**N-Acetylcysteine amide**

**Cat. No.**: HY-110256  
**CAS No.**: 38520-57-9  
**Molecular Formula**: C₅H₁₀N₂O₂S  
**Molecular Weight**: 162.21  
**Target**: Others  
**Pathway**: Others  
**Storage**: Powder -20°C 3 years  
4°C 2 years  
In solvent -80°C 6 months  
-20°C 1 month

### Solvent & Solubility

**In Vitro**  
DMSO : ≥ 100 mg/mL (616.48 mM)  
*“≥” means soluble, but saturation unknown.*

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Solvent Concentration</th>
<th>Mass 1 mg</th>
<th>Mass 5 mg</th>
<th>Mass 10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 mM</td>
<td>6.1648 mL</td>
<td>30.8242 mL</td>
<td>61.6485 mL</td>
</tr>
<tr>
<td></td>
<td>5 mM</td>
<td>1.2330 mL</td>
<td>6.1648 mL</td>
<td>12.3297 mL</td>
</tr>
<tr>
<td></td>
<td>10 mM</td>
<td>0.6165 mL</td>
<td>3.0824 mL</td>
<td>6.1648 mL</td>
</tr>
</tbody>
</table>

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

### BIOLOGICAL ACTIVITY

**Description**
N-Acetylcysteine amide is a cell membranes and blood brain barrier permeant thiol antioxidant and neuroprotective agent.

**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 level and lipid peroxidation induced by DOX, and restores GSH/GSSG ratio and activities of antioxidant enzymes, such as catalase (CAT), glutathione peroxidase (GPx), glutathione reductase (GR)[1]. N-Acetylcysteine amide (1 mM) protects the human brain microvascular endothelial (HBMVEC) from methamphetamine (METH)-induced cell death[3].

**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].
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.

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.

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REFERENCES

