Acetyl Coenzyme A trisodium

Cat. No.: HY-113596

CAS No.: 102029-73-2

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

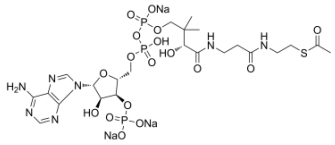
Molecular Weight: 875.52

Target: Endogenous Metabolite

Pathway: Metabolic Enzyme/Protease

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

H_2O : 125 mg/mL (142.77 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.

BIOLOGICAL ACTIVITY

Description

Acetyl Coenzyme A trisodium (Acetyl-CoA trisodium) is a central metabolic intermediate. Acetyl Coenzyme A trisodium is the actual molecule through which glycolytic pyruvate enters the tricarboxylic acid (TCA) cycle, is a key precursor of lipid synthesis, and is the sole donor of the acetyl groups for acetylation. Acetyl Coenzyme A trisodium acts as a potent allosteric activator of pyruvate carboxylase (PC)[1].

In Vitro

Acetyl-coenzyme A (Acetyl-CoA) is a membrane-impermeant molecule constituted by an acetyl moiety (CH3CO) linked to coenzyme A (CoA), a derivative of vitamin B5 and cysteine, through a thioester bond. As thioester bonds are energy rich, the chemical structure of acetyl-CoA facilitates the transfer of the acetyl moiety to a variety of acceptor molecules, including amino groups on proteins[1].

In most mammalian cells, Acetyl-coenzyme A (Acetyl-CoA) is predominantly generated in the mitochondrial matrix by various metabolic circuitries, namely glycolysis, β-oxidation, and the catabolism of branched amino acids. Cytosolic Acetyl-coenzyme A is the precursor of multiple anabolic reactions that underlie the synthesis of fatty acids and steroids, as well as specific amino acids including glutamate, proline, and arginine[1].

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

In Vivo	Mice deprived of food (but with access to water ad libitum) for 24 hr exhibit a significant reduction in total Acetyl-coenzyme A (Acetyl-CoA) 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 concentrations in the brain and actually increase hepatic Acetyl-coenzyme A and protein acetylation levels. Ethanol intake augments Acetyl-coenzyme A levels in hepatic mitochondria[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

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

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