Sulforaphane

Cat. No.: HY-13755

CAS No.: 4478-93-7

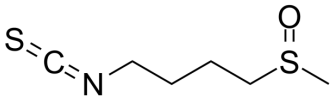
Molecular Formula: $C_6H_{11}NOS_2$

Molecular Weight: 177.29

Target: HDAC; Keap1-Nrf2; Apoptosis; Bcl-2 Family; Caspase; Reactive Oxygen Species

Pathway: Cell Cycle/DNA Damage; Epigenetics; NF-κB; Apoptosis; Immunology/Inflammation; Metabolic Enzyme/Protease

Storage: -20°C, sealed storage, away from moisture and light
* The compound is unstable in solutions, freshly prepared is recommended.



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 62.5 mg/mL (352.53 mM)

H_2O : 50 mg/mL (282.02 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	5.6405 mL	28.2024 mL	56.4048 mL
5 mM	1.1281 mL	5.6405 mL	11.2810 mL
10 mM	0.5640 mL	2.8202 mL	5.6405 mL

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

In Vivo

1. Add each solvent one by one: 30 % SBE-β-CD
   Solubility: 10 mg/mL (56.40 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (14.10 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (14.10 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (14.10 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Sulforaphane is an orally active inducer of the Keap1/Nrf2/ARE pathway. Sulforaphane promotes the transcription of tumor-suppressing proteins and effectively inhibits the activity of HDACs. Through the activation of the Keap1/Nrf2/ARE pathway and further induction of HO-1 expression, Sulforaphane protects the heart. Sulforaphane suppresses high glucose-induced pancreatic cancer through AMPK-dependent signal transmission. Sulforaphane exhibits both anticancer and anti-

	inflammatory properties[1][2][3][4][5][6].

IC_{50} & Target	HDAC	Bax	Caspase-3

In Vitro

Sulforaphane (0-30 μM) induces cell cycle arrest and apoptosis in a dose-dependent manner. Sulforaphane-induced cell cycle arrest is associated with an increase in the expression of cyclin A and B1[1].
Sulforaphane (0-30 μM) inhibits the re-initiation of growth and decreases cell viability in HT29 cells, exhibiting lower toxicity towards differentiated cells[1].
Sulforaphane (10 μM, 24 hours) pre-treatment reduces the number of apoptotic cells, decreases the expression of pro-apoptotic proteins (Bax, caspase-3, cytochrome c), and counteracts the increase in mitochondrial membrane potential induced by Doxorubicin (HY-15142A) (1 μM, 2 hours) in H9c2 cells[2].Sulforaphane (10 μM, 2 or 24 hours) effectively reduces ROS production and cell apoptosis in H9c2 cells induced by Doxorubicin (1 μM, 2 or 24 hours) through the activation of the Keap1/Nrf2/ARE pathway and further induction of HO-1 expression[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[2]

Cell Line:	H9c2 cells
Concentration:	Sulforaphane: 10 μM; Doxorubicin: 1 μM
Incubation Time:	Sulforaphane: 2h; Doxorubicin: 2, 24h
Result:	Prevented the release of cytochrome c into the cytosol. Prevented the translocation of Bax into the cytosol. Attenuated the doxorubicin-induced increase in the levels of cleaved caspase-3. Induced a significant increase in HO-1 protein expression. Induced a significantly higher level of Nrf2 expression in the nucleus compared to the cytoplasm.

Apoptosis Analysis[2]

Cell Line:	H9c2 cells
Concentration:	Sulforaphane: 10 μM; Doxorubicin: 1 μM
Incubation Time:	Sulforaphane: 2h; Doxorubicin: 24h
Result:	Protected the H9c2 cells against doxorubicin-induced cell death. Increased cell viability in a dose-dependent manner. Significantly reduced the number of apoptotic cells treated with Doxorubicin.

RT-PCR[2]

Cell Line:	H9c2 cells
Concentration:	Sulforaphane: 10 μM; Doxorubicin: 1 μM
Incubation Time:	Sulforaphane: 2, 24h; Doxorubicin: 2, 24h
Result:	Induced heme oxygenase-1 (HO-1) mRNA expression in a dose-dependent manner.

In Vivo

Sulforaphane (13.3, 17.7, 26.6 mg/kg; Oral gavage; 5 days) is capable of inhibiting the formation of mammary tumors in female Sprague-Dawley rats following a single-dose treatment with DMBA (HY-W011845) (8 mg/mL)[3].
Sulforaphane (13.3, 17.7, 26.6 mg/kg; Oral gavage; 5 days) can reduce the incidence, multiplicity, and weight of mammary tumors induced by DMBA (8 mg/mL) in female Sprague-Dawley rats, and delay their development[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Female Sprague-Dawley rat[3]
Dosage:	Sulforaphane: 13.3, 17.7, 26.6 mg/kg; DMBA (HY-W011845): 8 mg/mL
Administration:	Oral gavage; 5 days
Result:	Prevented the occurrence of tumors in a dose-dependent manner. Significantly reduced the incidence of tumors.

CUSTOMER VALIDATION

- Nat Commun. 2023 Sep 18;14(1):5778.
- Acta Pharm Sin B. 2021 May;11(5):1246-1260.
- Theranostics. 2020 Jun 5;10(16):7319-7334.
- Acta Pharmacol Sin. 2021 Jul 16.
- Phytomedicine. 2024 Feb 10:126:155441.

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REFERENCES

[1]. De Nicola GR, et al. Novel gram-scale production of enantiopure R-sulforaphane from Tuscan black kale seeds. Molecules. 2014 May 27;19(6):6975-86.

[2]. Abdull Razis AF, et al. The natural chemopreventive phytochemical R-sulforaphane is a far more potent inducer of the carcinogen-detoxifying enzyme systems in rat liver and lung than the S-isomer. Int J Cancer. 2011 Jun 15;128(12):2775-82.

[3]. Gamet-Payrastre L, et al. Sulforaphane, a naturally occurring isothiocyanate, induces cell cycle arrest and apoptosis in HT29 human colon cancer cells. Cancer Res. 2000 Mar 1;60(5):1426-33.

[4]. Li B, et al. Sulforaphane prevents doxorubicin-induced oxidative stress and cell death in rat H9c2 cells. Int J Mol Med. 2015 Jul;36(1):53-64.

[5]. Zhang Y, et al. Anticarcinogenic activities of sulforaphane and structurally related synthetic norbornylisothiocyanates. Proc Natl Acad Sci U S A. 1994 Apr 12;91(8):3147-50.

[6]. Chen X, et al. Activation of Nrf2 by Sulforaphane Inhibits High Glucose-Induced Progression of PancreaticCancer via AMPK Dependent Signaling. ell Physiol Biochem. 2018;50(3):1201-1215.

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