STF-31

Cat. No.: HY-18728

CAS No.: 724741-75-7

Molecular Formula: $C_{23}H_{25}N_3O_3S$

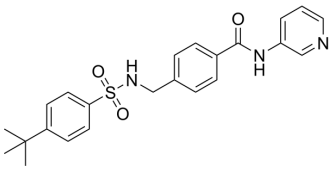
Molecular Weight: 423.53

Target: Autophagy; GLUT

Pathway: Autophagy; Membrane Transporter/Ion Channel

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 : ≥ 34 mg/mL (80.28 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3611 mL	11.8055 mL	23.6111 mL
	5 mM	0.4722 mL	2.3611 mL	4.7222 mL
	10 mM	0.2361 mL	1.1806 mL	2.3611 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 (5.90 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.90 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

STF-31 is a selective inhibitor of glucose transporter 1 (GLUT1), with an IC_{50} of 1 μM. STF-31 is also a NAMPT inhibitor. STF-31 inhibits glucose uptake in renal cell carcinoma (RCC) 4 cells[1][2].

IC_{50} & Target

GLUT1

1 μM (IC_{50})

In Vitro

STF-31 (0.01-10 μM; 10 days) is specifically toxic to RCC4 cells, whereas RCC4/VHL cells are relatively unaffected. RCC4/VHL cells treated with STF-31 (5 μM; 10 days) largely recovery, whereas RCC4 cells under the same conditions does not[1].

?STF-31 (1.25-5 μM; 3 days) does not induce autophagy, apoptosis, or DNA damage. STF-31 causes a necrotic cell death[1].

?STF-31 reduces the efferocytosis of wild type and SLC2A1-overexpressing LR73 cells[4].

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

Cell Viability Assay[1]

Cell Line:	Wild-type RCC4 cells (RCC4/VHL) and RCC4 cells without VHL
Concentration:	0.01, 0.1, 1, 10 μM
Incubation Time:	10 days
Result:	Reduced viability of RCC4 cells without VHL in a concentration-dependent manner when compared to their wild-type counterparts.

In Vivo

STF-31 (10 mg/kg; i.p.; twice daily for 2 days, followed by once daily for another 3 days) does not affect normal mice body weight, behavior, and ERG responses. STF-31 reduces light-induced CX3CR1$^{gfp/+}$ mice microglial activation and retinal degeneration[3].

?STF-31 (10 mg/kg; i.p.) promotes accumulation of necrotic thymocytes after Dexamethasone-induced apoptosis in vivo[4].

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

Animal Model:	Twelve-week old C57BL/6J and CX3CR1$^{gfp/+}$ mice[3]
Dosage:	10 mg/kg
Administration:	I.p. injections; twice daily for 2 days, followed by once daily for another 3 days
Result:	Improved photoreceptor survival and reduced microglial activation of CX3CR1$^{gfp/+}$ mice, and not induced any C57BL/6J mice retinal cell death.

CUSTOMER VALIDATION

- Cell Biosci. 2024 Apr 16;14(1):48.
- Arch Toxicol. 2022 Nov;96(11):2913-2926.
- Arch Toxicol. 2022 May 4.
- J Agric Food Chem. 2021 Oct 25.
- Fish Shellfish Immunol. 2022 Aug 3;S1050-4648(22)00414-4.

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REFERENCES

[1]. Morioka S, et, al. Efferocytosis induces a novel SLC program to promote glucose uptake and lactate release. Nature. 2018 Nov;563(7733):714-718.

[2]. Chan DA, et al. Targeting GLUT1 and the Warburg effect in renal cell carcinoma by chemical synthetic lethality. Sci Transl Med. 2011 Aug 3;3(94):94ra70.

[3]. Dedda CD, et al. Pharmacological Targeting of GLUT1 to Control Autoreactive T Cell Responses. International Journal of Molecular Sciences. 2019 Oct 8; 20(19):4962.

[4]. Wang L, et al. Glucose transporter 1 critically controls microglial activation through facilitating glycolysis. Molecular Neurodegeneration, 2019 Jan 11; 14(1):2.

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

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