Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

Daidzein

Catalog No: tcsc2332

Available Sizes

Size: 100g

Specifications

CAS No:
486-66-8

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

Pathway:
Cell Cycle/DNA Damage

Target:
PPAR

Purity / Grade:
>98%

Solubility:
DMSO : ≥ 50 mg/mL (196.66 mM)

Observed Molecular Weight:
254.24

Product Description

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

IC50 & Target: PPAR[1]

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].

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].