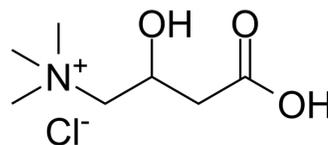


(±)-Carnitine chloride

Cat. No.:	HY-B1453
CAS No.:	461-05-2
Molecular Formula:	C ₇ H 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

H₂O : ≥ 100 mg/mL (505.92 mM)
 DMSO : 25 mg/mL (126.48 mM; Need ultrasonic)
 * "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent Concentration	Mass		
		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

- Add each solvent one by one: PBS
Solubility: 150 mg/mL (758.88 mM); Clear solution; Need ultrasonic
- 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
- 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
- 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 H₂O₂-treated HL7702 cells. L-carnitine protects HL7702 cells against H₂O₂-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].

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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].

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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 acid stress. *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.

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

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