

Product Data Sheet

Product Name: L-Selenocystine

Cat. No.: GC44084

Chemical Properties

Cas. No. 29621-88-3

SMILES OC([C@@H](N)C[Se][Se]C[C@H](N)C(O)=O)=O

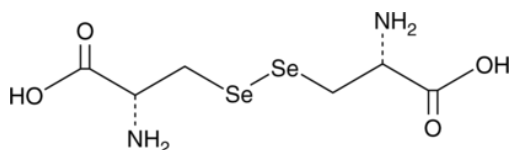
Formula $C_6H_{12}N_2O_4Se_2$ M.Wt 334.1

Solubility H_2O : 5.88 mg/mL (17.60 mM); Storage Store at $-20^{\circ}C$
ultrasonic and warming and adjust
pH to 1 with HCl and heat to $60^{\circ}C$

General For obtaining a higher solubility , please warm the tube at $37^{\circ}C$ and shake it in the
tips ultrasonic bath for a while. Stock solution can be stored below $-20^{\circ}C$ for several months.

Shipping Evaluation sample solution : ship with blue ice All other available size: ship with RT , or
Condition blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines HepG2 cells

HepG2 cells were cultured in DMEM medium supplemented with 10% fetal bovine serum (FBS) and antibiotics (penicillin 100U/ml, streptomycin 0.1mg/ml) under normal conditions (5% CO_2 with 95% humidified air). HepG2 cells (6×10^4 per well) were inoculated in 96-well cell culture plate and incubated at $37^{\circ}C$ with 5% CO_2 for 24h. Then, the cells were treated with 2 μ M, 4 μ M, 8 μ M, 16 μ M, and 32 μ M L-Selenocystine for 24h, respectively, and cell viability was measured.

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

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Reaction Conditions 2, 4, 8, 16, and 32μM; 24h

Applications L-Selenocystine treatment reduced the cell viability of HepG2 cells in a dose-dependent manner.

Animal experiment [2]:

Animal models Female nude mice

Preparation Method Thirty female nude mice were divided into three groups. After adaptation for 1-week, JEG-3 cells (10^7 cells in 100μl serum-free medium) were subcutaneously injected into the left outer of mice. After growing for 7 days (tumor volume was about 60mm³), the mice were administrated with L-Selenocystine. Group 1 was set as the control group without treatment and Group 2 was administrated with 10mg/kg of L-Selenocystine. L-Selenocystine was injected from the caudal vein every other day for 14 days (a total of eight times). After administration was completed, tumors were harvested and measured with the following formula: volume = $L \times w^2 / 2$, thereinto L was the maximal length and w was the width.

Dosage form 10mg/kg/day for 14 days; vein injection

Applications L-Selenocystine treatment significantly inhibited JEG-3 choriocarcinoma tumor xenograft growth in mice.

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References:

- [1] Chen H, Su J, Chen D, et al. L-Selenocystine induce HepG2 cells apoptosis through ROS-mediated signaling pathways[J]. Biocell, 2022, 46(10): 2267.
- [2] Zhao M, Hou Y, Fu X, et al. Selenocystine inhibits JEG-3 cell growth in vitro and in vivo by triggering oxidative damage-mediated S-phase arrest and apoptosis[J]. Journal of Cancer Research and Therapeutics, 2018, 14(7): 1540-1548.

Background

L-Selenocystine is an oxidation diselenide product of selenocysteine with strong antioxidant activity [1]. L-Selenocystine can promote the phosphorylation of JNK, p38 MAPK, ERK and Akt in cells, and induce the activation of caspase, PARP cleavage and DNA fragmentation [2]. L-Selenocystine has been widely used to induce apoptosis in various cancer cells and to generate reactive oxygen species (ROS)[3].

In vitro, L-Selenocystine treatment for 24 hours significantly inhibited the proliferation of HepG2 cells and L02 cells, with IC₅₀ values of 71.880μM and 5.444μM, respectively [4]. Treatment with 50μM L-Selenocystine for 24 hours significantly inhibited the activation of Nrf2 and the nuclear translocation of downstream proteins in WiDr cells, and induced autophagy in the cells [5]. The combined treatment of 10μM L-Selenocystine and 5-fluorouracil (400μM) for 24 hours triggered mitochondrial-mediated apoptosis in A375 cells, inducing mitochondrial dysfunction and enhancing DNA damage [6].

In vivo, L-Selenocystine treatment via vein injection at a dose of 10mg/kg/day for 14 days effectively suppressed JEG-3 choriocarcinoma tumor xenograft growth in mice [7]. Every other day, 5mg/kg dose of L-Selenocystine and 2mg/kg dose of auranofin were injected via the tail vein for 16 days, which inhibited the tumor volume and weight of A549 human lung adenocarcinoma xenografts in nude mice, without affecting the body weight of the mice [8].

References:

- [1] Chuai H, Zhang S Q, Bai H, et al. Small molecule selenium-containing compounds: Recent development and therapeutic applications[J]. European journal of medicinal chemistry, 2021, 223: 113621.

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- [2] Chen T, Wong Y S. Selenocystine induces S-phase arrest and apoptosis in human breast adenocarcinoma MCF-7 cells by modulating ERK and Akt phosphorylation[J]. Journal of agricultural and food chemistry, 2008, 56(22): 10574-10581.
- [3] Chen T, Wong Y S. Selenocystine induces reactive oxygen species-mediated apoptosis in human cancer cells[J]. Biomedicine & Pharmacotherapy, 2009, 63(2): 105-113.
- [4] Chen H, Su J, Chen D, et al. L-Selenocystine induce HepG2 cells apoptosis through ROS-mediated signaling pathways[J]. Biocell, 2022, 46(10): 2267.
- [5] Hsu W L, Wang C M, Yao C L, et al. Blockage of Nrf2 and autophagy by L-selenocystine induces selective death in Nrf2-addicted colorectal cancer cells through p62-Keap-1-Nrf2 axis[J]. Cell Death & Disease, 2022, 13(12): 1060.
- [6] Fan C, Chen J, Wang Y, et al. Selenocystine potentiates cancer cell apoptosis induced by 5-fluorouracil by triggering reactive oxygen species-mediated DNA damage and inactivation of the ERK pathway[J]. Free Radical Biology and Medicine, 2013, 65: 305-316.
- [7] Zhao M, Hou Y, Fu X, et al. Selenocystine inhibits JEG-3 cell growth in vitro and in vivo by triggering oxidative damage-mediated S-phase arrest and apoptosis[J]. Journal of Cancer Research and Therapeutics, 2018, 14(7): 1540-1548.
- [8] Fan C, Zheng W, Fu X, et al. Enhancement of auranofin-induced lung cancer cell apoptosis by selenocystine, a natural inhibitor of TrxR1 in vitro and in vivo[J]. Cell death & disease, 2014, 5(4): e1191-e1191.

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