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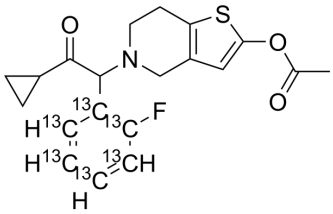
Prasugrel-$^{13}C_6$

Cat. No.:	HY-15284S3
CAS No.:	1261394-83-5
Molecular Formula:	$C_{14}{}^{13}C_6H_{20}FNO_3S$
Molecular Weight:	379.4
Target:	P2Y Receptor; Isotope-Labeled Compounds
Pathway:	GPCR/G Protein; Others
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Prasugrel-13C6 is a deuterated labeled Prasugrel[1]. Prasugrel (PCR 4099), a thienopyridine and proagent, inhibits platelet function. Prasugrel is an orally active and potent P2Y12 receptor antagonist, and inhibits ADP-induced platelet aggregation [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]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	In rat platelets, Prasugrel active metabolite inhibits in vitro platelet aggregation induced by adenosine ADP (10μM) with an IC_{50} value of 1.8 μM[3]. ?Prasugrel acts faster and is significantly more potent than Clopidogrel in vivo. Prasugrel is an inactive prodrug that requires metabolic processing in vivo to generate the active antiplatelet metabolite. Prasugrel is rapidly absorbed from the gut. After oral administration of standard-loading doses of 60 mg, maximum plasma levels of the active metabolite are achieved within 1 h, effective, maximum inhibition of platelet aggregation at 1-2 h[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Wijeyeratne YD, et al. Anti-platelet therapy: ADP receptor antagonists. Br J Clin Pharmacol. 2011 Oct;72(4):647-57.

[2]. Sugidachi A, et al. The greater in vivo antiplatelet effects of prasugrel as compared to clopidogrel reflect more efficient generation of its active metabolite with similar antiplatelet activity to that of clopidogrel's active metabolite. J Thromb Haemost. 2007 Jul;5(7):1545-51.

[3]. 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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