L-Buthionine-(S,R)-sulfoximine

Cat. No.: HY-106376A

CAS No.: 83730-53-4

Molecular Formula: $C_8H_{18}N_2O_3S$

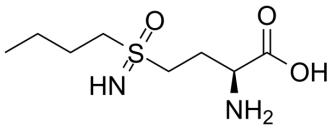
Molecular Weight: 222.31

Target: Ferroptosis

Pathway: Apoptosis

Storage:

Powder	-20°C	3 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

H_2O : 50 mg/mL (224.91 mM; Need ultrasonic)

DMSO : < 1 mg/mL (ultrasonic) (insoluble or slightly soluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.4982 mL	22.4911 mL	44.9822 mL
5 mM	0.8996 mL	4.4982 mL	8.9964 mL
10 mM	0.4498 mL	2.2491 mL	4.4982 mL

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

BIOLOGICAL ACTIVITY

Description

L-Buthionine-(S,R)-sulfoximine is a cell-permeable, potent, fast acting and irreversible inhibitor of g-glutamylcysteine synthetase and depletes cellular glutathione levels. The IC_{50} value of L-Buthionine-(S,R)-sulfoximine on melanoma, breast and ovarian tumor specimens are 1.9 μM, 8.6 μM, and 29 μM, respectively.

IC_{50} & Target

γ-glutamylcysteine synthetase[1].

In Vitro

L-Buthionine-(S,R)-sulfoximine (BSO: 50 μM) treatment for 48 hr results in a 95% decrease in ZAZ and M14 melanoma cell line GSH levels, and a 60% decrease in GST enzyme activity. GST-π protein and mRNA levels are significantly reduced in both cell lines[1]. L-Buthionine-(S,R)-sulfoximine (BSO) induces oxidative stress in a cell by irreversibly inhibiting g-glutamylcysteine synthetase, an essential enzyme for the synthesis of glutathione (GSH)[2].

L-Buthionine-(S,R)-sulfoximine (BSO) induces ferroptosis in cancer cells[3].

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

In Vivo

BSO causes an elevated frequency of DNA deletions during mouse development. BSO treatment reduced GSH concentration in mouse fetuses by 55% and 70% at 2 mM and 20 mM BSO doses, respectively, compared to untreated mice. Co-treatment

with 2 mM BSO and 20 mM NAC depleted GSH to a similar extent as 2 mM BSO, consistent with the function of BSO to inhibit the g-GCS enzyme indispensable for GSH synthesis. Like GSH, cysteine levels dropped following BSO treatment[2].

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

CUSTOMER VALIDATION

- Acta Pharm Sin B. 21 October 2021.

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REFERENCES

[1]. Fruehauf JP, et al. Selective and synergistic activity of L-S,R-buthionine sulfoximine on malignant melanoma is accompanied by decreased expression of glutathione-S-transferase. Pigment Cell Res. 1997 Aug;10(4):236-49.

[2]. Reliene R, et al. Glutathione depletion by buthionine sulfoximine induces DNA deletions in mice. Carcinogenesis. 2006 Feb;27(2):240-4.

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