Sulfasalazine

Cat. No.: HY-14655

CAS No.: 599-79-1

Molecular Formula: $C_{18}H_{14}N_4O_5S$

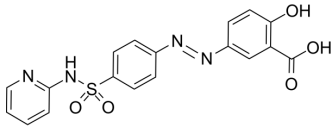
Molecular Weight: 398.39

Target: NF-κB; Autophagy; Apoptosis; Ferroptosis; Bacterial; Antibiotic

Pathway: NF-κB; Autophagy; Apoptosis; Anti-infection

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	1 year
	-20°C	6 months



SOLVENT & SOLUBILITY

In Vitro

NH4OH : 150 mg/mL (376.52 mM; ultrasonic and adjust pH to 9 with NH4OH)
DMSO : 80 mg/mL (200.81 mM; Need ultrasonic and warming)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5101 mL	12.5505 mL	25.1010 mL
	5 mM	0.5020 mL	2.5101 mL	5.0202 mL
	10 mM	0.2510 mL	1.2551 mL	2.5101 mL

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

In Vivo

1. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 10 mg/mL (25.10 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (6.28 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.28 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Sulfasalazine (NSC 667219) is an anti-rheumatic agent for the research of rheumatoid arthritis and ulcerative colitis. Sulfasalazine can suppress NF-κB activity. Sulfasalazine is a type 1 ferroptosis inducer[1][2][3][4].

IC_{50} & Target	RelA	Autophagy

In Vitro

Treatment of SW620 colon cells with sulfasalazine inhibits TNFα-, LPS-, or phorbol ester-induced NFκB activation. NFκB-

	dependent transcription is inhibited by sulfasalazine at micro- to millimolar concentrations. TNFα-induced nuclear translocation of NFκB is prevented by sulfasalazine through inhibition of IκBα degradation[1]. Pre-incubation with 5 mM of sulfasalazine alone significantly increases basal mRNA expression of all pro-inflammatory cytokines with levels of IL-6 mRNA increased by 80-fold compared with vehicle control[2]. Once digested, sulfasalazine is cleaved into sulfapyridine and 5-aminosalicylic acid by colonic bacteria, and the latter, too, is reported to suppress NF-kappaB activity[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	At low doses (0.25 mM), SAS is able to suppress glioma growth by over 60% compared to untreated controls[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay NF-κB [1]	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Cell Assay [1]	Sulfasalazine is dissolved in culture medium. SW620 cells are grown in Dulbecco's modified Eagle medium, supplemented with 10% heat-inactivated FCS, 2 mmol/liter glutamine, and 1% (wt/vol) penicillin/streptomycin. SW620 cells are transfected with the 3xIgkBLuc reporter construct. After 18 h, cells are incubated with either medium alone or with sulfasalazine (0.1, 0.2, 0.5, 1, 2, 5 mM) before stimulation with TNFα, LPS, or PMA. Luciferase assay is performed[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Mice: Sulfasalazine is dissolved in 0.1 M NaOH, and then neutralized by titrating with 0.1 M HCl. U-87MG glioma cells are implanted into the cranium of a SCID mouse. After 7 days, animals are randomized into three groups of five animals each. One group receives 1 mL i.p. saline injections twice daily for 3 weeks. The two test groups receives 8 mg of sulfasalazine in 1 mL saline twice daily for 3 weeks. Tumor growth and animal health were monitored. After perfusion with 4% paraformaldehyde, mouse brains were collected, rinsed, and placed in 30% sucrose[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Res. 2018 Dec;28(12):1171-1185.
- Brain Behav Immun. 2020 Nov;90:108-137.
- Water Res. 2023 May 21, 120110.
- Cell Death Differ. 2022 Nov 29.
- Mol Ther. 2021 Mar 17;S1525-0016(21)00142-8.

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REFERENCES

[1]. Wahl C, et al. Sulfasalazine: a potent and specific inhibitor of nuclear factor kappa B. J Clin Invest. 1998 Mar 1;101(5):1163-74.

[2]. Sykes L, et al. Sulfasalazine augments a pro-inflammatory response in interleukin-1β-stimulated amniocytes and myocytes. Immunology. 2015 Dec;146(4):630-44.

[3]. Chung WJ, et al. Sulfasalazine inhibits the growth of primary brain tumors independent of nuclear factor-kappaB. J Neurochem. 2009 Jul;110(1):182-93.

[4]. Mao C, et al. DHODH-mediated ferroptosis defence is a targetable vulnerability in cancer. Nature. 2021;593(7860):586-590.

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

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