Product Data Sheet

Product Name: (±)-Myristoylcarnitine chloride
Cat. No.: GC10056

Chemical Properties

Cas. No.	14919-38-1		
Chemical Name	(R)-3-carboxy-N,N,N-trimethyl-2-(tetradecanoyloxy)propan-1-aminium chloride		
SMILES	O=C(CCCCCCCCCCCCC)O[C@H](CC(O)=O)C[N+](C)(C)C.[Cl-]		
Formula	$C_{21}H_{42}ClNO_4$	M.Wt	408.02
Solubility	<10.2mg/ml in Water	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ 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	(±)-Myristoylcarnitine chloride		

Background

(±)-Myristoylcarnitine chloride is an agonist for cholinergic and a homolog of acetylcarnitine chloride.

Acetylcholine receptor (AChR) is an integral membrane protein receptor for acetylcholine. There are two kinds of AChRs: nicotinic acetylcholine receptors and muscarinic acetylcholine receptors.

(±)-Myristoylcarnitine chloride is a cholinergic agonist and an intermediate in lipid metabolism [1]. In retinal ganglion cells, acetylcarnitine and acetylcholine inhibited GABAergic responses to exogenous GABA and GABAergic inhibitory postsynaptic currents [2].

In dogs with coronary ligation, (-)-carnitine chloride (LCC) (300 mg/kg) and acetyl (-)-carnitine chloride (ALCC) (300 mg/kg) inhibited the ventricular arrhythmia. Also, LCC and ALCC improved oxidative phosphorylation rate and the mitochondrial function [1]. In the

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mouse hot plate test, acetyl-l-carnitine (ALCAR) (100 mg/kg) exhibited analgesia. While, U-73122 and neomycin (the phospholipase C (PLC) inhibitors) blocked the increase of the pain threshold induced by ALCAR. LiCl that impairing phosphatidylinositol synthesis antagonized the antinociception in a dose-dependent way. PMA and PDBu (PKC activators) blocked the increase of the pain threshold in a dose-dependent way. These results suggested that ALCAR analgesia required the participation of the PLC-IP3 pathway [3].

References:

[1]. Imai S, Matsui K, Nakazawa M, et al. Anti-arrhythmic effects of (-)-carnitine chloride and its acetyl analogue on canine late ventricular arrhythmia induced by ligation of the coronary artery as related to improvement of mitochondrial function. Br J Pharmacol, 1984, 82(2): 533-542.

[2]. B?hring R, Standhardt H, Martelli EA, et al. GABA-activated chloride currents of postnatal mouse retinal ganglion cells are blocked by acetylcholine and acetylcarnitine: how specific are ion channels in immature neurons? Eur J Neurosci, 1994, 6(7): 1089-1099.

[3]. Galeotti N, Bartolini A, Calvani M, et al. Acetyl-L-carnitine requires phospholipase C-IP3 pathway activation to induce antinociception. Neuropharmacology, 2004, 47(2): 286-294.