Acetyl Coenzyme A trisodium

Cat. No.: HY-113596

CAS No.: 102029-73-2

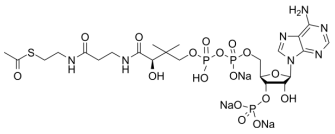
Molecular Formula: $C_{23}H_{35}N_7Na_3O_{17}P_3S$

Molecular Weight: 875.52

Target: Endogenous Metabolite; Autophagy; Oxidative Phosphorylation

Pathway: Metabolic Enzyme/Protease; Autophagy

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 : 83.33 mg/mL (95.18 mM; Need ultrasonic)
DMSO : < 1 mg/mL (insoluble or slightly soluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.1422 mL	5.7109 mL	11.4218 mL
5 mM	0.2284 mL	1.1422 mL	2.2844 mL
10 mM	0.1142 mL	0.5711 mL	1.1422 mL

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

In Vivo

1. Add each solvent one by one: PBS
Solubility: 100 mg/mL (114.22 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Acetyl-coenzyme A (Acetyl-CoA) trisodium is a membrane-impermeant central metabolic intermediate, participates in the TCA cycle and oxidative phosphorylation metabolism. Acetyl-coenzyme A trisodium, regulates various cellular mechanisms by providing (sole donor) acetyl groups to target amino acid residues for post-translational acetylation reactions of proteins. Acetyl Coenzyme A trisodium is also a key precursor of lipid synthesis[1][2][3][4].

IC_{50} & Target

Human Endogenous Metabolite

In Vitro

Acetyl coenzyme A trisodium increases cytoplasmic protein acetylation in starved U2OS cells while reducing starvation-induced autophagic fluxes. (U2OS cells stably expressing GFP-LC3 and are microinjected with Acetyl coenzyme A trisodium; incubated in nutrient-free conditions in the presence of 100 nM BafA1 and fixed after 3 h)[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Acetyl coenzyme A trisodium blunts pressure overload-induced cardiomyopathy in a mice cardiac pressure overload model

by Suppressing maladaptive autophagy[2][3].?Mice deprived of food (but with access to water ad libitum) for 24 h exhibit a significant reduction in total Acetyl coenzyme A trisodium levels in several organs, including the heart and muscles, corresponding to a decrease in protein acetylation levels. However, the same experimental conditions have no major effects on Acetyl coenzyme A trisodium concentrations in the brain and actually increase hepatic Acetyl coenzyme A trisodium and protein acetylation levels[4].

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

CUSTOMER VALIDATION

- J Cell Physiol. 2023 Feb 6.

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REFERENCES

[1]. Choudhary C, et al. The growing landscape of lysine acetylation links metabolism and cell signalling. Nat Rev Mol Cell Biol. 2014 Aug;15(8):536-50.

[2]. Mariño G, et al. Regulation of autophagy by cytosolic acetyl-coenzyme A. Mol Cell. 2014 Mar 6;53(5):710-25.

[3]. Zhu H, et al. Cardiac autophagy is a maladaptive response to hemodynamic stress. J Clin Invest. 2007 Jul;117(7):1782-93.

[4]. Pietrocola F, et al. Acetyl coenzyme A: a central metabolite and second messenger. Cell Metab. 2015 Jun 2;21(6):805-21.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA