Oleoylethanolamide

Cat. No.: HY-107542

CAS No.: 111-58-0

Molecular Formula: $C_{20}H_{39}NO_2$

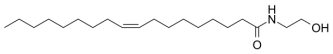
Molecular Weight: 325.53

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)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.0719 mL	15.3596 mL	30.7191 mL
	5 mM	0.6144 mL	3.0719 mL	6.1438 mL
	10 mM	0.3072 mL	1.5360 mL	3.0719 mL

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

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

IC_{50} & Target

Human Endogenous Metabolite	PPAR-α

In Vitro

Oleoylethanolamide (OEA), an endogenous PPAR-α ligand, attenuates liver fibrosis targeting hepatic stellate cells. Oleoylethanolamide suppresses TGF-β1 induced hepatic stellate cells (HSCs) activation in vitro via PPAR-α. To assess the impact of Oleoylethanolamide 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]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
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]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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% CO_2 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]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
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]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Chen L, et al. Oleoylethanolamide, an endogenous PPAR-α ligand, attenuates liver fibrosis targeting hepatic stellate cells. Oncotarget. 2015 Dec 15;6(40):42530-40

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

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