Acetylcysteine

Cat. No.: HY-B0215

CAS No.: 616-91-1

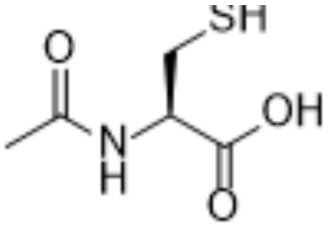
Molecular Formula: $C_5H_9NO_3S$

Molecular Weight: 163.19

Target: Reactive Oxygen Species; Endogenous Metabolite; Apoptosis; Ferroptosis; Influenza Virus

Pathway: Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB; Apoptosis; Anti-infection

Storage: 4°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 163 mg/mL (998.84 mM; ultrasonic and adjust pH to 7 with NaOH)
DMSO : ≥ 100 mg/mL (612.78 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	6.1278 mL	30.6391 mL	61.2783 mL
5 mM	1.2256 mL	6.1278 mL	12.2557 mL
10 mM	0.6128 mL	3.0639 mL	6.1278 mL

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

In Vivo

1. Add each solvent one by one: PBS
Solubility: 120 mg/mL (735.34 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Acetylcysteine (N-Acetylcysteine) is a mucolytic agent which reduces the thickness of the mucus. Acetylcysteine is a ROS inhibitor[1]. Acetylcysteine is a cysteine precursor, prevents hemin-induced ferroptosis by neutralizing toxic lipids generated by arachidonate-dependent activity of 5-lipoxygenases[5]. Acetylcysteine induces cell apoptosis[2][3]. Acetylcysteine also has anti-influenza virus activities[7].

IC_{50} & Target

Human Endogenous Metabolite

In Vitro

Acetylcysteine prevents apoptotic DNA fragmentation and maintains long-term survival in the absence of other trophic support in serum-deprived PC12 cells. Acetylcysteine also prevents death of PC12 cells and sympathetic neurons[2]. Acetylcysteine causes dose-dependent reductions in viability in rat and human aortic smooth muscle cells[3].

	Acetylcysteine activates the Ras-extracellular signal-regulated kinase (ERK) pathway in PC12 cells. Acetylcysteine protects neuronal cells from death evoked by withdrawal of trophic support. Acetylcysteine increases nitric oxide (NO) release from protein-bound stores in vascular tissue. Acetylcysteine pretreatment of PC12 cells interferes with NGF-dependent signaling and neurite outgrowth, and it is suggested that Acetylcysteine interferes with redox-sensitive steps in the NGF mechanism[4] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Acetylcysteine (150, 300 mg/kg) treatment significantly reduces liver transaminases in all groups of treatment, mostly in group Acetylcysteine 300. Lung glutathione peroxidase is significantly increases in group Acetylcysteine 300 (P=0.04), while the other oxidation biomarkers show no significant differences[6]. Acetylcysteine improves cognition of 12-month-old SAMP8 mice in both the T-maze footshock avoidance paradigm and the lever press appetitive task without inducing non-specific effects on motor activity, motivation to avoid shock, or body weight[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	For survival experiments, washed cells are resuspended in RPM1 1640 medium and plated in 0.5 mL at a density of 8-10×10^5 per well in 24 well plastic culture dishes coated with rat tail collagen. To feed, but to avoid loss of floating cells, fresh medium (0.2 mL) is added to the cultures on days 1, 5, and 10. For experiments involving "primed" PC12 cells, cultures are pretreated for l-2 weeks with NGF in RPM1 1640 medium supplemented with 1% heat-iN-acetylcysteinetivated horse serum. The cells are then washed and passaged into serum-free RPM1 1640 medium. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [6]	Rats: Rats are randomLy allocated into five groups: sham group (n=5), control group with IIR (n=8) and three groups with IIR who are given Acetylcysteine in different dosages: 150 mg/kg intraperitoneally 5 min before ischemia (n=8, group Acetylcysteine 150), 300 mg/kg i.p 5 min before ischemia (n=7, group Acetylcysteine 300), and 150 mg/kg i.p 5 min before ischemia plus 150 mg/kg 5 min before reperfusion (n=7, group Acetylcysteine 150 + 150). After 4 h of reperfusion, the animals are euthanized by exsanguination from the abdominal aorta. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Signal Transduct Target Ther. 2021 May 28;6(1):188.
- Signal Transduct Target Ther. 2020 May 8;5(1):51.
- Adv Mater. 2022 Dec 28;e2209910.
- Cell Metab. 2019 Dec 3;30(6):1107-1119.e8.
- Sci Immunol. 2022 Feb 4;7(68):eabk2092.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Halasi M, et al. ROS inhibitor N-acetyl-L-cysteine antagonizes the activity of proteasome inhibitors. Biochem J. 2013 Sep 1;454(2):201-8.

[2]. Ferrari G, et al. N-acetylcysteine (D- and L-stereoisomers) prevents apoptotic death of neuronal cells. J Neurosci. 1995 Apr;15(4):2857-66.

[3]. Tsai JC, et al. Induction of apoptosis by pyrrolidinedithiocarbamate and N-acetylcysteine in vascular smooth muscle cells. J Biol Chem. 1996 Feb 16;271(7):3667-70.

[4]. Yan CY, et al. Prevention of PC12 cell death by N-acetylcysteine requires activation of the Ras pathway. J Neurosci. 1998 Jun 1;18(11):4042-9.

[5]. Farr SA, et al. The antioxidants alpha-lipoic acid and N-acetylcysteine reverse memory impairment and brain oxidative stress in aged SAMP8 mice. J Neurochem. 2003 Mar;84(5):1173-83.

[6]. Kalimeris K, et al. N-acetylcysteine ameliorates liver injury in a rat model of intestinal ischemia reperfusion. J Surg Res. 2016 Dec;206(2):263-272.

[7]. Garigliany MM, et al. N-acetylcysteine lacks universal inhibitory activity against influenza A viruses. J Negat Results Biomed. 2011 May 9;10:5.

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