Stattic

Cat. No.: HY-13818

CAS No.: 19983-44-9

Molecular Formula: $C_8H_5NO_4S$

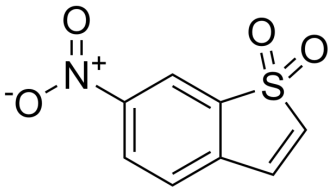
Molecular Weight: 211.19

Target: STAT; Apoptosis

Pathway: JAK/STAT Signaling; Stem Cell/Wnt; Apoptosis

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (236.75 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.7351 mL	23.6754 mL	47.3507 mL
5 mM	0.9470 mL	4.7351 mL	9.4701 mL
10 mM	0.4735 mL	2.3675 mL	4.7351 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (11.84 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (11.84 mM); Clear solution
3. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 1.67 mg/mL (7.91 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Stattic is a potent STAT3 inhibitor and inhibits STAT3 phosphorylation (at Y705 and S727)[1]. Stattic inhibits the binding of a high affinity phosphopeptide for the SH2 domain of STAT3[2]. Stattic ameliorates the renal dysfunction in Alport syndrome (AS) mice[3].

IC_{50} & Target

STAT3

In Vitro

Stattic (20 μM; 24 hours) inhibits STAT3 phosphorylation (Y705) and selectively inhibits P-STAT3 as demonstrated by the lack

	of inhibition of P-ERK1/2 in ALDH$^+$ and D44$^+$/CD24$^+$ subpopulations of Panc-1 and HPAC pancreatic cancer cell lines[1]. Stattic (2.5, 5, 10 μM; for 4 h) significantly reduces the nuclear level of pSTAT3 and survivin in PC3M-1E8 cells at 10 μM. Stattic (2.5-10 μM; for 24 h) inhibits IL-6-induced STAT3 activation in a dose-dependent manner[2]. Stattic (2.5, 5, 10 μM; for 48 h) suppresses both the growth and induces apoptosis prostate cancer cells (PC3M-1E8 cells) with 10 μM. Stattic does not induce significant cell apoptosis with 2.5 μM, 5 μM[2]. Stattic (2.5, 5, 10 μM; for 48 h) shows significant S phase accumulation[2]. Stattic can not lead to significant morphological changes or apoptosis and has little STAT3 phosphorylation in A2780 cells and HUVECs[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Stattic (10 mg/kg; i.p.; three times per week for 10 week) ameliorates the renal dysfunction in Alport syndrome (AS) mice[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Age-matched wild-type (WT) C57BL/6 mice[3]
Dosage:	10 mg/kg
Administration:	IP; three timesper week for 10 week
Result:	Increased levels of proteinuria, BUN and serum creatinine. Significantly suppressed the gene expression levels of renal injury markers (Lcn2, Kim-1), pro-inflammatory cytokines (Il-6, KC), pro-fibrotic genes (Tgf-β, Col1a1, α-Sma) and Mmp9.

CUSTOMER VALIDATION

- Mol Cancer. 2019 Mar 30;18(1):64.
- Cell Metab. 2019 Jan 8;29(1):141-155.e9.
- Ann Rheum Dis. 2024 Aug 20:ard-2024-226067.
- Gut. 2020 Jan;69(1):122-132.
- Sci Transl Med. 2021 Oct 6;13(614):eabg6428.

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REFERENCES

[1]. Lin L, et al. STAT3 as a potential therapeutic target in ALDH+ and CD44+/CD24+ stem cell-like pancreatic cancer cells. Int J Oncol. 2016 Oct 12.

[2]. John S McMurray, et al. A new small-molecule Stat3 inhibitor. Chem Biol. 2006 Nov;13(11):1123-4.

[3]. Tsubasa Yokota, et al. STAT3 inhibition attenuates the progressive phenotypes of Alport syndrome mouse model. Nephrol Dial Transplant. 2018 Feb 1;33(2):214-223.

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