(±)-Carnitine chloride

Cat. No.: HY-B1453

CAS No.: 461-05-2

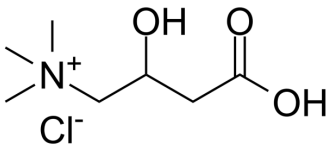
Molecular Formula: $C_7H_{16}ClNO_3$

Molecular Weight: 197.66

Target: Reactive Oxygen Species

Pathway: Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : ≥ 100 mg/mL (505.92 mM)
DMSO : 25 mg/mL (126.48 mM; Need ultrasonic)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	5.0592 mL	25.2960 mL	50.5919 mL
5 mM	1.0118 mL	5.0592 mL	10.1184 mL
10 mM	0.5059 mL	2.5296 mL	5.0592 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 150 mg/mL (758.88 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 (12.65 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (12.65 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (12.65 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

(±)-Carnitine chloride exists in two isomers, known as D and L. L-carnitine plays an essential role in the β-oxidation of fatty acids and also shows antioxidant, and anti-inflammatory activities.

In Vitro

The main role of L-carnitine is to shuttle long-chain fatty acids across the inner mitochondrial membrane. After L-carnitine and acyl-CoA become acyl-carnitine by activation of carnitine palmitoyl transferase (CPT)-I, the transported acyl-carnitine is

	changed into acyl-CoA by CPT-II in the mitochondria matrix. Palmitoyl-CoA-induced mitochondrial respiration is increased by L-carnitine treatment, and then is accelerated by the presence of ADP. This acceleration is induced by treatment with L-carnitine in a concentration-dependent manner, and is saturated at 5 mM L-carnitine[1]. Pretreatment with L-carnitine augments Nrf2 nuclear translocation, DNA binding activity and heme oxygenase-1 (HO-1) expression in H_2O_2-treated HL7702 cells. L-carnitine protects HL7702 cells against H_2O_2-induced cell damage through Akt-mediated activation of Nrf2 signaling pathway[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	L-carnitine is found to down-regulate the ubiquitin proteasome pathway and increase IGF-1 concentrations in animal models. L-carnitine administration for 2 weeks of hindlimb suspension alleviates the decrease in weight and fiber size in the soleus muscle. In addition, L-carnitine suppresses atrogin-1 mRNA expression, which has been reported to play a pivotal role in muscle atrophy[3]. Simultaneous treatment with L-carnitine attenuates the renal fibrosis (which correlated with a reduction of plasma TGF-β1 levels) and the pro-oxidative and proinflammatory status reported in L-NAME groups, with a concomitant increase in the expression of PPAR-γ[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [1]	Mitochondria (0.6 mg protein/mL) are incubated in 2.5 mM Hepes (pH7.4) containing 225 mM mannitol, 75 mM sucrose and 100 μM ethylene glycol tetraacetic acid (EGTA) with or without 5 mM L-carnitine at 25°C. To measure oxygen uptake, 10 min after inorganic phosphate (Pi) 4 mM are added, the mitochondria are treated with palmitoyl-CoA (50 μM) and then ADP is added (200 μM). Oligomycin (5 μM) and rotenone (10 μM) are added 3-4 min after the ADP treatment. HPG (0-10 mM), which can specifically inhibit carnitine palmitoyl transferase (CPT)-I activity in the mitochondria, is added in the Hepes medium before incubation of the mitochondria[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Rats: After 1 week of acclimatization, rats are randomly assigned to a hindlimb suspension group, hindlimb suspension with L-carnitine administration group, and a pair-fed group. The L-carnitine group are administered a 1250 mg L-carnitine/kg dissolved in distilled water orally using a sonde. The body weight is measured every morning at 09:00 and L-carnitine solution is ingested every morning at 10:00. The experiment is conducted for 14 days[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Toxicol Appl Pharmacol. 2019 Apr 1;368:18-25.

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REFERENCES

[1]. Oyanagi E, et al. Protective action of L-carnitine on cardiac mitochondrial function and structure against fatty acidstress. Biochem Biophys Res Commun. 2011 Aug 19;412(1):61-7.

[2]. Li J, et al. l-carnitine protects human hepatocytes from oxidative stress-induced toxicity through Akt-mediated activation of Nrf2 signaling pathway. Can J Physiol Pharmacol. 2016 May;94(5):517-25.

[3]. Jang J, et al. l-Carnitine supplement reduces skeletal muscle atrophy induced by prolonged hindlimb suspension in rats. Appl Physiol Nutr Metab. 2016 Dec;41(12):1240-1247.

[4]. Zambrano S, et al. L-carnitine attenuates the development of kidney fibrosis in hypertensive rats by upregulating PPAR-γ. Am J Hypertens. 2014 Mar;27(3):460-70.

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