**Tubeimoside I**

**Cat. No.:** HY-N0890  
**CAS No.:** 102040-03-9  
**Molecular Formula:** C₆₃H₉₈O₂₉  
**Molecular Weight:** 1319.44  
**Target:** Apoptosis  
**Pathway:** Apoptosis  
**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: ≥ 100 mg/mL (75.79 mM)  
*"≥" means soluble, but saturation unknown.*

<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>1 mM</td>
<td></td>
<td>0.7579 mL</td>
<td>3.7895 mL</td>
<td>7.5790 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td></td>
<td>0.1516 mL</td>
<td>0.7579 mL</td>
<td>1.5158 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td></td>
<td>0.0758 mL</td>
<td>0.3789 mL</td>
<td>0.7579 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 (1.89 mM); Clear solution

2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)  
   Solubility: ≥ 2.5 mg/mL (1.89 mM); Clear solution

3. Add each solvent one by one: 10% DMSO >> 90% corn oil  
   Solubility: ≥ 2.5 mg/mL (1.89 mM); Clear solution

**BIOLOGICAL ACTIVITY**

**Description**
Tubeimoside I (Lobatoside-H) is an extract from Chinese herbal medicine Bolbostemma paniculatum (MAXIM.) FRANQUET (Cucurbitaceae) has been shown as a potent anti-tumor agent for a variety of human cancers. IC50 value: Target: Anticancer natural compound in vitro: TBMS I inhibited the proliferation of both HepG2 and L-02 cells in a dose- and time-dependent manner, but HepG2 cells appeared more sensitive to the agent. When exposed to TBMS I for 24, 48 and 72 h, IC50 for HepG2 cells versus L-02 cells were 15.5 vs. 23.1, 11.7 vs. 16.2, 9.2 vs. 13.1 (μM, p<0.01), respectively. TBMS I induced cell shrinkage, nuclear condensation and fragmentation, cell cycle arrest at the G2/M phase, mitochondrial membrane disruption, release...
of cytochrome c from the mitochondria, activation of caspase 3 and 9, and shifting Bax/Bcl-2 ratio from being anti-apoptotic to pro-apoptotic, all indicative of initiation and progression of apoptosis involving mitochondrial dysfunction [1]. TBMS1-induced molecular events were related to mitochondria-induced intrinsic apoptosis and P21-cyclin B1/cdc2 complex-related G2/M cell cycle arrest [2]. TBMS1 combined with CDDP promoted cell apoptosis, decreased proliferation activity and increased cytosolic Ca2+ levels. Bcl-2 protein expression was down-regulated but Bax was up-regulated. Moreover, GST-π mRNA and protein expression were decreased. TBMS1 reduced the resistance of the cells to CDDP-induced cytotoxicity [4]. Treatment with TBMS1 resulted in dose- and time-dependent inhibition of proliferation, led to arrest in phase G2/M of the cell cycle and increased the levels of intracellular Ca2+. Furthermore, TBMS1 up-regulated the levels of the glucose-regulated protein 78/immunoglobulin heavy chain binding protein (GRP78/Bip), C/EBP homologous protein (CHOP), Bax, and cleaved caspase-3 and down-regulated the levels of Bcl-2 [5].

in vivo: TBMS1 significantly inhibited the production of the pro-inflammatory cytokines, TNF-α, IL-6 and IL-1β in vitro and in vivo. Pretreatment with TBMS1 markedly attenuated the development of pulmonary edema, histological severities and inflammatory cells infiltration in mice with ALI [3].

REFERENCES


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