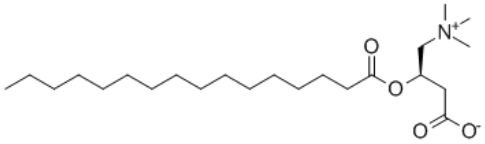
Product Data Sheet

Product Name: L-Palmitoylcarnitine
Cat. No.: GC30745

Chemical Properties

Cas. No.	2364-67-2		
SMILES	CCCCCCCCCCCCCCCC(O[C@H](CC([O-])=O)C[N+](C)(C)C)=O		
Formula	$C_{23}H_{45}NO_4$	M.Wt	399.61
Solubility	Ethanol : 25 mg/mL (Need ultrasound)	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure			



Protocol

Kinase experiment [1]:

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Preparation Method	The enzymatic reactions were conducted in 50mM Tris–HCl buffer (pH 7.8) at 37 °C. Initially, the proteases, plasmin (30nM) and tPA (20nM), and serial dilutions of L-Palmitoylcarnitine (6.25–100μM) were incubated in buffer at 37°C for 5min, followed by the addition of their corresponding chromogenic substrates, CS-41 (0.2mM) for plasmin and CS-05 (0.2mM) for tPA, to initiate the reaction. The rate of substrate hydrolysis was monitored continuously at 405nm.
Reaction Conditions	6.25–100μM; 5min
Applications	L-Palmitoylcarnitine enhanced the abilities of plasmin and tPA to hydrolyze their chromogenic substrates.L-Palmitoylcarnitine could directly bind to two enzymes (plasmin and tPA) that are responsible for thrombolysis and fibrin(ogen)olysis. Through high-affinity interactions with plasmin (K_D=6.47nM) and tPA (K_D=4.46nM).

Cell experiment [2]:

Cell lines	Guinea-pig ventricular myocytes
Preparation Method	Guinea-pig ventricular myocytes were incubated with L-Palmitoylcarnitine (1–10μM) at 35°C for 10min.
Reaction Conditions	1–10μM; 10min
Applications	L-Palmitoylcarnitine largely inhibited the outward current transient associated with Na/K pump current.

Animal experiment [3]:

Animal models	Male C57BL/6 mice
Preparation Method	$FeCl_3$-induced mouse thrombosis model and transient middle cerebral artery occlusion (tMCAO) mouse models were treated with L-Palmitoylcarnitine (0, 0.25, 1, 4mg/kg) administered via tail vein injections.
Dosage form	0, 0.25, 1, 4mg/kg; i.v.; a single administration
Applications	L-Palmitoylcarnitine administration significantly inhibited $FeCl_3$-induced arterial thrombosis. It also mitigated intracerebral thrombosis and inflammation in a transient middle cerebral artery occlusion (tMCAO) mouse model.

References:
[1] Yang J, Cha L, Wang Y P, et al. L-Palmitoylcarnitine potentiates plasmin and tPA to inhibit thrombosis. Nat Prod Bioprospect. 2023 Nov 8;13(1):48.
[2] Tanaka M, Gilbert J, Pappano A J. Inhibition of sodium pump by l-palmitoylcarnitine in single guinea-pig ventricular myocytes. J Mol Cell Cardiol. 1992 Jul;24(7):711-9.
[3] Yang J, Cha L, Wang Y P, et al. L-Palmitoylcarnitine potentiates plasmin and tPA to inhibit thrombosis. Nat Prod Bioprospect. 2023 Nov 8;13(1):48.

Background

L-Palmitoylcarnitine is an important endogenous fatty acid metabolite that simultaneously enhanced the ATP sensitivity of the ion channel with IC_{50} value fell from 62.0 ± 2.7 to 30.3 ±5.5μM [1]. L-Palmitoylcarnitine is a critical regulator in fatty acids metabolism, especially in mitochondrial fatty acid β-oxidation[2]. L-Palmitoylcarnitine weakly inhibits pyruvate-supported respiration in mitochondria but does not affect pyruvate utilization. It has stronger inhibitory effect on acetylcarnitine oxidation[3].

In vitro, L-Palmitoylcarnitine (1–10μM) incubated guinea-pig ventricular myocytes at 35°C for 10min largely inhibited the outward current transient associated with Na/K pump current[4]. L-

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Palmitoylcarnitine (10nM to10μM) incubated isolated guinea-pig ventricular myocytes for 40min produced concentration- and time-dependent changes of resting potential (RP) and action potential duration at 50% repolarization (APD_{50})[5].

In vivo, L-Palmitoylcarnitine (0, 0.25, 1, 4mg/kg) administrated into $FeCl_3$-induced mouse thrombosis model and transient middle cerebral artery occlusion (tMCAO) mouse models via tail vein injections significantly inhibited $FeCl_3$-induced arterial thrombosis, and also mitigated intracerebral thrombosis and inflammation in a transient middle cerebral artery occlusion (tMCAO) mouse model[6].

References:

[1] Haruna T, Horie M,Takano M,et al. Alteration of the membrane lipid environment by L-palmitoylcarnitine modulates K(ATP) channels in guinea-pig ventricular myocytes. Pflugers Arch. 2000;441(2-3):200-207.
[2] Liu Q Y, Li P W, Ma J L, et al. Arsenic exposure at environmentally relevant levels induced metabolic toxicity in development mice: Mechanistic insights from integrated transcriptome and metabolome. Environ Int. 2024 Aug:190:108819.
[3] Hutson S M, van Dop C, Lardy H A. Metabolism of pyruvate and carnitine esters in bovine epididymal sperm mitochondria. Arch Biochem Biophys. 1977 May;181(1):345-52.
[4] Tanaka M, Gilbert J, Pappano A J. Inhibition of sodium pump by l-palmitoylcarnitine in single guinea-pig ventricular myocytes. J Mol Cell Cardiol. 1992 Jul;24(7):711-9.
[5] Mészàros J, Pappano A J. Electrophysiological effects of L-palmitoylcarnitine in single ventricular myocytes. Am J Physiol. 1990 Apr;258(4 Pt 2):H931-8.
[6] Yang J, Cha L, Wang Y P, et al. L-Palmitoylcarnitine potentiates plasmin and tPA to inhibit thrombosis. Nat Prod Bioprospect. 2023 Nov 8;13(1):48.

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