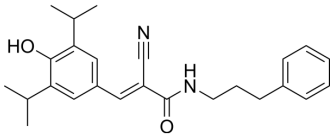
SU1498

Cat. No.:	HY-19326
CAS No.:	168835-82-3
Molecular Formula:	$C_{25}H_{30}N_2O_2$
Molecular Weight:	390.52
Target:	VEGFR
Pathway:	Protein Tyrosine Kinase/RTK
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 : 100 mg/mL (256.07 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5607 mL	12.8034 mL	25.6069 mL
	5 mM	0.5121 mL	2.5607 mL	5.1214 mL
	10 mM	0.2561 mL	1.2803 mL	2.5607 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 (6.40 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (6.40 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

SU1498 (AG 1498) is a selective inhibitor of the VEGFR2; inhibits Flk-1 with an IC_{50} of value of 700 nM[1].

IC_{50} & Target

Flk-1
700 nM (IC_{50})

In Vitro

SU1498 stimulates accumulation of phosphorylated ERKs in human umbilical vein endothelial cells and in human aortic endothelial cells in a manner that is dependent on the functioning of the upstream components of the MAPK pathway, B-Raf, and MEK kinases. The enhanced accumulation of phospho-ERKs is observed only in cells that have been stimulated with sphingosine 1-phosphate or protein growth factors; SU1498 by itself is ineffective[2]. SU1498 blocks signal transduction from VEGFR2 in MS1 VEGF cells.In the presence of SU1498, levels of Ets-1 are decreased, suggesting that VEGF-VEGFR-2

interactions contributed to baseline levels of Ets-1 expression, and interruption of this autocrine interaction with SU1498 led to decreased expression of Ets-1[3]. SU1498 treatment significantly impacts U87 cell proliferation and apoptosis. SU1498 induces a marked increase in lipids and a decrease in glycerophosphocholine. Accordingly, accumulation of lipid droplets is seen in the cytoplasm of SU1498-treated U87 cells[4].

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

PROTOCOL

Kinase Assay [2]

The ERK1 or ERK2 solution is pipetted into tubes (1 µL per tube) and mixed with 0-10 µL of 50 µM SU1498 (in kinase buffer without ATP). The blank tube receives buffer only. The volume is adjusted to 11 µL with the same buffer, and the mixtures are incubated for 10 min at 25°C. This is followed by the addition of 40 µL of the Elk1-ATP-buffer solution, and the incubations are continued for 30 min at 30°C. The reactions are stopped with 20 µL of 4× sample buffer mix and heating at 95°C for 10 min. Samples (15 µL) are fractionated by SDS-PAGE, and phosphorylated Elk1 is detected by immunoblotting with anti-phospho-Elk1 antibody[2].

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

Cell Assay [4]

For cell proliferation assay, U87 cells are seeded in 24-well plates (30,000 cells/well) and allowed to attach overnight. Cells are then treated for 24 or 72 h with different concentrations of Bevacizumab (from 10 ng/mL to 250 µg/mL) or SU1498 (from 1 µM to 30 µM) in triplicate wells. The cell viability is then assessed with the MTT assay[4].

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

CUSTOMER VALIDATION

- Adv Funct Mater. 2021 May 24.
- Phytomedicine. 2020 Nov;78:153300.
- Glia. 2023 Mar 24.
- Cell Signal. 2021 Jan;77:109812.
- Cell Mol Neurobiol. 2023 Jul 7.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Strawn LM, et al. Flk-1 as a target for tumor growth inhibition.Cancer Res. 1996 Aug 1;56(15):3540-5.

[2]. Boguslawski G, et al. SU1498, an inhibitor of vascular endothelial growth factor receptor 2, causes accumulation of phosphorylated ERK kinases and inhibits their activity in vivo and in vitro.J Biol Chem. 2004 Feb 13;279(7):5716-24.

[3]. Arbiser JL, et al. Overexpression of VEGF 121 in immortalized endothelial cells causes conversion to slowly growing angiosarcoma and high level expression of the VEGF receptors VEGFR-1 and VEGFR-2 in vivo.Am J Pathol. 2000 Apr;156(4):1469-76.

[4]. Mesti T, et al. Metabolic impact of anti-angiogenic agents on U87 glioma cells.PLoS One. 2014 Jun 12;9(6):e99198.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA