SU16f

Cat. No.: HY-108628

CAS No.: 251356-45-3

Molecular Formula: $C_{24}H_{22}N_2O_3$

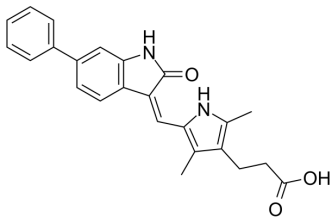
Molecular Weight: 386.44

Target: PDGFR

Pathway: Protein Tyrosine Kinase/RTK

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (129.39 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5877 mL	12.9386 mL	25.8772 mL
	5 mM	0.5175 mL	2.5877 mL	5.1754 mL
	10 mM	0.2588 mL	1.2939 mL	2.5877 mL

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

BIOLOGICAL ACTIVITY

Description

SU16f is a potent and selective PDGFRβ inhibitor with IC_{50}s of 10 nM, 140 nM, 2.29 μM for PDGFRβ, PDGFR1, PDGFR2, respectively[1]. Neutralization of PDGFRβ receptor by SU16f blocks the promoting role of GC-MSCs (gastric cancer-derived mesenchymal stem cells) conditioned medium in gastric cancer cell proliferation and migration[2].

IC_{50} & Target

PDGFRβ	PDGFR2	PDGFR1
10 nM (IC_{50})	140 nM (IC_{50})	2.29 μM (IC_{50})

In Vitro

SU16f (20 μM; for 8 hours) pretreatment inhibits the promoting role of GC-MSC-CM in SGC-7901 cell proliferation[1].
SU16f (20 μM; for 8 hours) significantly abolishes PDGFRβ activation in SGC-7901 by GC-MSC-CM. SU16f pretreatment results in the upregulation of E-cadherin and downregulation of N-cadherin, Vimentin, and α-SMA. SU16f pretreatment leads to downregulation of p-AKT, Bcl-xl, and Bcl-2 levels and upregulation of Bax expression in SGC-7901 cells by GC-MSC-CM [1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	SGC-7901 cells in GC-MSC/SGC-7901 co-culture system

Concentration:	20 μM
Incubation Time:	8 hours
Result:	Inhibited the promoting role of GC-MSC-CM in SGC-7901 cell proliferation.

Western Blot Analysis[1]

Cell Line:	SGC-7901 cells
Concentration:	20 μM
Incubation Time:	8 hours
Result:	Significantly abolished PDGFRβ activation in SGC-7901 by GC-MSC-CM, and resulted in the upregulation of E-cadherin and downregulation of N-cadherin, Vimentin, and α-SMA.

REFERENCES

[1]. Huang F, et al. Gastric cancer-derived MSC-secreted PDGF-DD promotes gastric cancer progression. J Cancer Res Clin Oncol. 2014 Nov;140(11):1835-48.

[2]. Sun L, et al. Design, synthesis, and evaluations of substituted 3-[(3- or 4-carboxyethylpyrrol-2-yl)methylidenyl]indolin-2-ones as inhibitors of VEGF, FGF, and PDGF receptor tyrosine kinases. J Med Chem. 1999 Dec 16;42(25):5120-30.

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