

Product Data Sheet

Product Name: Sulforaphane

Cat. No.: GC14016

Chemical Properties

Cas. No. 4478-93-7

Chemical Name 1-isothiocyanato-4-(methylsulfinyl)-butane

SMILES CS(CCCCN=C=S)=O

Formula $C_6H_{11}NOS_2$ M.Wt 177.3

Solubility $\geq 67.6\text{mg/mL}$ in DMSO Storage Store at -20°C , sealed storage, away from moisture and light

General tips For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice All other available size: ship with RT, or blue ice upon request.

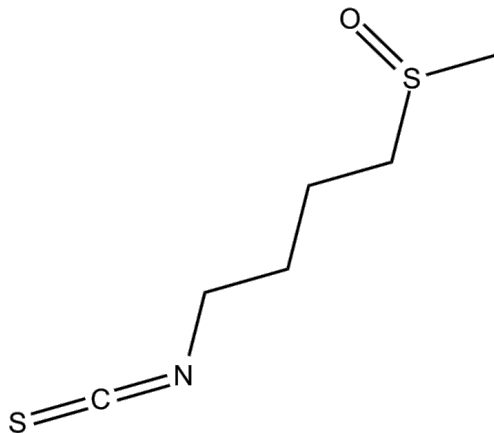
Caution: Product has not been fully validated for medical applications. For research use only.

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Structure



Protocol

Cell experiment [1]:

Cell lines HepaRG cells

HepaRG cells at a concentration of 1.5×10^4 cells/mL were seeded on uncoated plastic tissue culture dishes and treated with sulforaphane at different concentrations (0 -40 μ M) over a time course (12-48 h). The cells as well as the homogenates from liver tissues were solubilized, and the activity solution including a reporter dye were added to the supernatants.

Reaction Conditions 0 -40 μ M sulforaphane for 12-48 h

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Applications Sulforaphane amplifies ALDH2 activity in HepaRG cells and suppresses acetaldehyde-induced proliferation and activation in LX-2 cells.

Animal experiment [2]:

Animal models Ten-week-old female C57BL/6J mice

Preparation Method Mice in the CD/ CCl₄ group were fed the normal liquid diet and received 1 mL/kg body weight CCl₄ dissolved in corn oil by intraperitoneal injection twice weekly. Mice in the ED/ CCl₄ group were fed a 2.5% (v/v) ethanol-containing Lieber-DeCarli liquid diet (Research Diets) (ED) and received intraperitoneal CCl₄ injection twice weekly (1 mL/kg body weight). Mice in the ED/ CCl₄/SFN group were fed the ED with sulforaphane and received intraperitoneal CCl₄ injection (1 mL/kg body weight) twice weekly; in this group, sulforaphane was administered peroral as a mixture of ED (5 µmol/d/body weight).

Dosage form Sulforaphane was administered peroral as a mixture of ED (5 µmol/d/body weight) twice weekly

Applications Sulforaphane exerts antioxidative effects with ALDH2 induction in liver fibrosis induced by ethanol exposure in CCl₄-treated mice.

References:

[1]. Ishida K, Kaji K, et.al. Sulforaphane ameliorates ethanol plus carbon tetrachloride-induced liver fibrosis in mice through the Nrf2-mediated antioxidant response and acetaldehyde metabolism with inhibition of the LPS/TLR4 signaling pathway. J Nutr Biochem. 2021 Mar;89:108573. doi: 10.1016/j.jnutbio.2020.108573. Epub 2020 Dec 31. PMID: 33388347.

Background

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Sulforaphane is an isothiocyanate that is present naturally in cruciferous vegetables with anti-oxidant, anti-inflammatory, and anti-bacterial effects^[1]. Sulforaphane can function by blocking initiation via inhibiting Phase 1 enzymes that convert procarcinogens to proximate or ultimate carcinogens, and by inducing Phase 2 enzymes that detoxify carcinogens and facilitate their excretion from the body^[2]. Sulforaphane has been widely used to inhibit the proliferation of various cancer cells and to improve oxidative stress^[3].

In vitro, Sulforaphane treatment for 24 hours significantly inhibited the viability of OECM-1 cells, with an IC₅₀ value of 5.7 μ M^[4]. Treatment with 10 μ M Sulforaphane for 48 hours significantly induced apoptosis in MDAH-2774 cells, accompanied by an increase in the expression of pro-apoptotic protein BAK, while the expression of anti-apoptotic protein Bcl-2 decreased^[5]. Treatment with 20 μ M Sulforaphane for 24 hours significantly reduced the expression of EGFR and EGFR signaling in A549 cells, and decreased cell viability^[6].

In vivo, Sulforaphane treatment via oral administration at a dose of 20mg/kg/day for 2 weeks alleviated dextran sulfate sodium (DSS)-induced ulcerative colitis and reversed gut dysbiosis in mice^[7]. Oral administration of Sulforaphane at a dose of 1g/kg/day for 6 weeks alleviated high-fat diet-induced visceral fat accumulation, adipocyte hypertrophy, and hepatic steatosis in mice^[8].

References:

- [1] Fimognari C, Hrelia P. Sulforaphane as a promising molecule for fighting cancer[J]. Mutation Research/Reviews in Mutation Research, 2007, 635(2-3): 90-104.
- [2] Clarke J D, Dashwood R H, Ho E. Multi-targeted prevention of cancer by sulforaphane[J]. Cancer letters, 2008, 269(2): 291-304.
- [3] Guerrero-Beltrán C E, Calderón-Oliver M, Pedraza-Chaverri J, et al. Protective effect of sulforaphane against oxidative stress: recent advances[J]. Experimental and Toxicologic Pathology, 2012, 64(5): 503-508.
- [4] Adtani P N, Al-Bayati S A A F, Elsayed W S. Sulforaphane from brassica oleracea induces apoptosis in oral squamous carcinoma cells via p53 activation and mitochondrial membrane potential dysfunction[J]. Pharmaceuticals, 2025, 18(3): 393.
- [5] Bryant C S, Kumar S, Chamala S, et al. Sulforaphane induces cell cycle arrest by protecting RB-E2F-1 complex in epithelial ovarian cancer cells[J]. Molecular cancer, 2010, 9(1): 47.
- [6] Chen C Y, Yu Z Y, Chuang Y S, et al. Sulforaphane attenuates EGFR signaling in NSCLC cells[J].

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Journal of biomedical science, 2015, 22(1): 38.

[7] Zhang Y, Tan L, Li C, et al. Sulforaphane alter the microbiota and mitigate colitis severity on mice ulcerative colitis induced by DSS[J]. Amb Express, 2020, 10(1): 119.

[8] Choi K M, Lee Y S, Kim W, et al. Sulforaphane attenuates obesity by inhibiting adipogenesis and activating the AMPK pathway in obese mice[J]. The Journal of nutritional biochemistry, 2014, 25(2): 201-207.

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