Chlorpyrifos-oxon

Cat. No.: HY-136610

CAS No.: 5598-15-2

Molecular Formula: $C_9H_{11}Cl_3NO_4P$

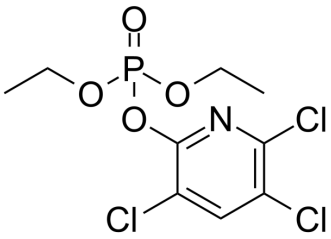
Molecular Weight: 334.52

Target: Cholinesterase (ChE)

Pathway: Neuronal Signaling

Storage: -20°C, stored under nitrogen

* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (298.94 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.9894 mL	14.9468 mL	29.8936 mL
	5 mM	0.5979 mL	2.9894 mL	5.9787 mL
	10 mM	0.2989 mL	1.4947 mL	2.9894 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (7.47 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.47 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.47 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

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

Treatment of tubulin with 1.5 mM Chlorpyrifos-oxon (CPO) leads to protein aggregation. However, even at 1.5 μM Chlorpyrifos-oxon cross-linked trimers are apparent. Chlorpyrifos-oxon promotes isopeptide bond cross-linking of tubulin monomers to make multimers[2].

In PC12 cells in culture, 24 hours of exposure to Chlorpyrifos at a concentration 10-fold below the concentration that inhibits AChE activity (3.0 μM) impaired neurite outgrowth while Chlorpyrifos-oxon inhibits neurite outgrowth at 1.0 nM[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo	Chlorpyrifos-oxon (CPO) is rapidly detoxified by human liver microsomes via CYP-dependent deethylation and dearylation, and by glutathione-S-transferase. In addition, reactions with A-esterases such as paraoxonase 1 (PON 1) or B-esterases such as carboxylesterase and butyrylcholinesterase (BChE) in the liver may rapidly degrade or scavenge Chlorpyrifos-oxon[1]. Chlorpyrifos-oxon (3 mg/kg, ip; once; wild-type mice) treatment shows the dimensions of microtubules from Chlorpyrifos-oxon-treated mice are about 60% of those from control mice. The microtubules from mice exposed to Chlorpyrifos-oxon have covalently modified amino acids and abnormal structure, suggesting disruption of microtubule function[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. 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.

[2]. 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.

[3]. 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.

[4]. 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.

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