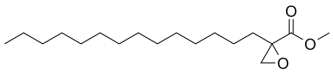
McN3716

Cat. No.:	HY-U00159
CAS No.:	69207-52-9
Molecular Formula:	$C_{18}H_{34}O_3$
Molecular Weight:	298.46
Target:	Mitochondrial Metabolism
Pathway:	Metabolic Enzyme/Protease
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



BIOLOGICAL ACTIVITY

Description	McN3716 is a carnitine palmitoyltransferase I (CPT-1) inhibitor.
IC_{50} & Target	Carnitine palmitoyltransferase I (CPT-1)[1]
In Vivo	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-$PGF_{1\alpha}$, a byproduct of PGI_2 (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]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [1]	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 CO_2 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. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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.

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