Product Data Sheet

Product Name: McN3716 (Methyl palmoxirate)
Cat. No.: GC31427

Chemical Properties

Cas. No.	69207-52-9		
SMILES	O=C(C1(CCCCCCCCCCCCCC)OC1)OC		
Formula	$C_{18}H_{34}O_3$	M.Wt	298.46
Solubility	Soluble in DMSO	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure			

Protocol

Animal experiment:

Rats[1] Male Sprague Dawley rats are used. The rats receive ad libitum access to standard chow and water. At 15 weeks of age, six rats were subjected to either high-energy, head-focused microwave irradiation or CO2 asphyxiation. A separate group of 11 rats were implanted with a tail vein catheter (intravenous catheter 24 gauge/0.75 inch) and received either an intravenous injection of vehicle or 10 mg/kg of McN3716. Fifteen minutes after injection, rats were rapidly euthanized by high-energy, head-focused microwave irradiation (13.5 kW for 1.6 seconds) to avert ischemia for accurate quantification of in vivo basal levels of nonenzymatic auto-oxidative PUFA metabolites and enzymatically derived metabolites. Previously, we reported that this method reduced β-oxidation of fatty acid by 23% to 74%. McN3716 (Methyl palmoxirate, MEP) readily crosses the blood–brain barrier with a plasma half-life of 0.6 minute in the rat. The brain was excised and stored at -80°C for lipidomics profiling.

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References:

[1]. Chen CT, et al. Inhibiting mitochondrial β-oxidation selectively reduces levels of nonenzymatic oxidative polyunsaturated fatty acid metabolites in the brain. J Cereb Blood Flow Metab. 2014 Mar;34(3):376-9.

Background

McN3716 is a carnitine palmitoyltransferase I (CPT-1) inhibitor.

Inhibition of brain mitochondrial β-oxidation by McN3716 (Methyl palmoxirate, MEP) significantly reduces the levels of all measured HETE and epoxytrienoic acids (EET), nonenzymatic auto-oxidative metabolites of ARA, by 23% to 44% and 32% to 50% compared with vehicle-injected rats, respectively, except for 15-HETE which was unaffected. There is a significant 34% reduction in the level of 6-keto-PGF1α, a byproduct of PGI2 (prostacyclin) in McN3716-treated rats. Similarly, the brain level of hydroxyeicosapentaenoic acids, nonenzymatic auto-oxidative metabolites of EPA, is reduced by 35% to 76% upon McN3716 treatment relative to vehicle[1].

[1]. Chen CT, et al. Inhibiting mitochondrial β-oxidation selectively reduces levels of

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