L-Buthionine-(S,R)-sulfoximine

Cat. No.: HY-106376A

CAS No.: 83730-53-4

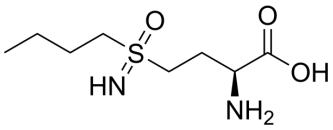
Molecular Formula: $C_8H_{18}N_2O_3S$

Molecular Weight: 222.31

Target: Ferroptosis

Pathway: Apoptosis

Storage: -20°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 18.33 mg/mL (82.45 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.

In Vivo

1. Add each solvent one by one: PBS
Solubility: 100 mg/mL (449.82 mM); Clear solution; Need ultrasonic

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.

Animal Model:	C57BL/6J pun/pun mice[2].
Dosage:	2 mM L-Buthionine-(S,R)-sulfoximine (BSO), 20 mM BSO, 2 mM BSO and 20 mM NAC, 20 mM NAC or unsupplemented water for 18 days from 0.5 to 18.5 d.p.c. The pH of supplemented water is as follows: 6.88, 20 mM BSO; 3.37, 2 mM BSO; 2.65, 2 mM BSO plus 20 mM NAC; and 2.58, 20 mM NAC. The pH of regular water used in our facility is ~4.
Administration:	Drinking water.
Result:	The average number of eye-spots (mean±SEM) is 5.36±0.29 (n=46), 7.79±0.45 (n=34) and 8.78±0.61 (n=32) in untreated controls, 2 mM L-Buthionine-(S,R)-sulfoximine (BSO) and 20 mM BSO treated mice, respectively. The 2 mM BSO treatment results in ~30% more eye-spots, and the 20 mM treatment results in 40% more eye-spots compared with untreated mice.

CUSTOMER VALIDATION

- Nat Commun. 2022 Jul 11;13(1):4007.
- Acta Pharm Sin B. 21 October 2021.
- J Colloid Interface Sci. 11 August 2022.
- J Obstet Gynaecol Res. 2023 Jun 20.
- Oxid Med Cell Longev. 2022 Jan 17;2022:8038857.

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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.

[3]. Satoru Nishizawa, et al. Low tumor glutathione level as a sensitivity marker for glutamate-cysteine ligase inhibitors. Oncol Lett. 2018 Jun;15(6):8735-8743.

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