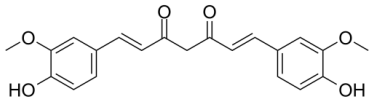
Data Sheet

Product Name:	Curcumin
Cat. No.:	CS-1490
CAS No.:	458-37-7
Molecular Formula:	C21H20O6
Molecular Weight:	368.38
Target:	Autophagy; Epigenetic Reader Domain; Ferroptosis; Histone Acetyltransferase; Influenza Virus; Keap1-Nrf2; Mitophagy
Pathway:	Anti-infection; Apoptosis; Autophagy; Epigenetics; NF-κB
Solubility:	DMSO : ≥ 100 mg/mL (271.46 mM); H2O : < 0.1 mg/mL (insoluble)



BIOLOGICAL ACTIVITY:

Curcumin (Diferuloylmethane) 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].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: Curcumin is dissolved in 10%DMSO.[2]T47D breast cancer cell line is grown in RPMI 1640 supplemented with 10% FBS, 2 mg/mL sodium bicarbonate, 0.05 mg/mL penicillin G, 0.08 mg/mL streptomycin. Culture is maintained on plastic flask and incubated at 37°C in 5% CO_2. After growing sufficient amount of cells, cytotoxic effect of silibinin and curcumin is studied by 24, 48 and 72 h MTT assays in which 1000 cell/well are cultivated in a 96 well plate. After 24 h incubation in 37°C with humidified atmosphere containing 5% CO_2, the cells are treated with serial concentrations of curcumin (5, 10, 20, 30, 40, 50, 60, 80, 100 μM), silibinin (20, 40, 60, 80, 100, 120, 140, 180, 200 μM), and curcumin-silibinin mixture (each of them 5, 10, 20, 30, 40, 50, 60, 80, 100 μM) for 24, 48 and 72 h in the quadruplicate manner, in addition to cells with 200 μL culture medium containing 10% DMSO for control. After incubation, the medium of all wells of the plate are exchanged with fresh medium and the cells are leaved for 24 h in incubator. Then, medium of all wells are removed carefully and 50 μL of 2 mg/mL MTT dissolved in PBS is added to each wells and the plate is covered with aluminum foil and incubated for 4.5 h again. After removing content of the wells, 200 μL pure DMSO is added to the wells. Then, 25 μL Sorensen's glycine buffer is added and immediately absorbance of each wells is read in 570 nm using EL×800 Microplate Absorbance Reader with reference wavelength of 630 nm. **Animal Administration:** Curcumin is suspended in saline.[4]Curcumin (10 mg/kg), freshly suspended in saline, is administrated by oral gavage once a day for 3 weeks. Forty rats are randomLy assigned to 4 groups (n=10/each

group): group I receives saline and serves as control, group II receives curcumin, group III is exposed to CMS andreceive saline and group IV are subjected to CMS andreceive curcumin.

References:

[1]. Gao S, et al. Curcumin attenuates arsenic-induced hepatic injuries and oxidative stress in experimental mice through activation of Nrf2 pathway, promotion of arsenic methylation and urinary excretion. Food Chem Toxicol. 2013 Jul 18. pii: S0278-6915(13)004

[2]. Nasiri M, et al. Curcumin and Silibinin Inhibit Telomerase Expression in T47D Human Breast Cancer Cells. Asian Pac J Cancer Prev. 2013;14(6):3449-53.

[3]. Cao A, et all. Curcumin induces apoptosis in human gastric carcinoma AGS cells and colon carcinoma HT-29 cells through mitochondrial dysfunction and endoplasmic reticulum stress. Apoptosis. 2013 Jul 24. [Epub ahead of print]

[4]. Jiang H, et al. Antidepressant-like effects of curcumin in chronic mild stress of rats: Involvement of its anti-inflammatory action. Prog Neuropsychopharmacol Biol Psychiatry. 2013 Jul 20. pii: S0278-5846(13)00150-4.

[5]. Guo H, et al. Curcumin induces cell cycle arrest and apoptosis of prostate cancer cells by regulating the expression of IkappaBalpha, c-Jun and androgen receptor. Pharmazie. 2013 Jun;68(6):431-4.

[6]. Zhu X, et al. Curcumin alleviates neuropathic pain by inhibiting p300/CBP histone acetyltransferase activity-regulated expression of BDNF and cox-2 in a rat model. PLoS One. 2014 Mar 6;9(3):e91303.

CAIndexNames:

1,6-Heptadiene-3,5-dione, 1,7-bis(4-hydroxy-3-methoxyphenyl)-, (1E,6E)-

SMILES:

O=C(CC(/C=C/C1=CC=C(O)C(OC)=C1)=O)/C=C/C2=CC=C(O)C(OC)=C2

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