Se-Methylselenocysteine

Cat. No.: HY-114245

CAS No.: 26046-90-2

Molecular Formula: $C_4H_9NO_2Se$

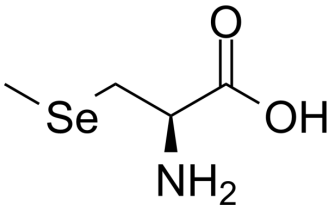
Molecular Weight: 182.08

Target: Apoptosis; Endogenous Metabolite; Beta-secretase

Pathway: Apoptosis; Metabolic Enzyme/Protease; Neuronal Signaling

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 : 83.33 mg/mL (457.66 mM; Need ultrasonic)

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	5.4921 mL	27.4605 mL	54.9209 mL
5 mM	1.0984 mL	5.4921 mL	10.9842 mL
10 mM	0.5492 mL	2.7460 mL	5.4921 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 (274.60 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Se-Methylselenocysteine, a precursor of Methylselenol, has potent cancer chemopreventive activity and anti-oxidant activity. Se-Methylselenocysteine is orally bioavailable, and induces apoptosis[1][2].

IC_{50} & Target

BACE1

In Vitro

Se-Methylselenocysteine (100-400 μM; 3 days) induces apoptosis in SKOV-33 cells[1].

?Se-Methylselenocysteine (100-400 μM; 3 days) induces caspase-3 mediated apoptosis[1].

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

Apoptosis Analysis[1]

Cell Line:	SKOV-3 cells
Concentration:	100, 200, 400 μM
Incubation Time:	3 days
Result:	Resulted in a markedly increased accumulation of Sub-G1 phase, which occurred in both SeMSC concentration and culture time-dependent.

Western Blot Analysis[1]

Cell Line:	SKOV-3 cells
Concentration:	100, 200, 400 μM
Incubation Time:	3 days
Result:	Resulted in a decrease in the expression of the 32 kDa form of procaspase-3.

In Vivo

Se-Methylselenocysteine (0.2?mg/mouse; p.o.; daily for 14 days) potentiates the antitumour activity of CDDP and Cyclophosphamide in nude mice bearing human FaDu and A253 head and neck xenografts[2].
?Alzheimer's disease (AD) mice are treats with Se-Methylselenocysteine (0.75 mg/kg BW per day) in their drinking water for 10 months. Se-Methylselenocysteine reduces oxidative stress and neuro-inflammation; Se-Methylselenocysteine modulates the distribution and levels of several metal ions; Se-Methylselenocysteine decreases amyloid-β peptide (Aβ) generation by inhibiting the expression of its precursor protein APP and β-secretase (BACE1), and attenuates tau hyperphosphorylation and neurofibrillary tangles (NFT) formation via promoting protein phosphatase 2A (PP2A) activity, thereby preserving synaptic proteins and neuron activities and finally improving spatial learning and memory deficits in AD model mice[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Female athymic nude mice (bearing human A253 and FaDu squamous cell carcinoma xenografts)[2]
Dosage:	0.2 mg/mouse
Administration:	p.o.; daily for 14 days (7 days before and 7 days after Cyclophosphamide or CDDP in a total of 14 days)
Result:	

CUSTOMER VALIDATION

- Redox Biol. 2024 Apr, 70, 103024.

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REFERENCES

[1]. Yeo JK, et al. Se-methylselenocysteine induces apoptosis through caspase activation and Bax cleavage mediated by calpain in SKOV-3 ovarian cancer cells. Cancer Lett. 2002 Aug 8;182(1):83-92.

[2]. Cao S, et al. Se-methylselenocysteine offers selective protection against toxicity and potentiates the antitumour activity of anticancer drugs in preclinical animal models. Br J Cancer. 2014 Apr 2;110(7):1733-43.

[3]. Xie Y, et al. Se-Methylselenocysteine Ameliorates Neuropathology and Cognitive Deficits by Attenuating Oxidative Stress and Metal Dyshomeostasis in Alzheimer Model Mice. Mol Nutr Food Res. 2018 Jun;62(12):e1800107.

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