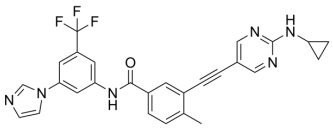
S116836

Cat. No.:	HY-123450
CAS No.:	1257628-57-1
Molecular Formula:	$C_{27}H_{21}F_3N_6O$
Molecular Weight:	502.49
Target:	Bcr-Abl; Apoptosis; PDGFR
Pathway:	Protein Tyrosine Kinase/RTK; Apoptosis
Storage:	4°C, stored under nitrogen * In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 32 mg/mL (63.68 mM; ultrasonic and warming and heat to 80°C)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.9901 mL	9.9504 mL	19.9009 mL
	5 mM	0.3980 mL	1.9901 mL	3.9802 mL
	10 mM	0.1990 mL	0.9950 mL	1.9901 mL

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

BIOLOGICAL ACTIVITY

Description

S116836, a potent, orally active BCR-ABL tyrosine kinase inhibitor, blocks both wild-type as well as T315I Bcr-Abl. S116836 arrests the cells in the G0/G1 phase of cell cycle, induces apoptosis, increases ROS production, and decreases GSH production in BaF3/WT and BaF3/T315I cells. S116836 also inhibits SRC, LYN, HCK, LCK and BLK, and receptor tyrosine kinases such as FLT3, TIE2, KIT, PDGFR-β. Antitumor activies[1][2][3]. S116836 is a click chemistry reagent, it contains an Alkyne group and can undergo copper-catalyzed azide-alkyne cycloaddition (CuAAc) with molecules containing Azide groups.

IC_{50} & Target	$Bcr\text{-}Abl^{WT}$	$Bcr\text{-}Abl^{T315I}$

In Vitro

S116836 (0.01-1 μM; 24 hours) significantly reduces the cellular proliferation of BaF3/WT and BaF3/T315I cells (IC_{50} values of 0.05 μM and 0.20 μM, respectively)[1].

S116836 (0.01-1 μM; 24 hours) significantly downregulates the expression level of p-BCR-ABL in BaF3/WT cells. S116836 (0.01-1 μM; 24 hours) also significantly downregulates the expression level of p-Crkl and p-STAT5 (downstream signaling proteins of BCR-ABL) in both BaF3/WT and BaF3/T315I cells[1].

S116836 (0.1, 0.3, and 0.5 μM; 24 hours) arrests the BaF3/WT and BaF3/T315I cells in G0/G1 phase of the cell cycle[1].

S116836 (0.3 and 0.5 μM; 24 hours) increases ROS production and decreases GSH levels in BaF3/WT and BaF3/T315I cells[1].

S116836 (0.1, 0.3, and 0.5 μM; 24 hours) induces apoptosis in BaF3/WT and BaF3/T315I cells[1].

	S116836 potently inhibits PDGFRα and its downstream signaling molecules such as STAT3, AKT, and Erk1/2. S116836 effectively inhibits the growth of the WT and T674I FIP1L1-PDGFRα-expressing neoplastic cells[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	S116836 (100 or 200 mg/kg; i.p.; q3d×6, athymic NCR nude mice) decreases the volume and weight of xenograft tumors expressing WT and T315I mutant BCR-ABL[1]. S116836 (200mg/kg/d, oral gavage for 14 days) inhibits the growth of xenografted T674I-FIP1L1-PDGFRα cells in nude mice[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Gupta P, et al. Preclinical development of a novel BCR-ABL T315I inhibitor against chronic myeloid leukemia. Cancer Lett. 2020;472:132-141.

[2]. Bu Q, et al. SAHA and S116836, a novel tyrosine kinase inhibitor, synergistically induce apoptosis in imatinib-resistant chronic myelogenous leukemia cells. Cancer Biol Ther. 2014;15(7):951-962.

[3]. Shen Y, et al. Antitumor activity of S116836, a novel tyrosine kinase inhibitor, against imatinib-resistant FIP1L1-PDGFRα-expressing cells. Oncotarget. 2014;5(21):10407-10420.

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