



## **Etomoxir**

**Catalog No: tcsc0325** 

**Product Description** 

Available Sizes
Size: 5mg
Size: 10mg
Size: 50mg
Specifications
CAS No: 124083-20-1
Formula: C <sub>17</sub> H <sub>23</sub> CIO <sub>4</sub>
Pathway: Others
Target: Others
Purity / Grade: >98%
<b>Solubility:</b> DMSO : ≥ 50 mg/mL (152.99 mM)
Alternative Names: (R)-(+)-Etomoxir
Observed Molecular Weight: 326.82





Etomoxir ((R)-(+)-Etomoxir) is a potent inhibitor of **carnitine palmitoyltransferase-I** (**CPT-1**).

IC50 & Target: CPT-1<sup>[1]</sup>

In Vitro: Etomoxir binds irreversibly to the catalytic site of CPT-1 inhibiting its activity, but also upregulates fatty acid oxidation enzymes. Etomoxir is developed as an inhibitor of the mitochondrial carnitine palmitoyltransferase-1 (CPT-1) located on the outer mitochondrial membrane. Etomoxir, in the liver can act as peroxisomal proliferator, increasing DNA synthesis and liver growth. Thus, etomoxir, in addition of being a CPT1 inhibitor could be considered as a PPARalpha agonist<sup>[1]</sup>. Etomoxir is a member of the oxirane carboxylic acid carnitine palmitoyl transferase I inhibitors and has been suggested as a therapeutic agent for the treatment of heart failure. Acute Etomoxir treatment irreversibly inhibits the activity of carnitine palmitoyltransferase I. As a result, fatty acid import into the mitochondria and  $\beta$ -oxidation is reduced, whereas cytosolic fatty acid accumulates and glucose oxidation is elevated. Prolonged incubation (24 h) with Etomoxir produces diverse effects on the expression of several metabolic enzyme<sup>[2]</sup>.

In Vivo: Etomoxir is an inhibitor of free fatty acid (FFA) oxidation-related key enzyme CPT1. P53 interacts directly with Bax, which is inhibited by Etomoxir, further confirming the direct interaction of P53 and Bax, and the involvement of FAO-mediated mitochondrial ROS generation in db/db mice<sup>[3]</sup>. Rats are injected daily with Etomoxir, a specific CPT-I inhibitor, for 8 days at 20 mg/kg of body mass. Etomoxir-treated rats display a 44% reduced cardiac CPT-I activity. The treatment of Lewis rats for 8 days with 20 mg/kg Etomoxir does not alter blood glucose, which is in line with comparable etomoxir-feeding studies. Similarly, Etomoxir feeding does not affect general growth characteristics such as gain in body mass, nor does it affect hindlimb muscle mass. However, heart mass and liver mass are both significantly increased by 11% in Etomoxir-treated rats<sup>[4]</sup>.

All products are for RESEARCH USE ONLY. Not for diagnostic & therapeutic purposes!