Xanthohumol

Cat. No.: HY-N1067

CAS No.: 6754-58-1

Molecular Formula: $C_{21}H_{22}O_5$

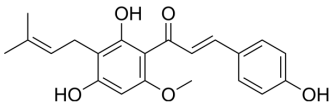
Molecular Weight: 354.4

Target: COX; Acyltransferase; Apoptosis; Influenza Virus; HSV; CMV

Pathway: Immunology/Inflammation; Metabolic Enzyme/Protease; Apoptosis; Anti-infection

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 83.33 mg/mL (235.13 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.8217 mL	14.1084 mL	28.2167 mL
	5 mM	0.5643 mL	2.8217 mL	5.6433 mL
	10 mM	0.2822 mL	1.4108 mL	2.8217 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (5.87 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (5.87 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Xanthohumol is one of the principal flavonoids isolated from hops, the inhibitor of diacylglycerol acetyltransferase (DGAT), COX-1 and COX-2, and shows anti-cancer and anti-angiogenic activities. Xanthohumol also has antiviral activity against bovine viral diarrhea virus (BVDV), rhinovirus, HSV-1, HSV-2 and cytomegalovirus (CMV).

IC_{50} & Target

COX-1	COX-2	HSV-1	HSV-2
DGAT1 40 μM (IC_{50})	DGAT2 40 μM (IC_{50})	CMV	

In Vitro

Xanthohumol significantly attenuates ADP-induced blood platelet aggregation, and significantly reduces the expression of

fibrinogen receptor (activated form of GPIIbIIIa) on platelets' surface[1].
Xanthohumol (5-50 nM) reduces the frequency of spontaneously occurring Ca^{2+} sparks and Ca^{2+} waves in control myocytes and in cells subjected to Ca^{2+} overload caused by: (1) exposure to low K^+ solutions, (2) periods of high frequency electrical stimulation, (3) exposures to isoproterenol or (4) caffeine. Xanthohumol (50-100 nM) reduces the rate of relaxation of electrically- or caffeine-triggered Ca^{2+} transients, without suppressing I_{Ca}, but this effect is small and reversed by isoproterenol at physiological temperatures. Xanthohumol also suppresses the Ca^{2+} content of the SR, and its rate of recirculation[2].
Treatment of endothelial cells with Xanthohumol leads to increased AMPK phosphorylation and activity. Functional studies using biochemical approaches confirm that AMPK mediates Xanthohumol anti-angiogenic activity. AMPK activation by Xanthohumol is mediated by CAMMKβ, but not LKB1. Analysis of the downstream mechanisms shows that Xanthohumol-induced AMPK activation reduces nitric oxide (NO) levels in endothelial cells by decreasing eNOS phosphorylation. Finally, AKT pathway is inactivated by Xanthohumol as part of its anti-angiogenic activity, but independently from AMPK, suggesting that these two signaling pathways proceed autonomously[3].
Xanthohumol significantly reduces cell viability and induces apoptosis via pro-caspase-3/8 cleavage and poly(ADP ribose) polymerase (PARP) degradation. Pro-caspase-9 cleavage, Bcl2 family expression changes, mitochondrial dysfunction, and intracellular ROS generation also participate in Xanthohumol-induced glioma cell death. Xanthohumol's inhibition of the IGFBP2/AKT/Bcl2 pathway via miR-204-3p targeting plays a critical role in mediating glioma cell death[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [3]

In vitro cell proliferation/viability is measured by the MTT test at different time points. 1000 cells/well are plated into 96-multiwell plates in complete medium. Following adhesion, medium is replaced with fresh medium containing the different treatments or vehicle (DMSO in medium). Xanthohumol and EGCG are used in a concentration range from 2.5 to 40 μM, up to 96 hours. 3 hours before each time point, MTT reagent (3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide) is added to the wells and plates are incubated at 37°C. At the indicated time points, absorbance at 540 nm is then measured by a FLUOstar spectrophotometer.
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Acta Pharm Sin B. 2021 Jan;11(1):143-155.
- Biomed Pharmacother. 2020 Sep;129:110369.
- Phytother Res. 2023 Mar 7.
- Food Funct. 2019 Dec 11;10(12):7865-7874.
- Eur J Pharmacol. 2023 Dec 8:176227.

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REFERENCES

[1]. Luzak B, et al. Xanthohumol from hop cones (Humulus lupulus L.) prevents ADP-induced platelet reactivity. Arch Physiol Biochem. 2016 Nov 18:1-7

[2]. Arnaiz-Cot JJ, et al. Xanthohumol modulates calcium signaling in rat ventricular myocytes: Possible Antiarrhythmic properties. J Pharmacol Exp Ther. 2016 Nov 4. pii: jpet.116.236588

[3]. Gallo C, et al. Hop derived flavonoid xanthohumol inhibits endothelial cell functions via AMPK activation. Oncotarget. 2016 Aug 1

[4]. Chen PH, et al. The miR-204-3p-targeted IGFBP2 pathway is involved in xanthohumol-induced glioma cell apoptotic death. Neuropharmacology. 2016 Nov;110(Pt

A):362-75.

[5]. Buckwold VE, et al. Antiviral activity of hop constituents against a series of DNA and RNA viruses. Antiviral Res. 2004 Jan;61(1):57-62.

[6]. Inokoshi J, et al. Expression of two human acyl-CoA:diacylglycerol acyltransferase isozymes in yeast and selectivity of microbial inhibitors toward the isozymes. J Antibiot (Tokyo). 2009;62(1):51-54.

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