Oleylethanolamide

**Cat. No.:** HY-107542  
**CAS No.:** 111-58-0  
**Molecular Formula:** C₂₀H₃₉NO₂  
**Molecular Weight:** 325.53  
**Target:** Endogenous Metabolite; PPAR  
**Pathway:** Metabolic Enzyme/Protease; Cell Cycle/DNA Damage  
**Storage:** Powder -20°C 3 years  
In solvent -80°C 6 months  
-20°C 1 month

### SOLVENT & SOLUBILITY

**In Vitro**

DMSO : 20.83 mg/mL (63.99 mM; Need ultrasonic)  
H₂O : < 0.1 mg/mL (insoluble)

<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></td>
<td>3.0719 mL</td>
<td>15.3596 mL</td>
<td>30.7191 mL</td>
</tr>
<tr>
<td></td>
<td>5 mM</td>
<td></td>
<td>0.6144 mL</td>
<td>3.0719 mL</td>
<td>6.1438 mL</td>
</tr>
<tr>
<td></td>
<td>10 mM</td>
<td></td>
<td>0.3072 mL</td>
<td>1.5360 mL</td>
<td>3.0719 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: ≥ 2.08 mg/mL (6.39 mM); Clear solution

2. Add each solvent one by one: 10% DMSO >> 90% corn oil  
Solubility: ≥ 2.08 mg/mL (6.39 mM); Clear solution

### BIOLOGICAL ACTIVITY

**Description**  
Oleylethanolamide is a high affinity endogenous PPAR-α agonist, which plays an important role in the treatment of obesity and arteriosclerosis.

**IC₅₀ & Target**  
<table>
<thead>
<tr>
<th>Human Endogenous Metabolite</th>
<th>PPAR-α</th>
</tr>
</thead>
</table>

**In Vitro**  
Oleylethanolamide (OEA), an endogenous PPAR-α ligand, attenuates liver fibrosis targeting hepatic stellate cells. Oleylethanolamide suppresses TGF-β1 induced hepatic stellate cells (HSCs) activation in vitro via PPAR-α. To assess the impact of Oleylethanolamide on HSCs activation, the expression levels of α-SMA and Col1a in TGF-β1-
stimulated HSCs are examined by qPCR. The mRNA levels of α-SMA and Col1a are markedly induced in the group of CFSC cells with TGF-β1 (5 ng/mL) stimulation for 48h, while the mRNA levels are suppressed when treated with Oleoylethanolamide in a dose-dependent manner. Immunofluorescence and western blot results show that Oleoylethanolamide treatment dose-dependently inhibits the protein expression of α-SMA, the marker of HSC activation. The inhibitory effects of Oleoylethanolamide on HSCs activation are completely blocked by PPAR-α antagonist MK886 (10 μM). Moreover, the mRNA and protein expression levels of PPAR-α are down-regulated with TGF-β1 stimulation, while Oleoylethanolamide treatment restores these changes in dose-dependent manner. In addition, the phosphorylation of Smad 2/3 is upregulated in the presence of TGF-β1 stimulation, consistent with the observed effects on HSC activation, while Oleoylethanolamide (10 μM) reduces the phosphorylation of Smad2/3 in CFSC simulated with TGF-β1[1].

In Vivo

Oleoylethanolamide (OEA) can significantly suppress the pro-fibrotic cytokine TGF-β1 negatively regulate genes in the TGF-β1 signaling pathway (α-SMA, collagen 1a, and collagen 3a) in mice models of hepatic fibrosis. Treatment with Oleoylethanolamide (5 mg/kg/day, intraperitoneal injection, i.p.) significantly attenuates the progress of liver fibrosis in both two experimental animal models by blocking the activation of hepatic stellate cells (HSCs)[1].

PROTOCOL

Cell Assay [1]

CFSC, HSC cell lines are first obtained from cirrhotic rat liver, and have a similar phenotype to that of early passage primary HSCs. CFSC cells are cultured in Dulbecco’s modified Eagle’s medium (DMEM) supplemented with 10% fetal bovine serum (FBS) and 1% penicillin/streptomycin. All cells are cultured in 6-well culture plates under 37°C and 5% CO₂ in an incubator. The medium is replaced every two days, and the cells are harvested and diluted at a ratio of 1:3 twice a week. In experiments, HSCs are pretreated with the experimental concentration of Oleoylethanolamide (30 μM, 10 μM, 3 μM) before stimulation with 5 ng/mL TGF-β1. mRNA expression levels of α-SMA (A) and Col1a (B) are analyzed by real-time PCR[1].

Animal Administration [1]

Mice[1]

The Sv/129 mice and PPAR-α knockout mice are maintained in a room with controlled temperature (21-23°C), humidity (55-60%) and lighting (12 h light/dark cycles) and given water ad libitum. Mice are randomly divided for methionine choline-deficient (MCD) and thioacetamide (TAA) experiments. In the MCD-diet feeding experiment, wild-type Sv/129 mice and PPAR-α knockout mice are each divided into three groups (n=8/group): (i) control group receive normal diet; (ii) fed with MCD diet and injected with the vehicle (5% Tween-80+5% PEG400+90% saline, 5 mL/kg/day, 8 weeks, intraperitoneal injection, i.p.); (iii) fed with MCD diet along with Oleoylethanolamide administration (5 mg/kg/day; 8 weeks, i.p.). In another set of experiment, all the wild-type mice and PPAR-α knockout mice are given standard chow diet, and are randomly separated into three groups: the control group is not administrated TAA or Oleoylethanolamide but is injected with the saline; the TAA group is injected with TAA (160 mg/kg, three times per week, 6 weeks, dissolved in saline, i.p.) plus the corresponding vehicle: the Oleoylethanolamide group is both injected with TAA and Oleoylethanolamide (5 mg/kg/day; 6 weeks, i.p.)[1].

REFERENCES

Caution: Product has not been fully validated for medical applications. For research use only.

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