Proteins

Product Data Sheet

Momordin Ic

Cat. No.: HY-N0330 CAS No.: 96990-18-0 Molecular Formula: $C_{41}H_{64}O_{13}$ 764.94 Molecular Weight:

Target: Apoptosis; Autophagy; PI3K; c-Myc Pathway: Apoptosis; Autophagy; PI3K/Akt/mTOR

4°C, protect from light Storage:

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

SOLVENT & SOLUBILITY

In Vitro

DMSO: 100 mg/mL (130.73 mM; Need ultrasonic)

| Preparing Stock Solutions | Solvent Mass Concentration | 1 mg | 5 mg | 10 mg |
|------------------------------|-------------------------------|-----------|-----------|------------|
| | 1 mM | 1.3073 mL | 6.5365 mL | 13.0729 mL |
| | 5 mM | 0.2615 mL | 1.3073 mL | 2.6146 mL |
| | 10 mM | 0.1307 mL | 0.6536 mL | 1.3073 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: ≥ 6.25 mg/mL (8.17 mM); Clear solution
- 2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.5 mg/mL (3.27 mM); Clear solution
- 3. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.5 mg/mL (3.27 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Momordin Ic is an orally active triterpenoid saponin that can be isolated from Kochia scoparia. It is also a SUMO specific protease 1 (SENP1) inhibitor, SENP1/c-MYC signaling pathway inhibitor, and apoptosis inducer. Momordin Ic induces autophagy and apoptosis in liver cancer cells through the PI3K/Akt and MAPK signaling pathways mediated by reactive oxygen species. Momordin Ic has the ability to control glucose induced blood glucose elevation, inhibit gastric emptying, resist rheumatoid arthritis, reduce CCl₄ (HY-Y0298) induced hepatotoxicity and anti-tumor activity^{[1][2]}.

In Vitro

Momordin Ic (0-20 μM, 48 h) relies on cholesterol and ganglioside GM1 to enhance the toxicity of recombinant protein MAP30 in breast cancer cells^[2].

 $Momordin \ Ic \ (10 \ \mu M, 24 \ h) \ induces \ colon \ cancer \ cell \ cycle \ arrest \ and \ apoptosis \ by \ inhibiting \ the \ SENP1/c-MYC \ signaling$

| pathway ^[3] . MCE has not independe | ntly confirmed the accuracy of these methods. They are for reference only. | |
|--|--|--|
| Cell Viability Assay ^{[1][2]} . | | |
| Cell Line: | HepG2 cell; MDA-MB-231, MCF-7, HepG2, H460, A549, HeLa cells | |
| Concentration: | 0-20 μΜ | |
| Incubation Time: | 4 h; 48 h | |
| Result: | Inhibited growth in HepG2 cells; Enhanced the cytotoxicity of MAP30 to breast cancer cells. | |
| Apoptosis Analysis ^[1] . | | |
| Cell Line: | HepG2 cell | |
| Concentration: | 0-15 μΜ | |
| Incubation Time: | 4 h | |
| Result: | Reduced the protein levels of caspase-3 and Bcl-2, and increased the protein levels of cytochrome c and Bax in the cytoplasmicsol. | |
| Western Blot Analysis ^[3] | | |
| Cell Line: | HCT-8 cell, HCT-116 cells | |
| Concentration: | 10 μΜ | |
| Incubation Time: | 24 h | |
| Result: | Reduced the level of c-Myc protein. | |

In Vivo

Momordin Ic (12.5, 25, 50 mg/kg, p.o.) accelerates gastrointestinal transport and inhibits gastric emptying in mice by stimulating the synthesis of serotonin (5-HT) $^{[4]}$.

 $Momordin\ lc\ (10\ mg/kg,\ p.o.)\ can\ inhibit\ ethanol\ induced\ gastric\ mucosal\ lesions\ in\ rats^{[5]}.$

 $Momordin\ Ic\ (30\ mg/kg, once\ a\ day\ for\ 14\ days, p.o.)\ can\ reduce\ CCl_4-induced\ hepatotoxicity\ in\ rats^{[6]}.$

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

| Animal Model: | Male ddY mice ^[4] . | |
|-----------------|--|--|
| Dosage: | 12.5, 25, 50 mg/kg | |
| Administration: | Oral gavage (p.o.) | |
| Result: | Accelerated gastrointestinal transit in fasted mice. | |
| | | |
| Animal Model: | Male Sprague-Dawley rat ^[5] . | |
| Dosage: | 10 mg/kg | |
| Administration: | Oral gavage (p.o.) | |
| Result: | Reduced the length of the lesions. | |

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| Animal Model: | Male Sprague–Dawley rat ^[6] . | |
|-----------------|---|--|
| Dosage: | 30 mg/kg | |
| Administration: | Oral gavage (p.o.), once a day for 14 days | |
| Result: | Reduced serum transaminase, lactic dehydrogenase, and γ-glutamyltransferase levels in the CCl ₄ -treated rats. | |

CUSTOMER VALIDATION

- J Pharmacol Sci. 2021 May 11.
- J Pharm Pharmacol. 2022 May 30;rgac033.

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REFERENCES

- [1]. Mi Y, et al. Momordin Ic couples apoptosis with autophagy in human hepatoblastoma cancer cells by reactive oxygen species (ROS)-mediated PI3K/Akt and MAPK signaling pathways. Free Radic Biol Med. 2016 Jan;90:230-42.
- [2]. Wang W, et al. Cytotoxic effects of recombinant proteins enhanced by momordin Ic are dependent on cholesterol and ganglioside GM1. Toxicon. 2023 Jun 15;229:107129.
- [3]. Xianjun F, et al. Momordin Ic induces G0/1 phase arrest and apoptosis in colon cancer cells by suppressing SENP1/c-MYC signaling pathway. J Pharmacol Sci. 2021 Aug;146(4):249-258.
- [4]. Li Y, et al. Acceleration of gastrointestinal transit by momordin Ic in mice: possible involvement of 5-hydroxytryptamine, 5-HT(2) receptors and prostaglandins. Eur J Pharmacol. 2000 Mar 24;392(1-2):71-7.
- [5]. Matsuda H, et al. Roles of capsaicin-sensitive sensory nerves, endogenous nitric oxide, sulfhydryls, and prostaglandins in gastroprotection by momordin Ic, an oleanolic acid oligoglycoside, on ethanol-induced gastric mucosal lesions in rats. Life Sci. 1999;65(2):PL27-32.
- [6]. Kim NY, et al. Momordin Ic and oleanolic acid from Kochiae Fructus reduce carbon tetrachloride-induced hepatotoxicity in rats. J Med Food. 2005 Summer;8(2):177-83.

Caution: Product has not been fully validated for medical applications. For research use only.

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