

Product Data Sheet

Product Name: Etomoxir

Cat. No.: GC16736

Chemical Properties

Cas. No. 124083-20-1

Chemical Name ethyl (2R)-2-[6-(4-chlorophenoxy)hexyl]oxirane-2-carboxylate

SMILES CCOC(=O)C1(CO1)CCCCCOC2=CC=C(C=C2)Cl

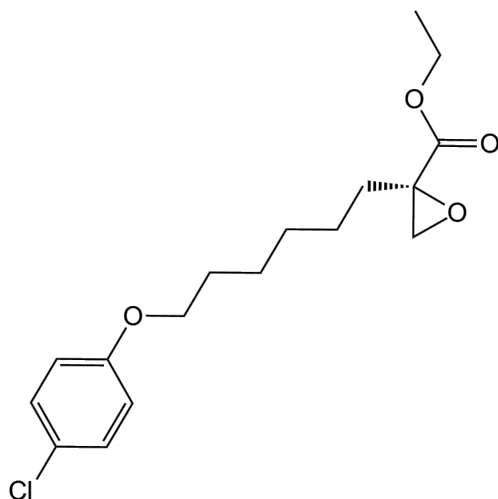
Formula $C_{17}H_{23}ClO_4$ M.Wt 326.82

Solubility ≥ 32.7 mg/mL in DMSO, ≥ 109.6 mg/mL in EtOH Storage Store at -20°C , protect from light, stored under nitrogen

General tips For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C 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

Cell experiment [1]:

Caution: Product has not been fully validated for medical applications. For research use only.

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Cell lines Rat heart H9c2 myoblastic cells

Preparation Method H9c2 cells were preincubated with 1–80µM Etomoxir for 2h and incubated with 0.1mM [1-¹⁴C]oleic acid bound to albumin for 2h, and radioactivity incorporated into cardiolipin (CL) determined.

Reaction
Conditions 1-80µM; 2h

Applications In rat cardiac H9c2 myoblasts, Etomoxir reduced [1-¹⁴C]oleic acid incorporation into phosphatidylglycerol (PtdGro) and cardiolipin (CL) in a concentration-dependent manner and increased [1,3-³H]glycerol incorporation into CL.

Animal experiment [2]:

Animal models Experimental autoimmune encephalomyelitis (EAE) mice model

Preparation Method EAE was induced with a peptide from myelin oligodendrocyte glycoprotein (MOG_{35–55}) and mice were administered Etomoxir.

Dosage form 15mg/kg, On days 8 and 15 after EAE induction, i.p.

Applications Etomoxir treatment reduced inflammatory responses and immune cell infiltration in the central nervous system of EAE mice.

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References:

- [1] Xu FY1, Taylor WA, Hurd JA, 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. Epub 2002 Nov 4.
- [2] Shriver LP1, Manchester M. Inhibition of fatty acid metabolism ameliorates disease activity in an animal model of multiple sclerosis. Sci Rep. 2011;1:79.

Background

Etomoxir is an irreversible inhibitor of carnitine palmitoyltransferase 1a (CPT-1a). Etomoxir inhibits fatty acid oxidation (FAO) and palmitate oxidation via CPT-1a^[1].

T cells cultured in a low-glucose medium in the presence of Etomoxir (100µM) significantly reduce interferon-gamma (IFN-γ) production and do not promote T-helper 2 (Th2)-related cytokine IL-4 production, while Etomoxir does not affect T cells cultured in the high-glucose medium^[2]. Etomoxir (500µM) can significantly inhibit FAO in A549 and A549T cells. Etomoxir at 500µM or 1mM can significantly increase the sensitivity of paclitaxel-resistant cells (A549T) to paclitaxel^[3].

In experimental autoimmune encephalomyelitis (EAE) mice, Etomoxir (15mg/kg) reduces inflammatory responses, immune cell infiltration in the central nervous system (CNS), as well as inflammation and demyelination in the CNS^[2]. In C57BLKS/J lar-Lepr^{db/db} and high-fat (HF)-fed mice, Etomoxir (1mg/kg) significantly reduces the decrease in bone mineral density (BMD) and bone-breaking strength and inhibits the decrease in osteoblasts differentiated from bone marrow stromal cells (BMSCs)^[4].

References:

- [1] Rupp H, et al. The use of partial fatty acid oxidation inhibitors for metabolic therapy of angina pectoris and heart failure. Herz. 2002 Nov;27(7):621-36.
- [2] Shriver L P, Manchester M. Inhibition of fatty acid metabolism ameliorates disease activity in an animal model of multiple sclerosis[J]. Scientific Reports, 2011, 1(1): 79.
- [3] Li J, Zhao S, Zhou X, et al. Inhibition of lipolysis by mercaptoacetate and etomoxir specifically sensitizes drug-resistant lung adenocarcinoma cells to paclitaxel[J]. PloS one, 2013, 8(9): e74623.
- [4] Li J, He W, Liao B, et al. FFA-ROS-P53-mediated mitochondrial apoptosis contributes to the

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reduction of osteoblastogenesis and bone mass in type 2 diabetes mellitus[J]. Scientific Reports, 2015, 5(1): 12724.

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