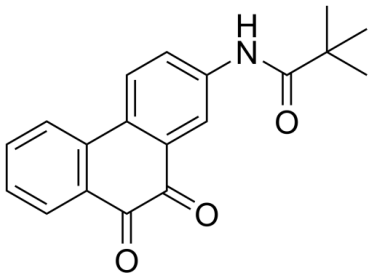
Data Sheet

Product Name:	SF1670
Cat. No.:	CS-6115
CAS No.:	345630-40-2
Molecular Formula:	$C_{19}H_{17}NO_3$
Molecular Weight:	307.34
Target:	Autophagy; Phosphatase; PTEN
Pathway:	Autophagy; Metabolic Enzyme/Protease; PI3K/Akt/mTOR
Solubility:	DMSO : ≥ 50 mg/mL



BIOLOGICAL ACTIVITY:

SF1670 is a potent and specific phosphatase and tensin homolog deleted on chromosome 10 (**PTEN**) inhibitor[1]. IC50 & 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]. ***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].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: [2]The human colon cancer cell lines **HT29** and **HCT116** are propagated in McCoy's medium (ATCC) supplemented with 10 % fetal bovine serum (FBS) and 5 % antibiotic-antimycotic solution (ABAM) in a humidified incubator (5 % CO_2:95 % air) at 37°C. Cells are seeded in six-well plates at an initial density of 2×10^5 per well, and treated (in culture medium) with Metformin (MET 60 μM), Lunasin (Lun 2 μM), β-conglycinin (β-con 3 μM), Glycinin (Gly 3 μM), and Genistein (Gen 2 μM), alone or in combination. β-con and Gly are isolated and purified as described below. Metformin, Lun, β-con, and Gly are dissolved in phosphate-buffered saline (PBS), whereas Gen is dissolved in DMSO. In other experiments, cells are treated with insulin (2 μM), PTEN inhibitor **SF1670 (2 μM)**, and 5-Fluorouracil (5-FU 50 μM). Treated cells are collected at select time points for subsequent analyses[2].

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.

CAIndexNames:

Propanamide, N-(9,10-dihydro-9,10-dioxo-2-phenanthrenyl)-2,2-dimethyl-

SMILES:

CC(C)(C)C(NC(C=C1C2=O)=CC=C1C3=C(C2=O)C=CC=C3)=O

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