

## 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.

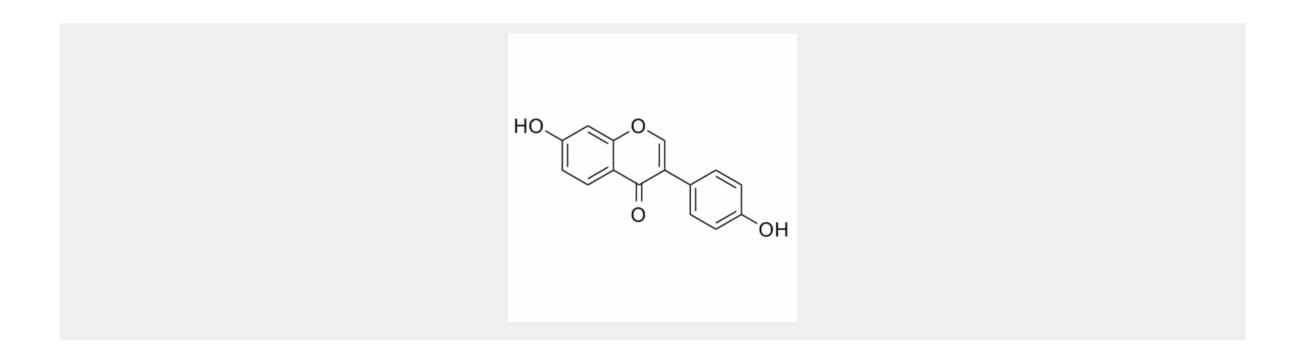
IC50 & Target: PPAR<sup>[1]</sup>

*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- $\gamma$  specific inhibitor. Daidzein attenuates the reduction of adiponectin expression in adipocytes, and a PPAR- $\gamma$  specific inhibitor abrogated this effect. Direct activation of PPAR- $\alpha$  and- $\gamma$  by Daidzein is confirmed by a luciferase reporter assay. In HEK293T cells, Daidzein significantly increases PPAR- $\alpha$  transcriptional activity in a concentration-dependent manner. Although an



obvious dose-dependency is not observed in PPAR- $\gamma$  transcriptional activity, Daidzein also significantly increases PPAR- $\gamma$  transcriptional activity over a similar range of concentrations at which Daidzein enhanced PPAR- $\alpha$  transcriptional activity, with a maximum increase at 25  $\mu$ M<sup>[1]</sup>. 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  $\mu$ M, increases APOE transcriptional activity<sup>[2]</sup>.

**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<sup>[2]</sup>.



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