**Forsythoside B**

**Cat. No.:** HY-N0029  
**CAS No.:** 81525-13-5  
**Molecular Formula:** C₃₄H₄₄O₁₉  
**Molecular Weight:** 756.7  
**Target:** TNF Receptor; NF-κB  
**Pathway:** Apoptosis; 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: 125 mg/mL (165.19 mM; Need ultrasonic)

<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>1.3215 mL</td>
<td>6.6076 mL</td>
<td>13.2153 mL</td>
</tr>
<tr>
<td></td>
<td>5 mM</td>
<td>0.2643 mL</td>
<td>1.3215 mL</td>
<td>2.6431 mL</td>
</tr>
<tr>
<td></td>
<td>10 mM</td>
<td>0.1322 mL</td>
<td>0.6608 mL</td>
<td>1.3215 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: ≥ 2.08 mg/mL (2.75 mM); Clear solution

2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)  
   Solubility: ≥ 2.08 mg/mL (2.75 mM); Clear solution

3. Add each solvent one by one: 10% DMSO >> 90% corn oil  
   Solubility: ≥ 2.08 mg/mL (2.75 mM); Clear solution

### BIOLOGICAL ACTIVITY

**Description**  
Forsythoside B is a phenylethanoid glycoside isolated from the leaves of Lamiophlomis rotata Kudo, a Chinese folk medicinal plant for treating inflammatory diseases and promoting blood circulation. Forsythoside B could inhibit TNF-alpha, IL-6, IkB and modulate NF-κB.

<table>
<thead>
<tr>
<th>IC₅₀ &amp; Target</th>
<th>TNF-α</th>
<th>NF-κB</th>
</tr>
</thead>
</table>

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### In Vitro
Forsythoside B concentration-dependently down-regulates the levels of TNF-α, IL-6 and high-mobility group-box 1 protein (HMGB1) in lipopolysaccharide (LPS)-stimulated RAW264.7 cells, inhibits the IkB kinase (IKK) pathway and modulated nuclear factor (NF)-κB.[1]

### In Vivo
Intravenous injection of forsythoside B alone or plus imipenem reduces serum levels of TNF-α, IL-6, HMGB1, triggering receptor expressed on myeloid cells (TREM-1) and endotoxin, while the serum level of IL-10 is up-regulated and myeloperoxidase (MPO) in lung, liver and small intestine is reduced.[1] Forsythoside B at doses higher than 8 mg/kg produces a significant neuroprotective potential in cerebral ischemia and reperfusion rats. Forsythoside B (20 mg/kg) demonstrates significant neuroprotective activity even after delayed administration at 1 h, 3 h and 5 h after cerebral ischemia and reperfusion. Forsythoside B 20 mg/kg attenuates histopathological damage as demonstrated by smaller brain infarct size and brain edema, decreased cerebral Evans blue extravasation and myeloperoxidase activity, inhibited cerebral phosphor-IkB-α and NF-κB expression.[2] Forsythoside B shows a significant recovery in myocardial function with improvement of LVSP and +/-dp/dt(max). The myocardial infarct volume, serum levels of Tn-T, TNF-alpha and IL-6, content of MDA and MPO activity in myocardial tissue are all reduced, protein expression of HMGB1, phosphor-I kappaB-alpha and phosphor-NF-kappaB are down-regulated, while attenuate the decrease of SOD and GPx activities.[3]

### PROTOCOL

#### Cell Assay [1]
Forsythoside B is dissolved in sterile saline solution and added to the medium at various concentrations (from 0.1 to 10 μM) and incubated with LPS stimulated RAW264.7 cells. Cell-free supernatants are collected after Forsythoside B treatment for 24 h. Cell viability is assessed by measuring lactate dehydrogenase (LDH) in the medium.[1]

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

#### Animal Administration [2]
Rats: Forsythoside B is dissolved in sterilized saline. For the dose–response study, forsythoside B at doses of 1.3, 3.2, 8, 20 or 50 mg/kg is administered as an intravenous bolus injection at 15 min after reperfusion. The sham or vehicle-treated rats are injected with saline. Neurological deficits are determined at 23 h after reperfusion followed by brain infarct volume examination.[2]

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

### REFERENCES

