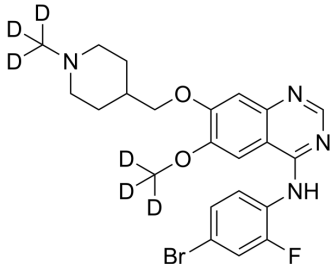
Vandetanib-d_6

Cat. No.:	HY-10260S
CAS No.:	1174683-49-8
Molecular Formula:	$C_{22}H_{18}D_6BrFN_4O_2$
Molecular Weight:	481.39
Target:	VEGFR; Autophagy; Apoptosis; Isotope-Labeled Compounds
Pathway:	Protein Tyrosine Kinase/RTK; Autophagy; Apoptosis; Others
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Vandetanib-d_6 is the deuterium labeled Vandetanib. Vandetanib (D6474) is a potent, orally active inhibitor of VEGFR2/KDR tyrosine kinase activity (IC50=40 nM). Vandetanib also has activity versus the tyrosine kinase activity of VEGFR3/FLT4 (IC50=110 nM) and EGFR/HER1 (IC50=500 nM)[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.

REFERENCES

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

[2]. Wedge SR, et al. ZD6474 inhibits vascular endothelial growth factor signaling, angiogenesis, and tumor growth following oral administration. Cancer Res. 2002 Aug 15;62(16):4645-55.

[3]. Hegedus C, et al. Interaction of the EGFR inhibitors gefitinib, vandetanib, pelitinib and neratinib with the ABCG2 multidrug transporter: implications for the emergence and reversal of cancer drug resistance. Biochem Pharmacol. 2012 Aug 1;84(3):260-7.

[4]. Takeda H, et al. Vandetanib is effective in EGFR-mutant lung cancer cells with PTEN deficiency. Exp Cell Res. 2013 Feb 15;319(4):417-23.

[5]. Inoue K, et al. Vandetanib, an inhibitor of VEGF receptor-2 and EGF receptor, suppresses tumor development and improves prognosis of liver cancer in mice. Clin Cancer Res. 2012 Jul 15;18(14):3924-33.

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