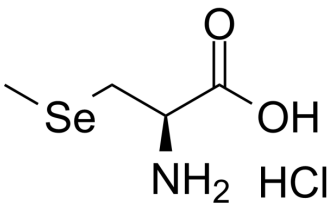
# Se-Methylselenocysteine hydrochloride

| | |
|---|---|
| **Cat. No.:** | HY-114245B |
| **CAS No.:** | 863394-07-4 |
| **Molecular Formula:** | $C_4H_{10}ClNO_2Se$ |
| **Molecular Weight:** | 218.54 |
| **Target:** | Endogenous Metabolite; Apoptosis; Beta-secretase |
| **Pathway:** | Metabolic Enzyme/Protease; Apoptosis; Neuronal Signaling |
| **Storage:** | Please store the product under the recommended conditions in the Certificate of Analysis. |




## BIOLOGICAL ACTIVITY

| | |
|---|---|
| **Description** | Se-Methylselenocysteine hydrochloride, a precursor of Methylselenol, has potent cancer chemopreventive activity and anti-oxidant activity. Se-Methylselenocysteine hydrochloride is orally bioavailable, and induces apoptosis[1][2]. |
| **In Vitro** | Se-Methylselenocysteine hydrochloride (100-400 μM; 3 days) induces apoptosis in SKOV-33 cells[1].<br>?Se-Methylselenocysteine hydrochloride (100-400 μM; 3 days) induces caspase-3 mediated apoptosis[1].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |
| **In Vivo** | Se-Methylselenocysteine hydrochloride (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].<br>?Alzheimer's disease (AD) mice are treats with Se-Methylselenocysteine hydrochloride (0.75 mg/kg BW per day) in their drinking water for 10 months. Se-Methylselenocysteine hydrochloride reduces oxidative stress and neuro-inflammation; Se-Methylselenocysteine hydrochloride modulates the distribution and levels of several metal ions; Se-Methylselenocysteine hydrochloride 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].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |

## CUSTOMER VALIDATION

- Redox Biol. 2024 Apr, 70, 103024.
- Cell Mol Life Sci. 2024 Jan 22;81(1):49.

See more customer validations on www.MedChemExpress.com

## 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.

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

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA