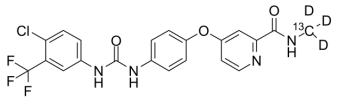
Sorafenib-^{13}C,d_3

Cat. No.:	HY-10201S2
CAS No.:	1210608-86-8
Molecular Formula:	$C_{20}{}^{13}CH_{13}D_3ClF_3N_4O_3$
Molecular Weight:	468.84
Target:	Raf; VEGFR; FLT3; Autophagy; Apoptosis; Ferroptosis
Pathway:	MAPK/ERK Pathway; Protein Tyrosine Kinase/RTK; Autophagy; Apoptosis
Storage:	Powder -20°C 3 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 16.67 mg/mL (35.56 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.1329 mL	10.6646 mL	21.3292 mL
	5 mM	0.4266 mL	2.1329 mL	4.2658 mL
	10 mM	0.2133 mL	1.0665 mL	2.1329 mL

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

BIOLOGICAL ACTIVITY

Description

Sorafenib-^{13}C,d_3 is the ^{13}C- and deuterium labeled Sorafenib. Sorafenib (Bay 43-9006) is a potent and orally active Raf inhibitor with IC50s of 6 nM and 20 nM for Raf-1 and B-Raf, respectively. Sorafenib is a multikinase inhibitor with IC50s of 90 nM, 15 nM, 20 nM, 57 nM and 58 nM for VEGFR2, VEGFR3, PDGFRβ, FLT3 and c-Kit, respectively. Sorafenib induces autophagy and apoptosis. Sorafenib has anti-tumor activity. Sorafenib is a ferroptosis activator[1].

In Vitro

Stable heavy isotopes of hydrogen, carbon, and other elements have been incorporated into drug molecules, largely as tracers for quantitation during the drug development process. Deuteration has gained attention because of its potential to affect the pharmacokinetic and metabolic profiles of drugs[1].

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

CUSTOMER VALIDATION

- BMC Cancer. 2023 Jan 25;23(1):87.

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REFERENCES

[1]. Russak EM, et al. Impact of Deuterium Substitution on the Pharmacokinetics of Pharmaceuticals. Ann Pharmacother. 2019;53(2):211-216.

[2]. Wilhelm SM, et al. BAY 43-9006 exhibits broad spectrum oral antitumor activity and targets the RAF/MEK/ERK pathway and receptor tyrosine kinases involved in tumor progression and angiogenesis. Cancer Res. 2004 Oct 1;64(19):7099-109.

[3]. El-Ashmawy NE, et al. Sorafenib effect on liver neoplastic changes in rats: more than a kinase inhibitor. Clin Exp Med. 2016 Apr 16.

[4]. Jin W, et al. Long non-coding RNA TUC338 is functionally involved in sorafenib-sensitized hepatocarcinoma cells by targeting RASAL1. Oncol Rep. 2017 Jan;37(1):273-280.

[5]. Li M, et al. Activation of an AKT/FOXM1/STMN1 pathway drives resistance to tyrosine kinase inhibitors in lung cancer. Br J Cancer. 2017 Aug 29.

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