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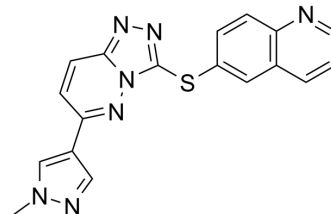
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SGX-523

Cat. No.:	HY-12019
CAS No.:	1022150-57-7
Molecular Formula:	C ₁₈ H ₁₃ N ₇ S
Molecular Weight:	359.41
Target:	c-Met/HGFR
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 : 30 mg/mL (83.47 mM; Need ultrasonic)				
	Preparing Stock Solutions	Solvent Concentration Mass	1 mg	5 mg	10 mg
		1 mM	2.7823 mL	13.9117 mL	27.8234 mL
		5 mM	0.5565 mL	2.7823 mL	5.5647 mL
		10 mM	0.2782 mL	1.3912 mL	2.7823 mL
Please refer to the solubility information to select the appropriate solvent.					
In Vivo	1. SGX-523 is prepared in 0.5% sodium carboxymethyl cellulose ^[2] .				

BIOLOGICAL ACTIVITY

Description	SGX523 is a exquisitely selective and ATP-competitive MET inhibitor. SGX523 potently inhibits MET with an IC ₅₀ of 4 nM and is >1,000-fold selective versus other protein kinases. Antitumor activity ^[1] .
In Vitro	SGX523 shows ATP-competitive inhibition with higher apparent affinity for the less active, unphosphorylated form of MET [MET-KD(0P), K_i=2.7 nM] versus the more active phospho-enzyme [MET-KD(3P), K_i=23 nM]^[1]. SGX523 inhibits the growth of gastric and lung cancer cell lines with amplification of the MET gene but has no effect, even at high micromolar concentration, on cell lines with normal MET gene copy number. The IC₅₀s of 0.02, 0.113, and 0.035 μM for NSCLC H1993, gastric cancer MKN45, and gastric cancer Hs746T cells, respectively^[1]. The IC₅₀ value for the inhibition of MET autophosphorylation is 0.040 μM in GTL16 cells^[1]. SGX523 (0.5, 1.5, 4.6, 13.7, 41, 123, 370, 1100, 3300, 10000 nM; 1 hour) inhibits MET autophosphorylation without affecting total MET or extracellular signal-regulated kinase protein levels in HGF-stimulated A549 cells^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only. Cell Viability Assay^[1]

	Cell Line:	Gastric cancer cell line GTL16
	Concentration:	4.6, 14, 40, 120, 370, 1100, 3300, 10000 nM
	Incubation Time:	1 hours
	Result:	Abolished constitutive signaling induced by MET gene amplification.
In Vivo	SGX523 exhibits antitumor activity in vivo. SGX523 inhibits MET-dependent tumor growth ^[2] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.	
	Animal Model:	Female Harlan nude mice (athymic nu/nu) were s.c. implanted with U87 cells ^[2]
	Dosage:	10 or 30 mg/kg
	Administration:	Oral gavage; twice daily starting at day 5 for 22 days
	Result:	Potently inhibited U87MG tumor growth at a dose of 10 mg/kg administered twice daily. Led to clear regression of U87MG tumors at 30 mg/kg dosed twice daily.

CUSTOMER VALIDATION

- Science. 2017 Dec 1;358(6367):eaan4368.
- Sci Transl Med. 2018 Jul 18;10(450):eaaq1093.
- Cell Rep Med. 2024 Feb 20;5(2):101388.
- Cancer Res. 2015 Nov 1;75(21):4548-59.
- Toxicol Lett. 2023 Oct 6:S0378-4274(23)01056-1.

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REFERENCES

[1]. Buchanan SG, et al. SGX523 is an exquisitely selective, ATP-competitive inhibitor of the MET receptor tyrosine kinase with antitumor activity in vivo. Mol Cancer Ther, 2009, 8(12), 3181-3190.

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

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