Prim-O-glucosylcimifugin can affect NO synthesis is examined in LPS-activated RAW 264.7 cells. Macrophages are treated with LPS (1 μg/mL) and various concentrations of Prim-O-glucosylcimifugin (15, 50, and 100 μg/mL) for 24 h. No concentrations are measured in the culture supernatants by

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Griess reaction. The concentrations of NO in the culture supernatants are markedly increased in response to LPS exposure, and Prim-O-glucosylcimifugin significantly inhibits LPS-induced NO production in a concentration-dependent manner[1].

**In Vivo**

Bronchoalveolar lavage fluid (BALF) is collected at 7 h after lipopolysaccharide (LPS) administration and the cytokine levels in BALF are measured by ELISA. The levels of TNF-α, IL-1β and IL-6 in BALF are increased dramatically compared with control group. However, pretreatment with Prim-O-glucosylcimifugin (2.5, 5 or 10 mg/kg) significantly down-regulates the levels of TNF-α, IL-1β and IL-6 in a dose-dependent manner (P<0.05, P<0.01)[1].

**PROTOCOL**

**Cell Assay**[1]

Cell Counting Kit (CCK-8) is used to determine the cytotoxic concentrations of Prim-O-glucosylcimifugin. In brief, the Raw 264.7 cells are plated at a density of 1×10⁴ cells per well in a 96-well and incubated overnight. Cells are then stimulated with 1 μg/mL LPS and treated with various concentrations of Prim-O-glucosylcimifugin (15, 50, and 100 μg/ml; MedChem Express, Princeton, NJ, USA) or DMSO. After incubation at 37°C for 24 h, CCK-8 solution is added to each well and incubated for another 1 h. The absorbance is measured at 450 nm using a microplate reader[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

**Animal Administration**[1]

Mice[1] BALB/c male mice, 8 weeks old and weighing approximately 18 to 20 g, are used. The mice are randomly divided into five groups: Control group; LPS group; LPS+Prim-O-glucosylcimifugin (2.5, 5 or 10 mg/kg bodyweight). Prime-O-glucosylcimifugin is given intraperitoneally. One hour later, LPS group and LPS+Prim-O-glucosylcimifugin group mice are given 50 μL PBS intranasally (i.n) (200 mg/L) to induce acute lung injury. Control mice are given 50 μL PBS intranasally (i.n) without LPS[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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**REFERENCES**


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