Teglicar

Cat. No.: HY-16482

CAS No.: 250694-07-6

Molecular Formula: $C_{22}H_{45}N_3O_3$

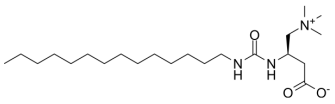
Molecular Weight: 399.61

Target: Endogenous Metabolite

Pathway: Metabolic Enzyme/Protease

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

Ethanol : 100 mg/mL (250.24 mM; ultrasonic and heat to 60°C)

DMSO : 19 mg/mL (47.55 mM; Need ultrasonic)

H_2O : 10 mg/mL (25.02 mM; Need ultrasonic)

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	2.5024 mL	12.5122 mL	25.0244 mL
5 mM	0.5005 mL	2.5024 mL	5.0049 mL
10 mM	0.2502 mL	1.2512 mL	2.5024 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 3.33 mg/mL (8.33 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1.9 mg/mL (4.75 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Teglicar is a selective and reversible orally active liver isoform of carnitine palmitoyl-transferase 1 (L-CPT1) inhibitor with an IC_{50} value of 0.68 μM and a K_i value of 0.36 μM. Teglicar has a potential antihyperglycemic propert. Teglicar can be used for the research of diabetes and neurodegenerative disease including Huntington's disease (HD)[1][2].

IC_{50} & Target

IC50: 0.68 μM (L-CPT1); Ki: 0.36 μM (L-CPT1)[1]

In Vitro

Teglicar has L-CPT1 inhibitory activity with an IC_{50} value of 0.68 μM and a K_i value of 0.36 μM[1].

Teglicar (10 μM; 2 h) induces a concentration-dependent reduction of ketone bodies and glucose production[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Teglicar (oral, 80 mg/kg, once a day, for 30 days or infusion, 5.3 mg/kg/h, for 3 h) reduces the endogenous glucose production (262%) without affecting peripheral glucose utilization in SD rats[1].
Teglicar (gavage, 50 mg/kg, single or long-term 100 mg/kg/day for 30 days) not affects heart 2-[^{3}H]deoxyglucose uptake in C57BL6/J mice[1].
Teglicar (gavage, 50 mg/kg, twice a day, for 45 days) reduces postabsorptive glycemia (238%), water consumption (231%), and fructosamine (230%) in db/db mice[1].
Teglicar (30 mg/kg, twice a day, for 26 days) normalized glycemia (219%) and insulinemia (253%) and increases HTGC but not affects liver and peripheral insulin sensitivity in high-fat diet C57BL/6J mice[1].
Teglicar (oral, 50 μM, was added to the surface of fly food, 1, 8, 12, and 15 days) ameliorates the neurodegenerative phenotype in a drosophila Huntington's Disease Model by acting on the expression of carnitine-related genes[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	SD rats[1]
Dosage:	80 mg/kg, 5.3 mg/kg
Administration:	oral, 80 mg/kg, once a day, for 30 days or infusion, 5.3 mg/kg/h, for 3 h
Result:	Reduced basal insulin levels, showed a higher triglyceride and low glycogen content in the liver, without any change in liver weight. Showed a rapid drop in glycemia, suppressed EGP (EGP2) diminished by 62% and not affected peripheral glucose utilization (GU).

Animal Model:	C57BL6/J mice[1]
Dosage:	50 mg/kg, 100 mg/kg
Administration:	gavage, 50 mg/kg, single or long-term 100 mg/kg/day for 30 days.
Result:	Did not modify etomoxir-induced M-CPT1 inhibition and failed to determine significant changes in 2-DG heart uptake, heart weights, and triglyceride content.

Animal Model:	db/db mice[1]
Dosage:	50 mg/kg
Administration:	gavage, 50 mg/kg, twice a day, for 45 days
Result:	Induced a significant reduction of postabsorptive serum glucose, reduced serum fructosamine and average daily water consumption, increased Serum FFAs, but did not change insulin levels, triglycerides, alanine aminotransferase, also induced a significant reduction of glucose AUC during ITT. Did not induce any variation in the content of PPAR-α and its target gene product MCAD and peroxisomal b-oxidation in liver and heart of db/db mice.

Animal Model:	High-fat diet C57BL/6J mice[1]
Dosage:	30 mg/kg
Administration:	30 mg/kg, twice a day, for 26 days

Result:	Did not affect food intake, did not change body weight and serum FFAs and triglycerides and did not affect glucose intolerant.

REFERENCES

[1]. Roberto Conti, et al. Selective reversible inhibition of liver carnitine palmitoyl-transferase 1 by teglicar reduces gluconeogenesis and improves glucose homeostasis. Diabetes. 2011 Feb;60(2):644-51.

[2]. Carla Bertapelle, et al. The Reversible Carnitine Palmitoyltransferase 1 Inhibitor (Teglicar) Ameliorates the Neurodegenerative Phenotype in a Drosophila Huntington's Disease Model by Acting on the Expression of Carnitine-Related Genes. Molecules

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