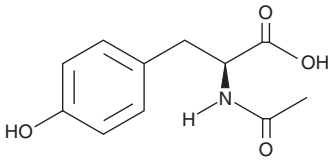
# PRODUCT INFORMATION

## N-Acetyl-L-tyrosine

*Item No. 34345*

**CAS Registry No.:** 537-55-3
**Synonyms:** N-Acetyltyrosine, NAT, NSC 10853
**MF:** $C_{11}H_{13}NO_4$
**FW:** 223.2
**Purity:** ≥98%
**Supplied as:** A solid
**Storage:** -20°C
**Stability:** ≥4 years




*Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.*

### Laboratory Procedures

N-Acetyl-L-tyrosine (NAT) is supplied as a solid. A stock solution may be made by dissolving the NAT in the solvent of choice, which should be purged with an inert gas. NAT is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide. The solubility of NAT in these solvents is approximately 25 mg/ml.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. Organic solvent-free aqueous solutions of NAT can be prepared by directly dissolving the solid in aqueous buffers. The solubility of NAT in PBS (pH 7.2) is approximately 10 mg/ml. We do not recommend storing the aqueous solution for more than one day.

### Description

NAT is an active metabolite of, and precursor to, L-tyrosine.[1,2] Dietary administration of NAT increases *B. mori* larvae heat stress tolerance and *A. mellifera* survival in a model of environmental stress.[1] Blood levels of NAT are increased during heat and restraint stress in mice, and dietary administration of NAT decreases blood lipid peroxidation and corticosterone levels in a mouse model of restraint stress. Dietary administration of NAT also decreases tumor volume in an HCT116 mouse xenograft model. Urinary excretion of NAT is increased in patients with phenylketonuria (PKU) or tyrosinemia, inborn errors of amino acid metabolism characterized by mutations in the gene encoding phenylalanine hydroxylase (PAH) and a deficiency in fumarylacetoacetate hydrolase (FAH), the final enzyme in tyrosine catabolism, respectively.[2] Formulations containing NAT have been used for amino acid supplementation in parenteral nutrition for preterm infants.

### References


1. Matsumura, T., Uryu, O., Matsuhisa, F., *et al.* N-acetyl-L-tyrosine is an intrinsic triggering factor of mitohormesis in stressed animals. *EMBO Rep.* **21(5)**, e49211 (2020).
2. Jellum, E., Horn, L., Thoresen, O., *et al.* Urinary excretion of N-acetyl amino acids in patients with some inborn errors of amino acid metabolism. *Scand. J. Clin. Lab. Invest. Suppl.* **184**, 21-26 (1986).





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