Formononetin

**Cat. No.:** HY-N0183  
**CAS No.:** 485-72-3  
**Molecular Formula:** C₁₆H₁₂O₄  
**Molecular Weight:** 268.26  
**Target:** FGFR  
**Pathway:** Protein Tyrosine Kinase/RTK  
**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: ≥ 35 mg/mL (130.47 mM)  
*“≥” means soluble, but saturation unknown.

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Solvent Concentration</th>
<th>Mass</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 mM</td>
<td>3.7277 mL</td>
<td>18.6386 mL</td>
<td>37.2773 mL</td>
<td></td>
</tr>
<tr>
<td></td>
<td>5 mM</td>
<td>0.7455 mL</td>
<td>3.7277 mL</td>
<td>7.4555 mL</td>
<td></td>
</tr>
<tr>
<td></td>
<td>10 mM</td>
<td>0.3728 mL</td>
<td>1.8639 mL</td>
<td>3.7277 mL</td>
<td></td>
</tr>
</tbody>
</table>

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

**BIOLOGICAL ACTIVITY**

**Description**

Formononetin (Formononetol; Flavosil) is a bioactive component extracted from the red clover; inhibits the proliferation of DU-145/PC-3 cells in a dose-dependent manner. IC50 value: Target: anti-cancer in vitro: formononetin inhibited the proliferation of DU-145 cells in a dose-dependent manner. DU-145 cells treated with different concentrations of formononetin displayed obvious morphological changes of apoptosis under fluorescence microscopy. In addition, formononetin increased the proportion of early apoptotic DU-145 cells, down-regulated the protein levels of Bcl-2 and up-regulated those of RASD1 and Bax [1]. Formononetin significantly inhibited the cell growth of PC-3 in a dose-dependent manner, but no such effect was observed in RWPE1 cells. Formononetin treatment contributed to the reduced Bcl-2 protein level and the elevated Bax expression in PC-3 cells, thereby resulting in the increasing Bax/Bcl-2 ratios. Furthermore, the phosphorylated level of p38 in PC-3 cells was activated through the FN treatment, whereas the endogenous Akt phosphorylation was blocked [2]. Compared with the control, formononetin inhibited the proliferation of MCF-7 cells and effectively induced cell cycle arrest. The levels of p-IGF-1?R, p-Akt, cyclin D1 protein expression, and cyclin D1 mRNA expression were also downregulated [3]. In vivo: formononetin also prevented the tumor growth of human breast cancer cells in nude mouse xenografts [3].
REFERENCES

