S-Allyl-L-cysteine

Cat. No.: HY-W013573

CAS No.: 21593-77-1

Molecular Formula: $C_6H_{11}NO_2S$

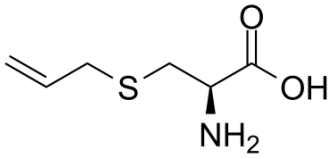
Molecular Weight: 161.22

Target: Apoptosis

Pathway: Apoptosis

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 : 33.33 mg/mL (206.74 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	6.2027 mL	31.0135 mL	62.0270 mL
	5 mM	1.2405 mL	6.2027 mL	12.4054 mL
	10 mM	0.6203 mL	3.1014 mL	6.2027 mL

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

BIOLOGICAL ACTIVITY

Description

S-Allyl-L-cysteine, one of the organosulfur compounds found in AGE, possess various biological effects including neurotrophic activity, anti-cancer activity, anti-inflammatory activity.

In Vitro

It is found that S-Allyl-L-cysteine could protect against amyloid-protein (A)-and tunicamycin-induced cell death in differentiated PC12 cells. Simultaneously applied S-Allyl-L-cysteine (1 μM) suppresses the cell death induced by $A\beta_{25-35}$ and $A\beta_{1-40}$ in a concentration-dependent manner, and neuronal integrity is almost completely retained. Simultaneously applied S-Allyl-L-cysteine significantly decreases the Aβ-induced level of ROS. The TEAC value of S-Allyl-L-cysteine is lower than that of oxidized GSH, and no antioxidant activity is observed. Intracellular GSH levels remains unaffected by treatment of neurons with S-Allyl-L-cysteine for 24 h. Furthermore, the increase in caspase-12 protein expression is suppressed by simultaneously adding 1 μM S-Allyl-L-cysteine [1]. S-Allyl-L-cysteine up to a concentration 1.0 mM does not exhibit any cytotoxic impact on morphology of myoblast and myotubes in culture observed under bright field microscope. TNF treatment leads to a significant decrease in the intracellular CK activity while S-Allyl-L-cysteine pre-treatment to TNF treated myotubes decreases the release of CK in media. S-Allyl-L-cysteine pre-treatment decreases the level of active form of this enzyme in S-Allyl-L-cysteine+TNF group. Similar observations are recorded at mRNA level for caspase-3. These results illustrate that S-Allyl-L-cysteine regulates apoptotic signals via suppressing the transcription and thus protein expression of

caspase-3[2].

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

REFERENCES

[1]. Kosuge Y, et al. S-allyl-L-cysteine selectively protects cultured rat hippocampal neurons from amyloid beta-protein- and tunicamycin-induced neuronal death. Neuroscience. 2003;122(4):885-95.

[2]. Dutt V, et al. S-allyl cysteine inhibits TNFα-induced skeletal muscle wasting through suppressing proteolysis and expression of inflammatory molecules. Biochim Biophys Acta. 2018 Apr;1862(4):895-906.

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