IKarisoside A

Cat. No.: HY-N0875
CAS No.: 55395-07-8
Molecular Formula: \( \text{C}_{26}\text{H}_{28}\text{O}_{10} \)
Molecular Weight: 500.49
Target: Others
Pathway: Others
Storage: Please store the product under the recommended conditions in the COA.

Solvent & Solubility

In Vitro 10 mM in DMSO

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mM</td>
<td>1.9980 mL</td>
<td>9.9902 mL</td>
<td>19.9804 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td>0.3996 mL</td>
<td>1.9980 mL</td>
<td>3.9961 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td>0.1998 mL</td>
<td>0.9990 mL</td>
<td>1.9980 mL</td>
</tr>
</tbody>
</table>

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

BIOLOGICAL ACTIVITY

Description
IKarisoside A (Ikarisoside-A) is a natural compound isolated from Epimedium koreanum (Berberidaceae); has anti-inflammatory properties. IC50 value: Target: in vitro: Ikarisoside A inhibited the expression of LPS-stimulated inducible nitric oxide synthase (iNOS) and the production of nitric oxide (NO) in LPS-stimulated RAW 264.7 cells and mouse bone marrow-derived macrophages (BMMs) in a concentration-dependent manner. In addition, Ikarisoside A reduced the release of pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-alpha) and interleukin-1 beta (IL-1 beta). Furthermore, Ikarisoside A inhibited the activity of p38 kinase and nuclear factor-kappaB (NF-kappaB) [1]. Ikarisoside A is a potent inhibitor of osteoclastogenesis in RANKL-stimulated RAW 264.7 cells as well as in bone marrow-derived macrophages. The inhibitory effect of Ikarisoside A resulted in decrease of osteoclast-specific genes like matrix metalloproteinase 9 (MMP9), tartrate-resistant acid phosphatase (TRAP), receptor activator of NF-kappaB (RANK), and cathepsin K. Moreover, Ikarisoside A blocked the resorbing capacity of RAW 264.7 cells on calcium phosphate-coated plates. Ikarisoside A also has inhibitory effects on the RANKL-mediated activation of NF-kappaB, JNK, and Akt [2].

REFERENCES

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