Acacetin

**Cat. No.:** HY-N0451  
**CAS No.:** 480-44-4  
**Molecular Formula:** C₁₆H₁₂O₅  
**Molecular Weight:** 284.26  
**Target:** Apoptosis; Autophagy  
**Pathway:** Apoptosis; Autophagy  
**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: ≥ 37 mg/mL (130.16 mM)  
*“≥” means soluble, but saturation unknown.*

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Concentration</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 mM</td>
<td>3.5179 mL</td>
<td>17.5895 mL</td>
<td>35.1791 mL</td>
</tr>
<tr>
<td></td>
<td>5 mM</td>
<td>0.7036 mL</td>
<td>3.5179 mL</td>
<td>7.0358 mL</td>
</tr>
<tr>
<td></td>
<td>10 mM</td>
<td>0.3518 mL</td>
<td>1.7590 mL</td>
<td>3.5179 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 >> 90% corn oil  
Solubility: ≥ 2.08 mg/mL (7.32 mM); Clear solution

**BIOLOGICAL ACTIVITY**

**Description**  
Acacetin (5,7-Dihydroxy-4'-methoxyflavone) is an orally active flavonoid derived from Tephroseris kirilowii (Turcz.) Holub. Acacetin docks in the ATP binding pocket of PI3Kγ. Acacetin causes cell cycle arrest and induces apoptosis and autophagy in cancer cells. Acacetin has potent anti-cancer and anti-inflammatory activity and has the potential for pain-related diseases research\[1\][2].

**In Vitro**  
Acacetin (5,7-Dihydroxy-4'-methoxyflavone; 10-200 μM; 24 hours) decreases cell viabilities in a dose-dependent manner. Acacetin has little effect on human normal glial cell line HEB and non-tumorigenic epithelial cell line MCF-10A\[1\]. Acacetin (50-150 μM; 24 hours) causes G2/M cell cycle arrest and induces apoptosis and autophagy\[1\]. Acacetin (50-150 μM; 24 hours) leads to decreases in levels of PI3K-p110, p-AKT, p-mTOR, p-p70S6K, and p-ULK in a dose-dependent manner\[1\]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
<table>
<thead>
<tr>
<th>Assay</th>
<th>Cell Line</th>
<th>Concentration</th>
<th>Incubation Time</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cell Viability Assay</td>
<td>Breast cancer MCF-7 cells, hepatocellular carcinoma SMMC-7721 cells, lung adenocarcinoma A549 cells, esophageal carcinoma Eca109 cells</td>
<td>10, 20, 40, 60, 80, 100, 150, 200 μM</td>
<td>24 hours</td>
<td>Decreased cancer cell viabilities in a dose-dependent manner. Had IC\textsubscript{50} values of 82.75 μM, 103.9 μM, 157.4 μM, 54.7 μM in MDA-MB-231, MCF-7, A549, Eca109 cells, respectively.</td>
</tr>
<tr>
<td>Cell Cycle Analysis</td>
<td>MDA-MB-231 cells</td>
<td>50, 100, 150 μM</td>
<td>24 hours</td>
<td>Resulted in increase in percentage of cells at G2/M phase and decrease in percentage of cells at G1 and S phase in a dose-dependent manner.</td>
</tr>
<tr>
<td>Apoptosis Analysis</td>
<td>MDA-MB-231 cells</td>
<td>50, 100, 150 μM</td>
<td>24 hours</td>
<td>Induced apoptosis.</td>
</tr>
<tr>
<td>Cell Autophagy Assay</td>
<td>MDA-MB-231 cells</td>
<td>50, 100, 150 μM</td>
<td>24 hours</td>
<td>Induced autophagy. Resulted in marked increases in EGFP-LC3 puncta formation and a dose-dependent accumulation of LC3-II.</td>
</tr>
<tr>
<td>Western Blot Analysis</td>
<td>MDA-MB-231 cells</td>
<td>50, 100, 150 μM</td>
<td>24 hours</td>
<td>Resulted in decrease in levels of Bcl-2 and Bcl-xL and increase in levels of p53. Led to decreases in levels of PI3K\textgamma-p110, p-AKT, p-mTOR, p-p70S6K, and p-ULK in a dose-dependent manner. Had little or no effect on expression of PI3K\alpha, PI3K\beta, PI3K\delta, p-ERK, p-p38, and p-JNK.</td>
</tr>
</tbody>
</table>
In Vivo

Acacetin (5,7-Dihydroxy-4'-methoxyflavone; 5, 20 mg/kg/day; orally; for 3 days) significantly suppresses microglial activation in an LPS-induced neuroinflammation mouse model[2].

Acacetin (25 mg/kg/day; orally; for 3 days) reduces neuronal cell death in an animal model of ischemia[2].

Acacetin (1.8-56.2 mg/kg/day; ip; single dose) decreases visceral and inflammatory nociception and prevented the formalin-induced oedema[3].

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

<table>
<thead>
<tr>
<th>Animal Model:</th>
<th>Male C57BL/6 mice, 7 weeks of age[2]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dosage:</td>
<td>5, 20 mg/kg</td>
</tr>
<tr>
<td>Administration:</td>
<td>Orally, once a day for 3 days</td>
</tr>
<tr>
<td>Result:</td>
<td>Significantly suppressed microglial activation in an LPS-induced (ip; 5mg/kg) neuroinflammation mouse model.</td>
</tr>
</tbody>
</table>

CUSTOMER VALIDATION

- Pharmacol Res. 2020 May;155:104751.

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REFERENCES

