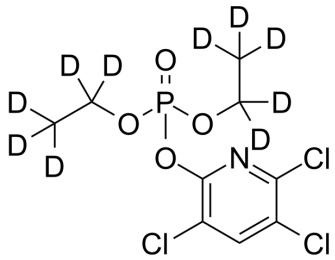
Chlorpyrifos-oxon-d10

Cat. No.:	HY-136610S
CAS No.:	1794779-85-3
Molecular Formula:	$C_9HD_{10}Cl_3NO_4P$
Molecular Weight:	344.58
Target:	AChE
Pathway:	Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Chlorpyrifos-oxon-d10 is the deuterium labeled Chlorpyrifos-oxon. Chlorpyrifos-oxon, an active metabolite of Chlorpyrifos, is a potent phosphorylating agent that potently inhibits AChE. Chlorpyrifos-oxon can induce cross-linking between subunits of tubulin and disrupt microtubule function[1][2][3][4].
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]. Florian Eyer, et al. Extreme variability in the formation of chlorpyrifos oxon (CPO) in patients poisoned by chlorpyrifos (CPF). Biochem Pharmacol. 2009 Sep 1;78(5):531-7.

[3]. Lawrence M Schopfer, et al. Chlorpyrifos oxon promotes tubulin aggregation via isopeptide cross-linking between diethoxyphospho-Lys and Glu or Asp: Implications for neurotoxicity. J Biol Chem. 2018 Aug 31;293(35):13566-13577.

[4]. Jie Gao, et al. Chlorpyrifos and chlorpyrifos oxon impair the transport of membrane bound organelles in rat cortical axons. Neurotoxicology. 2017 Sep;62:111-123.

[5]. Wei Jiang, et al. Mice treated with chlorpyrifos or chlorpyrifos oxon have organophosphorylated tubulin in the brain and disrupted microtubule structures, suggesting a role for tubulin in neurotoxicity associated with exposure to organophosphorus agents. Toxicol Sci. 2010 May;115(1):183-93.

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