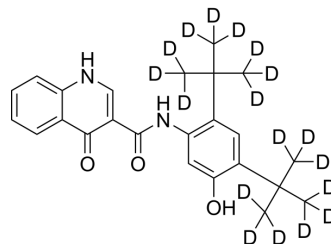


## Ivacaftor-d<sub>18</sub>

|                           |   |
|---------------------------|---|
| <b>Cat. No.:</b>          | HY-13017S2  |
| <b>CAS No.:</b>           | 1413431-05-6  |
| <b>Molecular Formula:</b> | C <sub>24</sub> H <sub>10</sub> D <sub>18</sub> N <sub>2</sub> O <sub>3</sub>             |
| <b>Molecular Weight:</b>  | 410.6   |
| <b>Target:</b>            | Autophagy; CFTR   |
| <b>Pathway:</b>           | Autophagy; Membrane Transporter/Ion Channel   |
| <b>Storage:</b>           | Please store the product under the recommended conditions in the Certificate of Analysis. |



### BIOLOGICAL ACTIVITY

|                    |  |
|--------------------|--|
| <b>Description</b> | Ivacaftor-d <sub>18</sub> is the deuterium labeled Ivacaftor[1]. Ivacaftor (VX-770) is a potent and orally bioavailable CFTR potentiator, targeting G551D-CFTR and F508del-CFTR with EC50s of 100 nM and 25 nM, respectively[2].   |
| <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 Feb;53(2):211-216.
- [2]. Delaunay JL, et al. Functional defect of variants in the adenosine triphosphate-binding sites of ABCB4 and their rescue by the cystic fibrosis transmembrane conductance regulator potentiator, ivacaftor (VX-770). *Hepatology*. 2017 Feb;65(2):560-570.

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

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