Etomoxir sodium salt

Cat. No.: HY-50202A

CAS No.: 828934-41-4

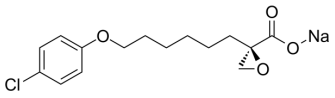
Molecular Formula: $C_{15}H_{18}ClNaO_4$

Molecular Weight: 320.74

Target: Apoptosis

Pathway: Apoptosis

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (311.78 mM; Need ultrasonic)
H_2O : 5 mg/mL (15.59 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.1178 mL	15.5890 mL	31.1779 mL
	5 mM	0.6236 mL	3.1178 mL	6.2356 mL
	10 mM	0.3118 mL	1.5589 mL	3.1178 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 3.33 mg/mL (10.38 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: 2.5 mg/mL (7.79 mM); Clear solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.79 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.79 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Etomoxir((R)-(+)-Etomoxir) sodium salt is an irreversible inhibitor of carnitine palmitoyltransferase 1a (CPT-1a), inhibits fatty acid oxidation (FAO) through CPT-1a and inhibits palmitate β-oxidation in human, rat and guinea pig[1].

IC_{50} & Target

CPT-1a[2]

In Vitro

Etomoxir mediates differential metabolic channeling of fatty acid and glycerol precursors into cardiolipin in H9c2 cells[2].
Etomoxir does not affect the activities of the cardiolipin biosynthetic and remodeling enzymes but causes a reduction in [1-^{14}C]palmitic acid or [1-^{14}C]oleic acid incorporation into cardiolipin[2].
Etomoxir increases [1,3-^{3}H]glycerol incorporation into cardiolipin. The mechanism is a 33% increase in glycerol kinase activity, which produces an increased glycerol flux through the de novo pathway of cardiolipin biosynthesis[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[2]

Cell Line:	Rat heart H9c2 myoblastic cells
Concentration:	1-80 μM
Incubation Time:	2 hours
Result:	Reduced the incorporation of [1-14C]fatty acids into CL and PtdGro in H9c2 cardiac myoblast cells but did not affect total incorporation of radioactivity into these cells.

In Vivo

Etomoxir significantly inhibits the decrease of bone mineral density (BMD) and bone breaking strength in db/db and high fat (HF)-fed mice and suppresses the reduction of BMSCs-differentiated osteoblasts[3].
Etomoxir inhibits the increase of mitochondrial ROS generation in db/db and HF-fed mice and osteoblasts[3].
Etomoxir-induced partial carnitine palmitoyltransferase-I (CPT-I) inhibition in vivo does not alter cardiac long-chain fatty acid uptake and oxidation rates[4]
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	80 male C57BLKS/J lar-Lepr$^{db/db}$ mice[3]
Dosage:	1 mg/kg
Administration:	Intraperitoneally injected; twice every week
Result:	Serum alkaline phosphatase was increased in db/db mice, which event was significantly suppressed by Etomoxir. Serum level of osteocalcin, a marker of bone formation, was reduced in db/db mice and Etomoxir markedly inhibited the reduction of osteocalcin. Serum tartrate-resistant acid phosphatase was elevated in db/db mice which phenomenon was significantly suppressed by Etomoxir.

Animal Model:	Rats[4]
Dosage:	20 mg/kg
Administration:	Injected daily; for 8 days
Result:	Etomoxir-treated rats displayed a 44% reduced cardiac CPT-I activity.

CUSTOMER VALIDATION

- Mol Cell. 2020 Oct 1;80(1):43-58.e7.
- Hepatology. 2022 Jan 14.
- Nat Commun. 2021 Jun 16;12(1):3660.
- Redox Biol. 2021 Jul 26;46:102082.

- Redox Biol. 2018 Oct;19:412-428.

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REFERENCES

[1]. Roddy S O'Connor, et al. The CPT1a inhibitor, etomoxir induces severe oxidative stress at commonly used concentrations.Sci Rep. 2018 Apr 19;8(1):6289.

[2]. Fred Y Xu,et al.Etomoxir mediates differential metabolic channeling of fatty acid and glycerol precursors into cardiolipin in H9c2 cells.J Lipid Res. 2003 Feb;44(2):415-23.

[3]. Jun Li, et al. FFA-ROS-P53-mediated mitochondrial apoptosis contributes to reduction of osteoblastogenesis and bone mass in type 2 diabetes mellitus.Sci Rep. 2015 Jul 31;5:12724.

[4]. Joost J F P Luiken, et al. Etomoxir-induced partial carnitine palmitoyltransferase-I (CPT-I) inhibition in vivo does not alter cardiac long-chain fatty acid uptake and oxidation rates.Biochem J. 2009 Apr 15;419(2):447-55.

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