Taiclone®
Quality Guarantee

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

Curcumin

Catalog No: tcsc1490

Available Sizes

Size: 100mg

Size: 500mg

Specifications

CAS No:
458-37-7

Formula:
$C_{21}H_{20}O_6$

Pathway:
Epigenetics;Autophagy;Epigenetics;NF-κB;Autophagy

Target:
Epigenetic Reader Domain;Autophagy;Histone Acetyltransferase;Keap1-Nrf2;Mitophagy

Purity / Grade:
>98%

Solubility:
DMSO :72.0 mg/mL (195.5 mM)
Ethanol: Insoluble
Water: Insoluble

Storage Instruction:
Powder -20°C for 3 years; Insolvent -80°C for 12 months

Alternative Names:
Turmeric yellow; Natural Yellow 3; Diferuloylmethane

Observed Molecular Weight:
368.38

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Notes

Formulation: 5%DMSO+10%PEG300+5%Tween80+80%water 4 mg/mL

Product Description

Curcumin is a natural phenolic compound with diverse pharmacologic effects including anti-inflammatory, antioxidant, antiproliferative and antiangiogenic activities. Curcumin is an inhibitor of p300 histone acetylatransferase (**(HATs)**) and also shows inhibitory effects on **NF-κB** and **MAPKs**.

IC50 & Target: Keap1-Nrf2[1], Histone acetyltransferase[6]

In Vitro: Curcumin exerts its chemopreventive effects partly through the activation of nuclear factor (erythroid-2 related) factor 2 (Nrf2) and its antioxidant and phase II detoxifying enzymes[1]. Curcumin inhibits T47D cells growth, with IC_{50}s of 25, 19 and 17.5 μM for 24, 48 and 72 h MTT assays respectively. IC_{50}s of curcumin and silibinin mixture against T47D cells, are 17.5, 15, and 12 μM for 24, 48, and 72 h exposure times, respectively[2]. Curcumin (2.5-80 μM) induces apoptotic cell death in AGS and HT-29 cell lines, and the IC_{50} is 21.9±0.1, 40.7±0.5 μM, respectively, in both AGS and HT-29 cell lines. Curcumin-induced apoptosis requires caspase activities in AGS and HT-29 cells. Curcumin induces ER Ca^{2+} decline and mitochondrial Ca^{2+} overloading[3]. Curcumin induces the G2/M cell cycle arrest of LNCaP and PC-3 cells in a dose dependent manner. Curcumin upregulates the protein level of NF-kappaB inhibitor IkappaBalpha and downregulates protein levels of c-Jun and AR[5].

In Vivo: Curcumin (10 mg/kg, p.o.) significantly prevents decrease in the percentage of sucrose consumption, as compared to the CMS-exposed rats. Curcumin treatment results in significant prevention of increase in TNF-α and IL-6 levels in stressed rats[4]. Curcumin decreases binding of p300/CREB-binding protein (CBP) at the brain-derived neurotrophic factor (BDNF) promoter at 20 mg/kg (i.p.), reduces binding of P300/CBP at the BDNF promoter at 40 mg/kg, and decreases binding all the four proteins of p300/CBP and H3K9ac/H4K5ac at the BDNF promoter at 60 mg/kg in chronic constriction injury (CCI) rats[6].