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

Xanthohumol

Catalog No: tcsc6024

Available Sizes

Size: 5mg

Size: 10mg

Size: 25mg

Specifications

CAS No:
6754-58-1

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

Pathway:
Metabolic Enzyme/Protease;Immunology/Inflammation

Target:
Acyltransferase;COX

Purity / Grade:
>98%

Solubility:
DMSO : ≥ 150 mg/mL (423.25 mM)

Observed Molecular Weight:
354.4

Product 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.

In Vitro:

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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].