Daidzein

Cat. No.: HY-N0019

CAS No.: 486-66-8

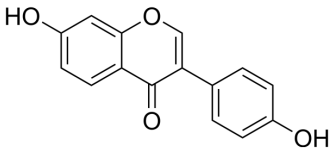
Molecular Formula: $C_{15}H_{10}O_4$

Molecular Weight: 254.24

Target: PPAR; Endogenous Metabolite

Pathway: Cell Cycle/DNA Damage; Metabolic Enzyme/Protease; Vitamin D Related/Nuclear Receptor

Storage: 4°C, stored under nitrogen
* In solvent : -80°C, 1 year; -20°C, 6 months (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (196.66 mM)

H_2O : 0.1 mg/mL (0.39 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.9333 mL	19.6665 mL	39.3329 mL
5 mM	0.7867 mL	3.9333 mL	7.8666 mL
10 mM	0.3933 mL	1.9666 mL	3.9333 mL

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

In Vivo

1. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 20 mg/mL (78.67 mM); Suspended solution; Need ultrasonic
2. 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
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (9.83 mM); Clear solution

BIOLOGICAL ACTIVITY

Description Daidzein is a soy isoflavone, which acts as a PPAR activator.

IC_{50} & Target	PPAR-α	PPAR-γ

In Vitro In 3T3-L1 adipocytes, Daidzein inverses the attenuation of adiponectin gene expression by co-culture, and these effects are inhibited by the PPAR-γ specific inhibitor. Daidzein attenuates the reduction of adiponectin expression in adipocytes, and a

	PPAR-γ specific inhibitor abrogated this effect. Direct activation of PPAR-α and-γ by Daidzein is confirmed by a luciferase reporter assay. In HEK293T cells, Daidzein significantly increases PPAR-α transcriptional activity in a concentration-dependent manner. Although an obvious dose-dependency is not observed in PPAR-γ transcriptional activity, Daidzein also significantly increases PPAR-γ transcriptional activity over a similar range of concentrations at which Daidzein enhanced PPAR-α transcriptional activity, with a maximum increase at 25 μM[1]. Daidzein is a soy isoflavone, which upregulates the expression of Abcg1, and it promotes axonal outgrowth in cultured hippocampal neurons via estrogen receptor signaling. Daidzein is a major component of soy with structural similarity to estrogen. It exerts an anti-inflammatory effect, lowers lipid levels, and increases mitochondrial biogenesis. As an activator of nuclear receptor peroxisome proliferator-activated receptors (PPARs), Daidzein enhances transcription of PPARs-dependent genes, including liver X receptors (LXRs, Nr1h gene family in mice). Incubation with different concentrations of Daidzein, from 5 to 100 μM, increases APOE transcriptional activity[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Treating Apoe KO mice with Daidzein increases Lxr and Abca1 gene expression at 1 month after stroke, showing that the absence of ApoE does not interfere with other cholesterol homeostasis genetic programs. Therefore, the findings suggest that Daidzein-induced ApoE upregulation is a critical component in fostering functional recovery in chronic stroke[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	HEK293T cells are plated on 24-well plates at a cell density of approximately 2.5×10^4 cells/well and are grown to 70-80% confluence. Cells are then transiently transfected with a PPAR-α or PPAR-γ expression plasmid, and a plasmid containing the luciferase gene under the control of three tandem PPAR response elements (PPRE × 3 TK-luciferase) using an X-treme GENE HP DNA Transfection Reagent. Renilla luciferase control vectors are co-transfected to control for transfection efficiency. After transfection, cells are cultured for another 24 h in medium containing DMSO or various concentrations (6.25, 12.5, 25 μM) of Daidzein. Cells are lysed, and luciferase activity is measured and expressed as fold induction, that is normalized to the activity of the renilla luciferase control plasmid[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice[2] Experiments are performed in 10- to 11-week-old male C57 (C57 bl/6) and Apoe KO (C57 background) mice. For long-term stroke recovery, mice receive Moxifloxacin (100 mg/kg) for 3 d. The prophylactic antibiotic treatment is shown to effectively reduce mortality in an animal model of stroke by attenuating peripheral infection. In addition, saline is subcutaneously administered daily, and hydrogel (Clear H_2O) is given to prevent dehydration. With the implementation of poststroke care (antibiotic regimen, rehydration, and feeding hydrogels with soft diet) during the acute period (<1 week), mice start to regain their body weight by day 5 and continue to recover from stroke. Animals are randomly selected for vehicle or Daidzein treatment. Vehicle or Daidzein (10 mg/kg) is administered subcutaneously within 30 min of reperfusion after confirming the reperfusion of blood flow, daily for 7 d and then every other day up to 1 month. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Rep Med. April 20, 2022.
- J Ethnopharmacol. 2024 Jan 24:117824.
- Mol Neurobiol. 2023 Dec 8.
- Eur J Pharmacol. 2022 Mar 15;919:174805.
- Eur J Pharmacol. 2020 Oct 15;885:173399.

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REFERENCES

[1]. Sakamoto Y1, et al. The Dietary Isoflavone Daidzein Reduces Expression of Pro-Inflammatory Genes through PPARα/γ and JNK Pathways in Adipocyte and Macrophage Co-Cultures. PLoS One. 2016 Feb 22;11(2):e0149676.

[2]. Kim E, et al. Daidzein Augments Cholesterol Homeostasis via ApoE to Promote Functional Recovery in Chronic Stroke. J Neurosci. 2015 Nov 11;35(45):15113-26.

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