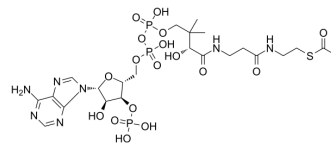


## Acetyl coenzyme A

<b>Cat. No.:</b>	HY-114293
<b>CAS No.:</b>	72-89-9
<b>Molecular Formula:</b>	C <sub>23</sub> H <sub>38</sub> N <sub>7</sub> O <sub>17</sub> P <sub>3</sub> S
<b>Molecular Weight:</b>	809.57
<b>Target:</b>	Endogenous Metabolite; Autophagy; Oxidative Phosphorylation
<b>Pathway:</b>	Metabolic Enzyme/Protease; Autophagy
<b>Storage:</b>	Please store the product under the recommended conditions in the Certificate of Analysis.



### BIOLOGICAL ACTIVITY

<b>Description</b>	Acetyl-coenzyme A (Acetyl-CoA) is a membrane-impermeant central metabolic intermediate, participates in the TCA cycle and oxidative phosphorylation metabolism. Acetyl-coenzyme A, 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 is also a key precursor of lipid synthesis <sup>[1][2][3][4]</sup> .
<b>IC<sub>50</sub> &amp; Target</b>	Human Endogenous Metabolite
<b>In Vitro</b>	Acetyl coenzyme A 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; incubated in nutrient-free conditions in the presence of 100 nM BafA1 and fixed after 3 h) <sup>[2]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.
<b>In Vivo</b>	Acetyl coenzyme A blunts pressure overload-induced cardiomyopathy in a mice cardiac pressure overload model by Suppressing maladaptive autophagy <sup>[2][3]</sup> . Mice deprived of food (but with access to water ad libitum) for 24 h exhibit a significant reduction in total Acetyl coenzyme A 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 <sup>[4]</sup> . 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.

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[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.

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

Tel: 609-228-6898

Fax: 609-228-5909

E-mail: [tech@MedChemExpress.com](mailto:tech@MedChemExpress.com)

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