S-Allylmercaptocysteine

Cat. No.: HY-145532

CAS No.: 2281-22-3

Molecular Formula: $C_6H_{11}NO_2S_2$

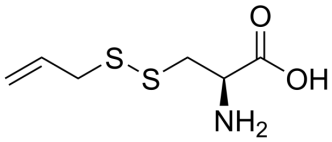
Molecular Weight: 193.29

Target: Apoptosis; NF-κB; Keap1-Nrf2

Pathway: Apoptosis; NF-κB

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

H_2O : 1 mg/mL (5.17 mM; ultrasonic and warming and heat to 60°C)

DMSO : < 1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble or slightly soluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	5.1736 mL	25.8679 mL	51.7357 mL
5 mM	1.0347 mL	5.1736 mL	10.3471 mL
10 mM	---	---	---

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

BIOLOGICAL ACTIVITY

Description

S-allylmercaptocysteine, an organic sulfur compound extracted from garlic, has anti-inflammatory and anti-oxidative effects for various pulmonary diseases. S-allylmercaptocysteine achieves its anti-cancer effect through a variety of pathways such as inducing the apoptosis of cancer cells through the TGF-β signaling pathway, or reducing the NF-κB activity and up-regulating Nrf2 to achieve the effects of anti-inflammation and anti-oxidation[1][2][3].

In Vitro

S-Allylmercaptocysteine attenuates cisplatin-induced nephrotoxicity through suppression of apoptosis, oxidative stress, and inflammation[2].

S-Allylmercaptocysteine (400 μM; 48 hours) induces apoptosis evaluated by detecting the activated caspase 3 and cleaved PARP in SW620, SW480, and Caco-2 cells. Both activated caspase 3 and cleaved PARP1 are found in the cells treated with SAMC while no activated PARP1 and caspase 3 are found in the untreated control cells[4].

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

In Vivo

S-Allylmercaptocysteine (25 and 50 mg/kg; oral gavage) could significantly ameliorate the pathological structure, and decrease inflammatory cell infiltration and pro-inflammatory cytokines in bronchoalveolar lavage fluid (BALF) in BLM-

induced pulmonary fibrosis mice. S-Allylmercaptocysteine shows an anti-fibrosis effect by increasing anti-oxidants like HO-1, GSH and SOD as well as decreasing hydroxyproline (HYP) in BLM-induced mice[1].

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

REFERENCES

[1]. Zhu X, et al. S-Allylmercaptocysteine Attenuates Cisplatin-Induced Nephrotoxicity through Suppression of Apoptosis, Oxidative Stress, and Inflammation. Nutrients. 2017;9(2):166. Published 2017 Feb 20.

[2]. Tong D, et al. S-allylmercaptocysteine promotes MAPK inhibitor-induced apoptosis by activating the TGF-β signaling pathway in cancer cells. Oncol Rep. 2014;32(3):1124-1132.

[3]. Li C, et al. S-Allylmercaptocysteine attenuates Bleomycin-induced pulmonary fibrosis in mice via suppressing TGF-β1/Smad and oxidative stress pathways. Int Immunopharmacol. 2020;79:106110.

[4]. Liang D, et al. S-allylmercaptocysteine effectively inhibits the proliferation of colorectal cancer cells under in vitro and in vivo conditions. Cancer Lett. 2011;310(1):69-76.

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