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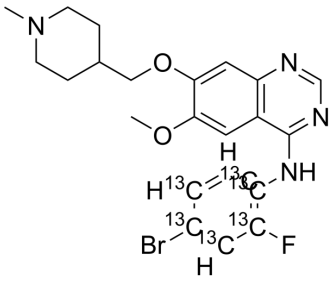
Vandetanib-$^{13}C_6$

Cat. No.:	HY-10260S2
CAS No.:	1261397-03-8
Molecular Formula:	$C_{16}{}^{13}C_6H_{24}BrFN_4O_2$
Molecular Weight:	481.31
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-13C6 is a deuterated labeled Vandetanib[1]. Vandetanib (D6474) is a potent, orally active inhibitor of VEGFR2/KDR tyrosine kinase activity (IC_{50}=40 nM). Vandetanib also has activity versus the tyrosine kinase activity of VEGFR3/FLT4 (IC_{50}=110 nM) and EGFR/HER1 (IC_{50}=500 nM)[2].
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]. Vandetanib inhibits VEGFR3 and EGFR with IC_{50} of 110 nM and 500 nM, respectively. Vandetanib is not sensitive to PDGFRβ, Flt1, Tie-2 and FGFR1 with IC_{50} of 1.1-3.6 μM, while almost has no activity against MEK, CDK2, c-Kit, erbB2, FAK, PDK1, Akt and IGF-1R with IC_{50} above 10 μM. Vandetanib inhibits VEGF-, EGF- and bFGF-stimulated HUVEC proliferation with IC_{50} of 60 nM, 170 nM and 800 nM, with no effect on basal endothelial cell growth. Vandetanib inhibits tumor cell growth with IC_{50} of 2.7 μM (A549) to 13.5 μM (Calu-6)[2]. Odanacatib is a weak inhibitor of antigen presentation, measured in a mouse B cell line (IC_{50}=1.5±0.4 μM), compared to the Cat S inhibitor LHVS (IC_{50}=0.001 μM) in the same assay. Odanacatib also shows weak inhibition of the processing of the MHC II invariant chain protein Iip10 in mouse splenocytes compared to LHVS (minimum inhibitory concentration 1-10 μM versus 0.01 μM, respectively)[3]. Vandetanib suppresses phosphorylation of VEGFR-2 in HUVECs and EGFR in hepatoma cells and inhibits cell proliferation[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Vandetanib (15 mg/kg, p.o.) has a superior anti-tumor effect than gefitinib in the H1650 xenograft model, and suppresses tumor growth with IC_{50} of 3.5±1.2 μM[4]. In tumor-bearing mice, vandetanib (50 or 75 mg/kg) suppresses phosphorylation of VEGFR-2 and EGFR in tumor tissues, significantly reduces tumor vessel density, enhances tumor cell apoptosis, suppresses tumor growth, improves survival, reduces number of intrahepatic metastases, and upregulates VEGF, TGF-α, and EGF in tumor tissues[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. 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.

[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]. 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.

[4]. 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.

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

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