**Astragalin**

**Cat. No.:** HY-N0015  
**CAS No.:** 480-10-4  
**Molecular Formula:** C₂₁H₂₀O₁₁  
**Molecular Weight:** 448.38  
**Target:** Apoptosis  
**Pathway:** Apoptosis  
**Storage:**  
- Powder: -20°C, 3 years  
- Powder: 4°C, 2 years  
- In solvent: -80°C, 6 months  
- In solvent: -20°C, 1 month

**SOLVENT & SOLUBILITY**

**In Vitro**

DMSO: ≥ 100 mg/mL (223.03 mM)  
*“≥” means soluble, but saturation unknown.*

<table>
<thead>
<tr>
<th>Concentration</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mM</td>
<td>2.2303 mL</td>
<td>11.1513 mL</td>
<td>22.3025 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td>0.4461 mL</td>
<td>2.2303 mL</td>
<td>4.4605 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td>0.2230 mL</td>
<td>1.1151 mL</td>
<td>2.230 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 >> 40% PEG300 >> 5% Tween-80 >> 45% saline  
   Solubility: ≥ 2.5 mg/mL (5.58 mM); Clear solution

2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)  
   Solubility: ≥ 2.5 mg/mL (5.58 mM); Clear solution

3. Add each solvent one by one: 10% DMSO >> 90% corn oil  
   Solubility: ≥ 2.5 mg/mL (5.58 mM); Clear solution

**BIOLOGICAL ACTIVITY**

**Description**

Astragalin (kaempferol-3-O-glucoside) is a flavonoid with anti-inflammatory activity and newly found in persimmon leaves and green tea seeds. IC50 value: Target: in vitro: Astragalin nontoxic at ≤ 20 μM suppressed cellular induction of Toll-like receptor 4 (TLR4) and ROS production enhanced by LPS. Both LPS and H2O2 induced epithelial eotaxin-1 expression, which was blocked by astragalin. LPS activated and induced PLCγ1, PKCβ2, and NADPH oxidase subunits of p22phox and p47phox in epithelial cells and such activation and induction were demoted by astragalin or TLR4 inhibition antagonizing eotaxin-1 induction. H2O2-upregulated phosphorylation of JNK and p38 MAPK was dampened by adding astragalin to epithelial cells,
while this compound enhanced epithelial activation of Akt and ERK. H2O2 and LPS promoted epithelial apoptosis concomitant with nuclear condensation or caspase-3 activation, which was blunted by astragalin [1]. Astragalin suppressed the expression of tumor necrosis factor α, interleukin 6, and nitric oxide in a dose-dependent manner in mMECs [2]. Astragalin attenuated the infiltration of inflammatory cells, the activity of myeloperoxidase (MPO) and the expression of tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6) and interleukin-1β (IL-1β) in a dose-dependent manner. Additionally, Western blotting results showed that astragalin efficiently blunt decreased nuclear factor-kappaB (NF-κB) activation by inhibiting the degradation and phosphorylation of IkBα and the nuclear translocation of p65 [3]. Astragalin significantly reduced LPS-induced expression of iNOS, COX-2 and cytokines/chemokines, and production of NO in J774A.1 mouse macrophages. Astragalin inhibited LPS-induced activation of NF-κB as indicated by inhibition of degradation of IkBα, nuclear translocation of NF-κB, and NF-κB dependent gene reporter assay [4].

**in vivo:** Mice were injected intraperitoneally (i.p.) with lipopolysaccharide (LPS) (dose range: 5-40 mg/kg). Pretreatment with astragalin can improve survival during lethal endotoxemia and attenuate inflammatory responses in a murine model of lipopolysaccharide-induced acute lung injury [4].

**REFERENCES**


