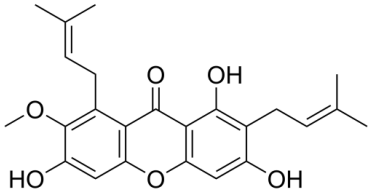
Data Sheet

Product Name:	alpha-Mangostin
Cat. No.:	CS-6435
CAS No.:	6147-11-1
Molecular Formula:	$C_{24}H_{26}O_6$
Molecular Weight:	410.46
Target:	Apoptosis; Bacterial; Fungal; Reactive Oxygen Species; Virus Protease
Pathway:	Anti-infection; Apoptosis; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB
Solubility:	H_2O : < 0.1 mg/mL (ultrasonic);DMSO : 110 mg/mL (ultrasonic)



BIOLOGICAL ACTIVITY:

alpha-Mangostin (α-Mangostin) is a dietary xanthone with broad biological activities, such as antioxidant, anti-allergic, antiviral, antibacterial, anti-inflammatory and anticancer effects. It is an inhibitor of mutant IDH1 (**IDH1-R132H**) with a K_i of 2.85 μM. IC50 & Target: IC50: 2.85 μM (IDH1-R132H)[1] ***In Vitro:*** alpha-Mangostin (α-Mangostin) exhibits a selective inhibitory effect on IDH1-R132H, but not on IDH1. alpha-Mangostin (α-Mangostin) competitively inhibits the binding of alpha-mangostin (α-KG) to IDH1-R132H. The structure–relationship study reveals that alpha-Mangostin (α-Mangostin) exhibits the strongest core inhibitor structure. alpha-Mangostin (α-Mangostin) selectively promotes demethylation of 5-methylcytosine (5mC) and histone H3 trimethylated lysine residues in IDH1 (+/R132H) MCF10A cells[1]. Cell proliferation significantly decreases in a dose-dependent manner in the cells treated with alpha-mangostin. Alpha-mangostin also increases the levels of Bax (pro-apoptotic), cleaved caspase-3, cleaved caspase-9 and cleaved-poly(ADP-ribose) polymerase (PARP)[2]. alpha-Mangostin (α-Mangostin) significantly inhibits light-induced degeneration of photoreceptors and 200 μM H_2O_2-induced apoptosis of RPE cells. 200 μM H_2O_2-induced generation of reactive oxygen species (ROS) and light-induced generation of malondialdehyde (MDA) are suppressed by alpha-Mangostin (α-Mangostin)[3]. ***In Vivo:*** alpha-Mangostin (α-Mangostin) reduces risk of liver fibrosis through the decrease in p53 expression as compared to the TAA_DMSO treatment. The serum levels of the liver enzymes AST and ALT after treatment with α-mangostin decrease as compared to DMSO alone[4].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: [1]IDH1$^{+/+}$ and IDH1 MCF10A cells are grown in DMEM/F-12 media, supplemented with 5% horse serum, 20 ng/mL EGF, 0.5 μg/mL hydrocortisone, 10 μg/mL insulin. IDH1$^{+/+}$ and IDH1 MCF10A cells are seeded in 6 well plates. After an exposure to 5 μM alpha-mangostin. cells are collected after indicated times and the viable cell number is calculated, using hemacytometer counting[1]. **Animal Administration:** Alpha-mangostin is prepared in 80% DMSO, 20% water.[4]Rats: Male Wistar rats are divided into 3 groups and treated with intraperitoneal injections of TAA (200 mg/kg). One subgroup is left untreated whereas the other two are treated either with 100 mg/kg alpha-mangostin or vehicle alone (80% DMSO, 20% water), which are administered intraperitoneally 3 times per weekfor a total of4 weeks. The incidence offibrotic nodules on the liver and the serum levels of the liver enzymes aspartate transaminase (AST) and alanine transaminase (ALT) are measured[4].

References:

[1]. Kim HJ, et al. Discovery of α-mangostin as a novel competitive inhibitor against mutant isocitrate dehydrogenase-1. Bioorg Med Chem Lett. 2015 Dec

1;25(23):5625-31.

[2]. Lee HN, et al. Antitumor and apoptosis-inducing effects of α-mangostin extracted from the pericarp of the mangosteen fruit (Garcinia mangostana L.) in YD-15 tongue mucoepidermoid carcinoma cells. Int J Mol Med. 2016 Apr;37(4):939-48.

CAIndexNames:

9H-Xanthen-9-one, 1,3,6-trihydroxy-7-methoxy-2,8-bis(3-methyl-2-buten-1-yl)-

SMILES:

O=C1C2=C(OC3=C1C(C/C=C(C)\C)=C(OC)C(O)=C3)C=C(O)C(C/C=C(C)\C)=C2O

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