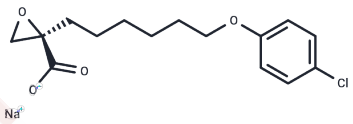
Data Sheet (Cat.No.T4535)

TargetMol

Etomoxir sodium salt

Chemical Properties



CAS No. : 828934-41-4

Formula: C15H18ClO4.Na

Molecular Weight: 320.74

Storage: Store at low temperature
Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	Etomoxir sodium salt ((R)-Etomoxir sodium salt) is a carnitine palmitoyltransferase 1a (CPT-1a) inhibitor that inhibits fatty acid oxidation (FAO). Etomoxir sodium salt has antitumor activity.
Targets(IC50)	Apoptosis
In vitro	**METHODS**: BCa cell lines UM-UC-3 and T24 were treated with Etomoxir sodium salt (20-200 μM) for 24-72 h. Cell viability was examined using MTT. **RESULTS**: Etomoxir inhibited the viability of UM-UC-3 and T24 cells in a dose-dependent manner. [1] **METHODS**: Human breast cancer cells MCF-7 and T47D were treated with Etomoxir sodium salt (0.1-50 μM) for 24 h. Cellular FAO activity was measured by 3H palmitic acid. **RESULTS**: Low micromolar concentrations of Etomoxir were sufficient to achieve maximal inhibition of FAO in MCF-7 and T47D cells. [2]
In vivo	**METHODS**: To test the antitumor activity in vivo, Etomoxir sodium salt (40 mg/kg) was intraperitoneally injected into BALB/c nude mice carrying human bladder cancer tumor T24 every two days for twenty days. **RESULTS**: Etomoxir significantly inhibited tumor growth. [1] **METHODS**: To test the activity against multiple sclerosis, Etomoxir sodium salt (15 mg/kg) was intraperitoneally injected into a C57BL/6J mouse model of EAE induction once a week or every two days for two weeks. **RESULTS**: Etomoxir-treated mice showed reduced disease severity as well as reduced inflammation and demyelination. Disruption of fatty acid metabolism promotes downregulation of CNS inflammation and this metabolic pathway is a potential therapeutic target for multiple sclerosis. [3]

Solubility Information

Solubility	DMSO: 3.21 mg/mL (10.01 mM),Sonication is recommended. H2O: 5 mg/mL (15.59 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	Saline: 3.5 mg/mL (10.91 mM),Solution. *Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and*

In vivo Formulation	*used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.*

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	3.1178 mL	15.589 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
50 mM	0.0624 mL	0.3118 mL	0.6236 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Cheng S, et al. Fatty acid oxidation inhibitor etomoxir suppresses tumor progression and induces cell cycle arrest via PPARγ-mediated pathway in bladder cancer. Clin Sci (Lond). 2019 Aug 7;133(15):1745-1758.
Huang D, Chowdhury S, Wang H, et al. Multiomic analysis identifies CPT1A as a potential therapeutic target in platinum-refractory, high-grade serous ovarian cancer. Cell Reports Medicine. 2021, 2(12): 100471.
Li M, Yang J, Ye C, et al. Integrated Metabolomics and Transcriptomics Analyses Reveal Metabolic Landscape in Neuronal Cells during JEV Infection. Virologica Sinica. 2021: 1-12.
Ma Y, et al. Functional analysis of molecular and pharmacological modulators of mitochondrial fatty acid oxidation. Sci Rep. 2020 Jan 29;10(1):1450.
Shriver LP,et al. Inhibition of fatty acid metabolism ameliorates disease activity in an animal model of multiple sclerosis. Sci Rep. 2011;1:79.
Luiken JJ, 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.