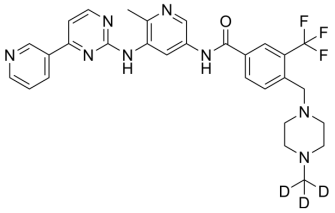
Flumatinib-d_3

Cat. No.:	HY-13904S
Molecular Formula:	$C_{29}H_{26}D_3F_3N_8O$
Molecular Weight:	565.61
Target:	Bcr-Abl; PDGFR; c-Kit; Isotope-Labeled Compounds
Pathway:	Protein Tyrosine Kinase/RTK; Others
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Flumatinib-d_3 is deuterium labeled Flumatinib. Flumatinib (HHGV678) is an orally available, selective inhibitor of Bcr-Abl. Flumatinib inhibits c-Abl, PDGFRβ and c-Kit with IC50s of 1.2 nM, 307.6 nM and 665.5 nM, respectively[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]. Gong A, et al. Metabolism of flumatinib, a novel antineoplastic tyrosine kinase inhibitor, in chronic myelogenous leukemia patients. Drug Metab Dispos. 2010 Aug;38(8):1328-40.

[3]. Luo H, et al. HH-GV-678, a novel selective inhibitor of Bcr-Abl, outperforms imatinib and effectively overrides imatinib resistance. Leukemia. 2010 Oct;24(10):1807-9.

[4]. Zhao J, et al. Flumatinib, a selective inhibitor of BCR-ABL/PDGFR/KIT, effectively overcomes drug resistance of certain KIT mutants. Cancer Sci. 2013 Nov 10.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA