Sulfosuccinimidyl oleate sodium

Cat. No.: HY-112847A

CAS No.: 1212012-37-7

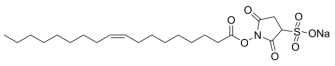
Molecular Formula: $C_{22}H_{36}NNaO_7S$

Molecular Weight: 481.58

Target: Mitophagy

Pathway: Autophagy

Storage: 4°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

DMSO : 62.5 mg/mL (129.78 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0765 mL	10.3825 mL	20.7650 mL
	5 mM	0.4153 mL	2.0765 mL	4.1530 mL
	10 mM	0.2076 mL	1.0382 mL	2.0765 mL

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

In Vivo

1. Add each solvent one by one: 0.5% Methylcellulose/saline water
   Solubility: 3.33 mg/mL (6.91 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 3.33 mg/mL (6.91 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (4.32 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.32 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Sulfosuccinimidyl oleate sodium (Sulfo-N-succinimidyl oleate sodium) is a long chain fatty acid that inhibits fatty acid transport into cells. Sulfosuccinimidyl oleate sodium is a potent and irreversible inhibitor of mitochondrial respiratory chain . Sulfosuccinimidyl oleate sodium binds the CD36 receptor on the surface of microglia. Anti-inflammatory effect[1][2].

In Vitro

Sulfosuccinimidyl oleate (20 μM and 50 μM, 24 hours) alone does not alter the cellular viability. Exposure to 100 ng/ml LPS+5 ng/mL IFNγ modestly, yet significantly reduces the viability of the BV2 cells. Co-treatment with Sulfosuccinimidyl oleate prevents the LPS+IFNγ-induced reduction in the cell viability[1].

Sulfosuccinimidyl oleate (50 μM, 24 hours) co-treatment significantly reduces the LPS+IFNγ-induced expression of NOS2 and COX-2 in BV2 cells. Western blot analysis reveals a significant LPS/IFNγ-induced upregulation in the phosphorylated form of the p38, which is prevented by co-treatment with Sulfosuccinimidyl oleate (50 μM, 24 hours)[1].

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

Cell Viability Assay[1]

Cell Line:	BV2 cells
Concentration:	20 μM and 50 μM
Incubation Time:	24 hours
Result:	Did not alter the viability of BV2 cells alone. Exposure of BV2 cells to 100 ng/mL LPS and 5 ng/mL IFNγ significantly reduced the viability of BV2 cells while simultaneous treatment with Sulfosuccinimidyl oleate prevented it.

Western Blot Analysis[1]

Cell Line:	BV2 cells
Concentration:	50 μM
Incubation Time:	24 hours
Result:	Drastically increased the levels of NOS2, COX-2, and P-p38/T-p38.

In Vivo

the Sulfosuccinimidyl oleate (50 mg/kg; administered once by single oral gavage) significantly reduces the cortical ischemic infarct size compared to vehicle-treated controls in male BALB/cABom mice with pMCAo model. In addition, Sulfosuccinimidyl oleate at 50 mg/kg is suitable to see a beneficial effect after stroke[1].

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

Animal Model:	4-month-old male BALB/cABom mice with pMCAo model[1]
Dosage:	50 mg/kg
Administration:	Administered once by single oral gavage
Result:	Reduced brain damage following ischemia. Attenuated infarct size.

CUSTOMER VALIDATION

- J Exp Med. 2023 Mar 6;220(3):e20221316.
- J Transl Med. 2023 Feb 6;21(1):89.
- Front Pharmacol. 2020 Dec 16;11:593832.
- CNS Neurosci Ther. 2023 May 8.
- Cell Immunol. 11 January 2022, 104475.

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REFERENCES

[1]. Dhungana H, et al. Sulfosuccinimidyl oleate sodium is neuroprotective and alleviates stroke-induced neuroinflammation. J Neuroinflammation. 2017 Dec 4;14(1):237.

[2]. Drahota Z, et al. Succinimidyl oleate, established inhibitor of CD36/FAT translocase inhibits complex III of mitochondrial respiratory chain. Biochem Biophys Res Commun. 2010 Jan 15;391(3):1348-51.

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