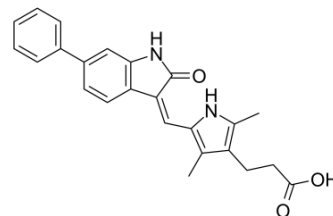


SU16f

Cat. No.:	HY-108628	
CAS No.:	251356-45-3	
Molecular Formula:	C ₂₄ H ₂₂ N ₂ O 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



BIOLOGICAL ACTIVITY

Description	SU16f is a potent and selective PDGFR β inhibitor with IC ₅₀ s of 10 nM, 140 nM, 2.29 μ M for PDGFR β , PDGFR1, PDGFR2, respectively ^[1] . Neutralization of PDGFR β 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] .																		
IC₅₀ & Target	PDGFR β 10 nM (IC ₅₀)	PDGFR2 140 nM (IC ₅₀)	PDGFR1 2.29 μ M (IC ₅₀)																
In Vitro	<p>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.</p> <p>Cell Proliferation Assay^[1]</p> <table border="1"> <tr> <td>Cell Line:</td> <td>SGC-7901 cells in GC-MSC/SGC-7901 co-culture system</td> </tr> <tr> <td>Concentration:</td> <td>20 μM</td> </tr> <tr> <td>Incubation Time:</td> <td>8 hours</td> </tr> <tr> <td>Result:</td> <td>Inhibited the promoting role of GC-MSC-CM in SGC-7901 cell proliferation.</td> </tr> </table> <p>Western Blot Analysis^[1]</p> <table border="1"> <tr> <td>Cell Line:</td> <td>SGC-7901 cells</td> </tr> <tr> <td>Concentration:</td> <td>20 μM</td> </tr> <tr> <td>Incubation Time:</td> <td>8 hours</td> </tr> <tr> <td>Result:</td> <td>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.</td> </tr> </table>			Cell Line:	SGC-7901 cells in GC-MSC/SGC-7901 co-culture system	Concentration:	20 μ M	Incubation Time:	8 hours	Result:	Inhibited the promoting role of GC-MSC-CM in SGC-7901 cell proliferation.	Cell Line:	SGC-7901 cells	Concentration:	20 μ M	Incubation Time:	8 hours	Result:	Significantly abolished PDGFR β 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.
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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)methylidene]indolin-2-ones as inhibitors of VEGF, FGF, and PDGF receptor tyrosine kinases. J Med Chem. 1999 Dec 16;42(25):5120-30.

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

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