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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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S-Allyl-L-cysteine

Cat. No.: HY-W013573

CAS No.: 21593-77-1

Molecular Formula: $C_6H_{11}NO_2S$

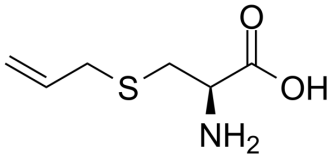
Molecular Weight: 161.22

Target: Apoptosis

Pathway: Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

H_2O : 16.67 mg/mL (103.40 mM; ultrasonic and warming and heat to 60°C)

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.

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 50 mg/mL (310.14 mM); Clear solution; Need ultrasonic

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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