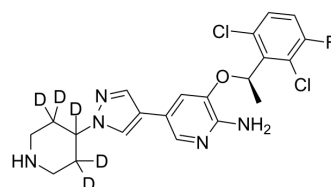


## Crizotinib-d<sub>5</sub>

|                           |  |       |          |
|---------------------------|--|-------|----------|
| <b>Cat. No.:</b>          | HY-50878S  |       |          |
| <b>CAS No.:</b>           | 1395950-84-1   |       |          |
| <b>Molecular Formula:</b> | C <sub>21</sub> H <sub>17</sub> D <sub>5</sub> Cl <sub>2</sub> FN <sub>3</sub> O |       |          |
| <b>Molecular Weight:</b>  | 455.37   |       |          |
| <b>Target:</b>            | c-Met/HGFR; ROS Kinase; Autophagy; Anaplastic lymphoma kinase (ALK)              |       |          |
| <b>Pathway:</b>           | Protein Tyrosine Kinase/RTK; Autophagy   |       |          |
| <b>Storage:</b>           | Powder   | -20°C | 3 years  |
|                           |  | 4°C   | 2 years  |
|                           | In solvent   | -80°C | 6 months |
|                           |  | -20°C | 1 month  |



### BIOLOGICAL ACTIVITY

|                    |   |
|--------------------|---|
| <b>Description</b> | Crizotinib-d <sub>5</sub> is the deuterium labeled Crizotinib. Crizotinib (PF-02341066) is an orally bioavailable, ATP-competitive ALK and c-Met inhibitor with IC <sub>50</sub> s of 20 and 8 nM, respectively. Crizotinib inhibits tyrosine phosphorylation of NPM-ALK and tyrosine phosphorylation of c-Met with IC <sub>50</sub> s of 24 and 11 nM in cell-based assays, respectively. Crizotinib is also a ROS1 inhibitor. Crizotinib has effective tumor growth inhibition <sup>[1][2][3]</sup> . |
| <b>In Vitro</b>    | 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 <sup>[1]</sup> .<br>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]. Shen A, et al. c-Myc alterations confer therapeutic response and acquired resistance to c-Met inhibitors in MET-addicted cancers. *Cancer Res.* 2015 Nov 1;75(21):4548-59.
- [3]. Umapathy G, et al. The kinase ALK stimulates the kinase ERK5 to promote the expression of the oncogene MYCN in neuroblastoma. *Sci Signal.* 2014 Oct 28;7(349):ra102.
- [4]. Tucker ER, et al. Immunoassays for the quantification of ALK and phosphorylated ALK support the evaluation of on-target ALK inhibitors in neuroblastoma. *Mol Oncol.* 2017 Aug;11(8):996-1006.
- [5]. Liu H, et al. Identifying and Targeting Sporadic Oncogenic Genetic Aberrations in Mouse Models of Triple Negative Breast Cancer. *Cancer Discov.* 2018 Mar;8(3):354-369.
- [6]. Cui JJ, et al. Structure based drug design of crizotinib (PF-02341066), a potent and selective dual inhibitor of mesenchymal-epithelial transition factor (c-MET) kinase and anaplastic lymphoma kinase (ALK). *J Med Chem.* 2011 Sep 22;54(18):6342-63.
- [7]. Cullinane C, et al. Differential (18)F-FDG and 3'-deoxy-3'-(18)F-fluorothymidine PET responses to pharmacologic inhibition of the c-MET receptor in preclinical tumor models. *J Nucl Med.* 2011 Aug;52(8):1261-7
- [8]. Christensen JG, et al. Cytoreductive antitumor activity of PF-2341066, a novel inhibitor of anaplastic lymphoma kinase and c-Met, in experimental models of anaplastic large-cell lymphoma. *Mol Cancer Ther.* 2007, 6(12 Pt 1), 3314-3322.

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[9]. Zou HY, et al. An orally available small-molecule inhibitor of c-Met, PF-2341066, exhibits cytoreductive antitumor efficacy through antiproliferative and antiangiogenic mechanisms. *Cancer Res.* 2007, 67(9), 4408-4417.

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

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