SF1670

Cat. No.: HY-15842

CAS No.: 345630-40-2

Molecular Formula: $C_{19}H_{17}NO_3$

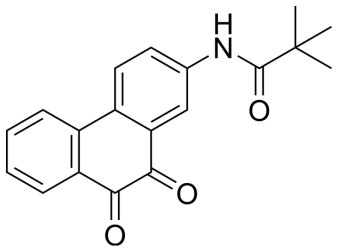
Molecular Weight: 307.34

Target: PTEN; Phosphatase; Autophagy

Pathway: PI3K/Akt/mTOR; Metabolic Enzyme/Protease; Autophagy

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (162.69 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.2537 mL	16.2686 mL	32.5373 mL
5 mM	0.6507 mL	3.2537 mL	6.5075 mL
10 mM	0.3254 mL	1.6269 mL	3.2537 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.13 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

SF1670 is a potent and specific phosphatase and tensin homolog deleted on chromosome 10 (PTEN) inhibitor[1].

IC_{50} & Target

PTEN[1]

In Vitro

SF1670 is a specific PTEN inhibitor with prolonged intracellular retention in neutrophils. SF1670 enhances PtdIns(3,4,5)P3 signaling in transplanted neutrophils. SF1670 also elevates Akt phosphorylation in murine cells. Consistent with the enhanced Akt phosphorylation, pretreatment with SF1670 also significantly augments PtdIns(3,4,5)P3 level in mouse neutrophils. SF1670-induced Akt hyperactivation is abolished in PTEN-null neutrophils, further demonstrating that this effect is mediated by specific inhibition of PTEN activity. At 500 nM fMLP stimulation, SF1670 (500 nM)–pretreated neutrophils show nearly 70% higher (maximal) superoxide production than untreated neutrophils[1]. HCT116 cells are pre-treated with the PTEN inhibitor SF1670 (2 μM) for 24 h (untreated HCT116 cells served as control); treated cells are

	subsequently plated under non-adherent conditions with added MET (60 μM), Lun (2 μM), or Gen (2 μM). SF1670 binds to the PTEN active site, resulting in elevated phosphatidylinositol (3,4,5) triphosphate signaling[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	SF1670 (3 mg/kg; i.p.) triggers postconditioning after inducing cerebral global ischaemia (17 min) and reperfusion (24 h) induced injury via occlusion of both carotid arteries in mice[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Swiss albino male mice (bodyweight 25-30 g)[1]
Dosage:	3 mg/kg
Administration:	Treatment with i.p.
Result:	Lead to attenuation of cerebral I/R-induced increase in thiobarbituric acid reactive substances (TBARS).

CUSTOMER VALIDATION

- Autophagy. 2021 Mar 22.
- Cell Death Dis. 2020 Aug 3;11(8):646.
- Cell Death Dis. 2019 Mar 13;10(3):248.
- Oncogene. 2020 Feb;39(8):1739-1755.
- Biochem Pharmacol. 2020 Jan;171:113674.

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REFERENCES

[1]. Li Y, et al. Pretreatment with phosphatase and tensin homolog deleted on chromosome 10 (PTEN) inhibitor SF1670augments the efficacy of granulocyte transfusion in a clinically relevant mouse model. Blood. 2011 Jun 16;117(24):6702-13.

[2]. Montales MT, et al. Metformin and soybean-derived bioactive molecules attenuate the expansion of stem cell-like epithelial subpopulation and confer apoptotic sensitivity in human colon cancer cells. Genes Nutr. 2015 Nov;10(6):49.

[3]. Amarjot Kaur Grewal, et al. Neuroprotective effect of pharmacological postconditioning on cerebral ischaemia-reperfusion-induced injury in mice. J Pharm Pharmacol. 2019 Jun;71(6):956-970.

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