Chrysophanol

Cat. No.: HY-13595

CAS No.: 481-74-3

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

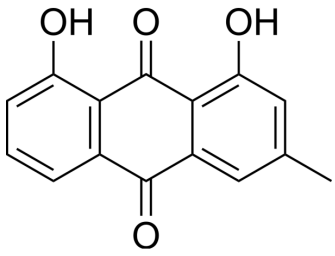
Molecular Weight: 254.24

Target: EGFR

Pathway: JAK/STAT Signaling; Protein Tyrosine Kinase/RTK

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMF : 4 mg/mL (15.73 mM; Need ultrasonic)

DMSO : 2 mg/mL (7.87 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (ultrasonic) (insoluble)

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.

BIOLOGICAL ACTIVITY

Description

Chrysophanol (Chrysophanic acid) is a natural anthraquinone, which inhibits EGF-induced phosphorylation of EGFR and suppresses activation of AKT and mTOR/p70S6K.

IC_{50} & Target

EGFR

In Vitro

Chrysophanol (Chrysophanic Acid) blocks proliferation of colon cancer cells by inhibiting EGFR/mTOR pathway. Chrysophanol, a natural anthraquinone, has anticancer activity in EGFR-overexpressing SNU-C5 human colon cancer cells. Chrysophanol treatment in SNU-C5 cells inhibits EGF-induced phosphorylation of EGFR and suppresses activation of downstream signaling molecules, such as AKT, extracellular signal-regulated kinase (ERK) and the mammalian target of Rapamycin (mTOR)/ribosomal protein S6 kinase (p70S6K). Chrysophanol (80 and 120 μM) significantly blocks cell proliferation when combined with the mTOR inhibitor, Rapamycin. Chrysophanol inhibits EGF-induced phosphorylation of EGFR and suppresses activation of AKT and mTOR/p70S6K, and significantly blocks cell proliferation. Chrysophanol dose dependently decreases CCK-8 and the viability of EGFR-overexpressing SNU-C5 cells. Chrysophanol treatment dose-

	dependently decreases EGF induced phosphorylation of EGFR at Tyr1068. Chrysophanol (80 and 120 μM) reduces the phosphorylation levels of mTOR at Ser2448. Chrysophanol (80 and 120 μM) also decreases the phosphorylation levels of p70S6K at Thr389. Chrysophanol inhibits EGF-induced EGFR activation and suppresses activation of the downstream signaling molecules, AKT and mTOR/p70S6K[1]. Chrysophanol (CA) inhibits lipid accumulation in 3T3-L1 adipocytes. Chrysophanol down-regulates adipogenic factors in 3T3-L1 adipocytes. Chrysophanol induces thermogenic factors in primary cultured brown adipocytes. Chrysophanol suppresses adipogenesis and induces thermogenesis via activation of AMPK pathway[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Chrysophanol (CA) improves HFD-induced obesity in C57BL/6 Mice. The in vivo performance of Chrysophanol is performed in male C57BL/6J mice to determine the efficacy of administered Chrysophanol. Mice fed the HFD gained significantly more weight than those fed the standard diet mice. On the other hand, weight gain of Chrysophanol group is significantly less than with the untreated HFD. Mice in the HFD-group gained 23.92 ± 1.74 g of weight, while those in the Chrysophanol group gained 16.72±2 g of weight after 16 weeks[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	The cells are seeded at 5×10^3 cells/mL in 96-well microplates and allowed to attach for 24 h. Chrysophanol (20, 50, 80 and 120 μM) is added to the medium at various concentrations up to 120 μM and for different durations. After treatment, cell cytotoxicity and/or proliferation is assessed by a Cell Counting Kit-8 (CCK-8). Briefly, highly water-soluble tetrazolium salt, WST-8, produces an orange colored water-soluble product, formazan. The amount of formazan dye generated by dehydrogenases in cells is directly proportional to the number of living cells.CCK-8 (10 μL) is added to each well and incubated for 3 h at 37°C, then cell proliferation and cytotoxicity are assessed by measuring the absorbance at 450 nm using a microplate reader.Three replicated wells are used for each experimental condition[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice[2] Male 4-week-old C57BL/6J mice are maintained for 1 week prior to experiments. Mice are maintained on a 12-h light/dark cycle in a pathogen-free animal facility, provided with laboratory diet and water ad libitum. To induce obesity, the mice are fed a HFD with 60% kcal% fat. Control group (C) are fed a commercial standard chow diet. HFD group (HFD) mice are fed with HFD only. HFD plus CA group (CA) Mice are fed with HFD for 4 weeks before administration of Chrysophanol (5 mg/kg/day). The mice are divided into three groups (n = 5) that are fed chow diet, HFD, and HFD plus Chrysophanol for 16 weeks. Body weight and food intake are measured three times per week. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Acta Pharm Sin B. 2019 Jul;9(4):782-793.
- Molecules. 2020 Apr 23;25(8):1980.
- Research Square Preprint. 2021 Nov.

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REFERENCES

[1]. Lee MS, et al. Chrysophanic acid blocks proliferation of colon cancer cells by inhibiting EGFR/mTOR pathway. Phytother Res. 2011 Jun;25(6):833-7.

[2]. Lim H, et al. Chrysophanic Acid Suppresses Adipogenesis and Induces Thermogenesis by Activating AMP-Activated Protein Kinase Alpha In vivo and In vitro. Front Pharmacol. 2016 Dec 8;7:476.

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