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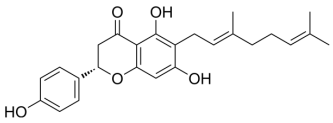
Bonannione A

Cat. No.:	HY-124896
CAS No.:	97126-57-3
Molecular Formula:	$C_{25}H_{28}O_5$
Molecular Weight:	408.49
Target:	Phosphatase; Apoptosis; Autophagy
Pathway:	Metabolic Enzyme/Protease; Apoptosis; Autophagy
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Bonannione A (6-Geranylnaringenin; Mimulone), a prenylflavonoid, is an orally active and potent protein tyrosine phosphatase 1B (PTP1B) inhibitor with an IC_{50} of 14 μM. Bonannione A triggers caspase-dependent apoptosis. Bonannione A induces autophagy through p53-mediated AMPK/mTOR pathway. Bonannione A shows anti-inflammatory, antiradical and anti-cancer activity[1][2][3].

In Vitro

Bonannione A (0-80 μM; 12, 24 hours) significantly inhibits cell proliferation in a dose- and time-dependent way in cancer cell lines[2].

Bonannione A (0-60 μM; 24 hours) triggers caspase-dependent apoptosis in A549 cells. Bonannione A increases accumulation of cells at the apoptotic sub-G1 phase and the number of cells at G2/M phase[2].

Bonannione A (60 μM, 24 h) triggers autophagy without impairment of autophagic flux in A549 cells. Bonannione A remarkably reduced p53 levels[2].

Bonannione A (60 μM, 0-24 h) remarkably decreased the levels of p53 and phospho-mTOR[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[2]

Cell Line:	Human lung cancer A549, breast cancer MCF-7, colon cancer HCT116 and osteosarcoma U2OS cells
Concentration:	20, 40, 60, 80 μM
Incubation Time:	12, 24 h
Result:	Significantly inhibited cell proliferation in a dose- and time-dependent way in these cancer cell lines.

Apoptosis Analysis[2]

Cell Line:	A549 cells
Concentration:	0-60 μM
Incubation Time:	24 h
Result:	Annexin V/PI-positive cells were markedly increased in a dose- and time-dependent way. Induced apoptosis through caspase-3 activation and PARP cleavage.

Cell Cycle Analysis[2]

Cell Line:	A549 cells
Concentration:	0-60 μM
Incubation Time:	24 h
Result:	Increased accumulation of cells at the apoptotic sub-G1 phase in a dose-dependent manner. The number of cells at G2/M phase also increased in a dose-dependent manner.

Cell Autophagy Assay[2]

Cell Line:	A549 cells
Concentration:	60 μM
Incubation Time:	24 h
Result:	Remarkably increased ATG7 and LC3-II protein levels in a dose-dependent way, but Beclin-1 level was slightly augmented.

Western Blot Analysis[2]

Cell Line:	A549 cells
Concentration:	60 μM
Incubation Time:	0-24 h
Result:	Remarkably decreased the levels of p53 and phospho-mTOR. Significantly increased whereas the levels of phospho-AMPK and phospho-Acetyl-CoA Carboxylase (ACC), an AMPK substrate.

In Vivo

Bonannione A (25 mg/kg; gavage; 48 and 24 h prior to DSS and every 24 h on the following days; for 5days) ameliorates the symptoms of colitis and delayed their onset[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Wistar rats (180-220 g)[3]
Dosage:	25 mg/kg
Administration:	Gavage; 48 and 24 h prior to DSS (10% (w/v)) and every 24 h on the following days; for 5days
Result:	Ameliorated the symptoms of colitis and delayed their onset. Showed the low DAI on the last day of the experiment.

REFERENCES

[1]. Lai-Bin Zhang, et al. Isoprenylated Flavonoids with PTP1B Inhibition from Macaranga denticulate. Nat Prod Bioprospect. 2016 Feb;6(1):25-30.

[2]. Hyun-Kyu An, et al. Mimulone-induced autophagy through p53-mediated AMPK/mTOR pathway increases caspase-mediated apoptotic cell death in A549 human lung cancer cells. PLoS One. 2014 Dec 9;9(12):e114607.

[3]. Zora Vochyánová, et al. Diplacone and mimulone ameliorate dextran sulfate sodium-induced colitis in rats. Fitoterapia. 2015 Mar;101:201-7.

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