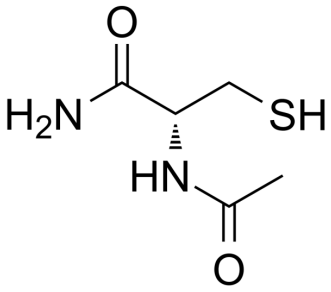
N-Acetylcysteine amide



Cat. No.: HY-110256

CAS No.: 38520-57-9

Molecular Formula: $C_5H_{10}N_2O_2S$

Molecular Weight: 162.21

Target: Reactive Oxygen Species

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

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 : 200 mg/mL (1232.97 mM; Need ultrasonic)

DMSO : ≥ 100 mg/mL (616.48 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	6.1648 mL	30.8242 mL	61.6485 mL
5 mM	1.2330 mL	6.1648 mL	12.3297 mL
10 mM	0.6165 mL	3.0824 mL	6.1648 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 (616.48 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (15.41 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (15.41 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (15.41 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

N-Acetylcysteine amide is a cell membranes and blood brain barrier permeant thiol antioxidant and neuroprotective agent, reduces ROS production.

In Vitro	N-Acetylcysteine amide shows no obvious effect on the viability of H9c2 cells treated with doxorubicin (DOX) at < 1 mM, but causes significant cytotoxicity at 10-20 mM. N-Acetylcysteine amide (750 μM) reduces the ROS levle and lipid peroxidation induced by DOX, and restores GSH/GSSG ratio and activities of antioxidant enzymes, such as catalase (CAT), gluthathione peroxidase (GPx), gluthathione reductase (GR)[1]. N-Acetylcysteine amide (1 mM) protects the human brain microvascular endothelial (HBMVEC) from methamphetamine (METH)- induced cell death[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	N-Acetylcysteine amide has increased CNS bioavailability. N-Acetylcysteine amide (150 mg/kg, i.p.) improves cortical sparing and functional outcome, reduces oxidative stress, improves mitochondrial bioenergetics, and maintains mitochondrial glutathione content following traumatic brain injury (TBI) in rats[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	To choose a sublethal concentration of N-Acetylcysteine amide and N-acetylcysteine for the study on their ability to protect cells from doxorubicin (DOX)-induced toxicity, H9c2 cells are exposed with N-Acetylcysteine amide or N-acetylcysteine at 0.25 mM, 0.50 mM, 0.75 mM, 1 mM, 2 mM, 5 mM, 10 mM, and 20 mM for 24 h. Untreated cells are used as the control for each experiment[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Rats[2] In order to assess mitochondrial respiration and glutathione content following traumatic brain injury (TBI), rats are randomly divided into three groups (n = 5 animals/group). (I.) N-Acetylcysteine amide group receives multiple bolus IP injections of N-Acetylcysteine amide (150 mg/kg) immediately after 5 minutes and then every 6 hours up to 24 hrs post-injury. (II.) Vehicle group receives equivalent v/v saline at 5 minutes and every 6 hours (6, 12, 18, 24 hrs) up to 24 hrs post-injury. (III.) Sham injured group animals do not receive any drug treatment. At 25 hrs post-injury, all animals are euthanized and mitochondria are isolated from the ipsilateral cortical hemisphere (6 mm punch) to carry out measurements of mitochondrial respiration and glutathione content[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Metab. 2021 Sep;3(9):1242-1258.
- Nanotoxicology. 2020 Jun;14(5):667-682.
- J Neurosci Res. 2019 Dec;97(12):1689-1705.
- J Cell Mol Med. 2020 Jan;24(2):1332-1344.
- Toxicology. 2019 Apr 15;418:22-31.

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REFERENCES

[1]. Shi R, et al. N-acetylcysteine amide decreases oxidative stress but not cell death induced by doxorubicin in H9c2 cardiomyocytes. BMC Pharmacol. 2009 Apr 15;9:7.

[2]. Pandya JD, et al. N-acetylcysteine amide confers neuroprotection, improves bioenergetics and behavioral outcome following TBI. Exp Neurol. 2014 Jul;257:106-13.

[3]. Zhang X, et al. N-Acetylcysteine amide protects against methamphetamine-induced oxidative stress and neurotoxicity in immortalized human brain endothelial cells.

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