C75

Cat. No.: HY-12364
CAS No.: 218137-86-1
Molecular Formula: C₁₄H₂₂O₄
Molecular Weight: 254.32
Target: Fatty Acid Synthase (FAS)
Pathway: Metabolic Enzyme/Protease
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 : ≥ 83.3 mg/mL (327.54 mM)
* "≥" means soluble, but saturation unknown.

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Solvent Mass Concentration</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></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% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (9.83 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (9.83 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (9.83 mM); Clear solution

BIOLOGICAL ACTIVITY

Description
C75 is a synthetic fatty-acid synthase (FASN) inhibitor; inhibits prostate cancer cells PC3 with an IC₅₀ of 35 μM.

IC₅₀ & Target
IC₅₀: 35 μM (PC3 cell)[1]

In Vitro
C75 inhibits PC3 cell growth with an IC₅₀ of 35 μM at 24 h. C75 (10-50 μM) also reduces the growth of LNCaP
spheroids in a concentration-dependent manner with an IC$_{50}$ of 50 μM$^{[1]}$. (-)-C75 inhibits FAS activity and has a cytotoxic effect on tumor cell lines, without affecting food consumption. (+)-C75 inhibits CPT1 and its administration produces anorexia, suggesting that central inhibition of CPT1 is essential for the anorectic effect of C75. The differential activity of C75 enantiomers may lead to the development of potential new specific drugs for cancer and obesity$^{[2]}$.

**In Vivo**

C75 blocks fasting-induced c-Fos expression in the arcuate nucleus (Arc), lateral hypothalamic area (LHA), and paraventricular nucleus (PVN) 10–24 h after i.p. injection. Intraperitoneal administration of C75 at 30 mg/kg body weight inhibits food intake of mice by ≥95% within 2 h after i.p. injection$^{[3]}$. C75-treated DIO mice has a 50% greater weight loss, and a 32.9% increased production of energy because of fatty acid oxidation. C75 treatment of rodent adipocytes and hepatocytes and human breast cancer cells increases fatty acid oxidation and ATP levels by increasing CPT-1 activity, even in the presence of elevated concentrations of malonyl-CoA$^{[4]}$.

**PROTOCOL**

**Cell Assay**$^{[1]}$

Cells are seeded in 96-well plates and incubated for 2 days to allow exponential phase growth. Cells are then rinsed twice with PBS and treated with C75. After 24 or 48 h incubation, MTT is added to a final concentration of 0.5 mg/ml and cultures are incubated for 2 h. Cells are then solubilized with DMSO before measuring absorbance at 570 nm. Cell growth is also measured, using MTT assay, every 24 h up to 96 h$^{[1]}$.

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

**Animal Administration**$^{[3]}$

Mice: C75 is administered either by i.p. (i.p.; 30 mg/kg of body weight) or i.c.v. (10 μg in 3 μL of RPMI medium 1640) injection. One, 11.5, and 24 h after i.p. injection, cumulative food intake is measured, mice are killed, brains are sectioned, and slices are subjected to immunohistochemical staining for c-Fos. All i.p. injections are given 1 h before the start of the dark cycle. For i.c.v. injection, mice are anesthetized with metofane and given 3 μL of RPMI medium 1640 (control) or C75 in RPMI medium 1640 into the lateral ventricle with a calibrated 10-μL Hamilton syringe$^{[3]}$.

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

**REFERENCES**


