Bakuchiol

Cat. No.: HY-N0235

CAS No.: 10309-37-2

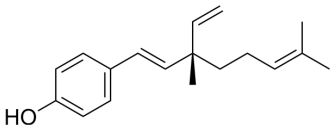
Molecular Formula: $C_{18}H_{24}O$

Molecular Weight: 256.38

Target: p38 MAPK; Autophagy; UGT; Carboxylesterase

Pathway: MAPK/ERK Pathway; Autophagy; Metabolic Enzyme/Protease

Storage: -20°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 62.5 mg/mL (243.78 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.9005 mL	19.5023 mL	39.0046 mL
	5 mM	0.7801 mL	3.9005 mL	7.8009 mL
	10 mM	0.3900 mL	1.9502 mL	3.9005 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.17 mg/mL (8.46 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.17 mg/mL (8.46 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Bakuchiol is a phytoestrogen that can be obtained from psoralen seeds. Bakuchiol has been proven to be a non-competitive inhibitor of multiple enzymes, including UDP-glucuronosyltransferase 2B7 (UGT2B7) [2] and human carboxylesterase 2 (hCE2) [3], with IC_{50}s values of 40.9 μM and 7.28 μM, respectively. Bakuchiol exhibits significant research and application potential in areas such as anti-inflammatory[5], antibacterial[4], antitumor[1] therapies, as well as drug metabolism regulation.

IC_{50} & Target

IC_{50}:40.9 nM (UGT2B7) ; IC_{50}:7.28 nM (hCE2)

In Vitro

Bakuchiol (5-20 μM, 72 h) exhibits selected cytotoxicity in A549 cells, as determined by the MTT assay. Bakuchiol displays stronger effect than its analogue Resveratrol (HY-16561) in reducing the viability of A549 cells with IC_{50}s vales of 9.58 μM and 33.02 μM, respectively[1].
Bakuchiol (5-20 μM, 24 h or 36h) blocks cell cycle progression at S phase and induction of Reactive Oxygen species (ROS) -

related apoptosis (more importantly) in A549 cells in a concentration-dependent manner[1].
Bakuchiol (0-80 μM, 120 min) is a non-competitive inhibitor of UDP-glucuronosyltransferase 2B7 (UGT2B7) with K_i value of 10.7 μM and IC_{50} value of 40.9 μM[2].
Bakuchiol (30 min) is a potent non-competitive inhibitor of human carboxylesterase 2 (hCE2) with low K_i value of 2.12 μM and IC_{50} value of 7.28 μM[3].
Bakuchiol (0.9775-3.91 μg/mL, 24 h) demonstrates a significant dose-dependent increase in membrane permeability of the fungal conidia of T. mentagrophytes[4].
Bakuchiol (3.91 μg/mL, 3 h) elicites a 187% elevation in Reactive Oxygen species (ROS) level in fungal cells[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	A549 cells, EA.hy926 cells, HUVEC cells and MEF
Concentration:	5, 10, and 20 μM
Incubation Time:	24 hours
Result:	Induced caspase 9/3 activaton, p53 and Bax up-regulation, as well as Bcl-2 down-regulation.

Apoptosis Analysis[1]

Cell Line:	A549 cells, EA.hy926 cells, HUVEC cells and MEF
Concentration:	5, 10, and 20 μM
Incubation Time:	36 hours
Result:	Resulted in typical apoptotic cells ($p < 0.05$), as determined by Annexin V/propidium iodide staining and flow cytometry.

Cell Cycle Analysis[1]

Cell Line:	A549 cells
Concentration:	5, 10, and 20 μM
Incubation Time:	24 hours
Result:	Increased S phase cell population accompanied with a concomitant reduction of cells in the G1 phase. Resulted in a less potent effect in S phase arrest than bakuchiol.

Cell Cytotoxicity Assay[1]

Cell Line:	A549 cells, EA.hy926 cells, HUVEC cells and MEF
Concentration:	5, 10, and 20 μM
Incubation Time:	72 hours
Result:	Inhibited the growth of A549 cells, while the growth rate of other cells were not changed. Exhibited concentration-dependent cytotoxicity in A549 cells, while bakuchiol showed a more potent effect than that of resveratrol with IC_{50}s vales of 9.58 μmol/L and 33.02 μmol/L, respectively.

In Vivo

Bakuchiol (10 and 20 mg/kg, p.o.) can attenuate allergic symptoms, decrease the inflammatory response, improve the T-cell balance, reduce oxidative stress and regulated Igs levels in ovalbumin (OVA) -induced allergic rhinitis (AR) mice[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	OVA -induced AR model (BALB/c mice, 6-week-old) [5]
Dosage:	10 and 20 mg/kg
Administration:	Oral route (p.o.)
Result:	Reduced the nasal symptoms and decreased the levels of IL-4, IL-5, IL-13, Igs (IgE and IgG1), histamine, IL-10, IL-33, and TNF-α. Reduced PGDA and LTC-4 levels in the nasal lavage fluid (NLF). Decreased the ROS and MDA levels, whereas boosting SOD activity. Decreased the eosinophil count in the nasal tissues and influenced the Th1 and Th2 cell proportions.

CUSTOMER VALIDATION

- J Ethnopharmacol. 2022 Aug 13;115593.

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REFERENCES

[1]. Yao HB, et al. Anti-Allergic and Anti-inflammatory Effects of Bakuchiol on Ovalbumin-Induced Allergic Rhinitis in Mice. Appl Biochem Biotechnol. 2024;196(6):3456-3470.

[2]. Chen Z, et al. Anti-tumor effects of bakuchiol, an analogue of resveratrol, on human lung adenocarcinoma A549 cell line. Eur J Pharmacol. 2010 Sep 25;643(2-3):170-9.

[3]. Xu Y, et al. In vitro evidence for bakuchiol's influence towards drug metabolism through inhibition of UDP-glucuronosyltransferase (UGT) 2B7. Afr Health Sci. 2014 Sep;14(3):564-9.

[4]. Li YG, et al. Fructus Psoraleae contains natural compounds with potent inhibitory effects towards human carboxylesterase 2. Fitoterapia. 2015 Jan 13;101C:99-106.

[5]. Lau KM, et al. Anti-dermatophytic activity of bakuchiol: in vitro mechanistic studies and in vivo tinea pedis-inhibiting activity in a guinea pig model. Phytomedicine. 2014 Jun 15;21(7):942-5.

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