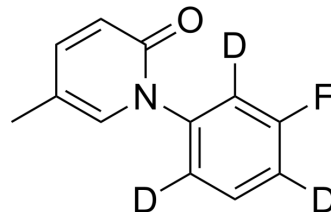


## Fluorofenidone-d<sub>3</sub>

<b>Cat. No.:</b>	HY-121246S
<b>Molecular Formula:</b>	C <sub>12</sub> H <sub>7</sub> D <sub>3</sub> FNO
<b>Molecular Weight:</b>	206.23
<b>Target:</b>	Isotope-Labeled Compounds; Akt; PI3K
<b>Pathway:</b>	Others; PI3K/Akt/mTOR
<b>Storage:</b>	Please store the product under the recommended conditions in the Certificate of Analysis.



### BIOLOGICAL ACTIVITY

<b>Description</b>	Fluorofenidone-d <sub>3</sub> is deuterium labeled Fluorofenidone. Fluorofenidone (AKF-PD), an analogue of AMR69, shows equivalent antifibrotic activity, lower toxicity and longer half-life. Fluorofenidone (AKF-PD) attenuates the progression of renal interstitial fibrosis partly by suppressing NADPH oxidase and extracellular matrix (ECM) deposition via the PI3K/Akt signalling pathway[1][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> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.

### REFERENCES

- [1]. Lou Q, et al. Design, synthesis and antifibrotic activities of carbohydrate-modified 1-(substituted aryl)-5-trifluoromethyl-2(1H) pyridones. *Molecules*. 2012 Jan 17;17(1):884-96.
- [2]. Qin J, et al. Fluorofenidone inhibits nicotinamide adenine dinucleotide phosphate oxidase via PI3K/Akt pathway in the pathogenesis of renal interstitial fibrosis. *Nephrology (Carlton)*. 2013 Oct;18(10):690-9.
- [3]. Russak EM, et al. Impact of Deuterium Substitution on the Pharmacokinetics of Pharmaceuticals. *Ann Pharmacother*. 2019;53(2):211-216.

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

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