

Product Data Sheet

Product Name: SNAP
Cat. No.: GC10767

Chemical Properties

Cas. No. 67776-06-1

Chemical Name (R)-2-acetamido-3-methyl-3-(nitrosothio)butanoic acid

SMILES CC(=O)N[C@@H](C(C)(C)SN=O)C(=O)O

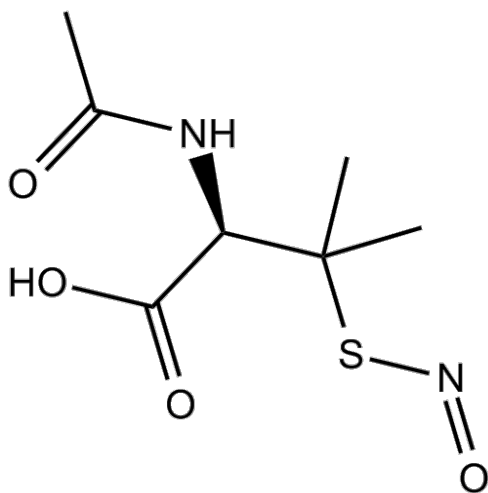
Formula $C_7H_{12}N_2O_4S$ M.Wt 220.24

Solubility <22.02mg/ml in DMSO Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines Fu5 rat hepatoma cells (rat hepatoma cell line)

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

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Preparation Method	Fu5 cells were maintained in Dulbecco's Modified Eagle Medium/Nutrient Mix F12 (1:1) supplemented with 2mM L-glutamine, 50µg/mL gentamycin, and 10% fetal-calf serum at 37°C under 5% CO ₂ . Cells were treated with SNAP at a concentration of 1mM for 8 hours.
Reaction Conditions	1mM; 8h
Applications	SNAP induced significant cytotoxicity in Fu5 cells. When combined with xanthine oxidase (20m-units/mL), SNAP synergistically enhanced cytotoxicity. The cytotoxic effects were mediated by nitric oxide (NO) release, independent of superoxide anion (O ₂ ⁻) or peroxynitrite (ONOO ⁻) pathways.
Animal experiment [2]:	
Animal models	SD rats
Preparation Method	Rats were intramuscularly administered a single dose of SNAP (1.0mg/kg) at 3 hours after critical limb ischemia (CLI) induction. Rats were sacrificed at day 14 for quadriceps muscle analysis.
Dosage form	1.0mg/kg; i.m.; 14 days
Applications	SNAP administration significantly increased the ischemic-to-normal blood flow (INBF) ratio and enhanced small vessel density in the ischemic limb. SNAP upregulated endothelial cell surface markers (CD31/vWF/eNOS) and angiogenesis factors, while reducing oxidative stress, apoptosis, and inflammatory biomarkers.

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References:

- [1] Ioannidis I, de Groot H. Cytotoxicity of nitric oxide in Fu5 rat hepatoma cells: evidence for co-operative action with hydrogen peroxide. *Biochem J.* 1993 Dec 1;296 (Pt 2)(Pt 2):341-5.
- [2] Yeh JN, Yip HK, Shao PL, et al. Combination of melatonin-delivered endothelial progenitor cells with S-nitroso-N-acetyl-DL-penicillamine for improving critical limb ischemia in the rat. *Am J Transl Res.* 2024 Sep 15;16(9):5020-5037.

Background

SNAP is a nitric oxide (NO) donor, commonly used as a tool compound for studying the physiological and pharmacological roles of nitric oxide^[1]. SNAP decomposes spontaneously or under the influence of light, heat, or metal ions to release nitric oxide, while generating the corresponding disulfide^[2]. SNAP is widely employed to investigate the mechanisms of cardiovascular regulation, neurotransmission, and immune responses^[3-4].

In vitro, treatment of Fu5 rat hepatoma cells with SNAP (1mM) for 8 hours significantly induced cytotoxicity^[5]. Exposure of Lewis lung carcinoma cells (LLC-F6) and B16 melanoma cells (B16-4A5-F6) to SNAP (2mM) for 24 hours markedly reduced the viability of both cell types^[6].

In vivo, a single intramuscular injection of SNAP (1.0mg/kg) administered 3 hours after the induction of critical limb ischemia (CLI) in SD rats significantly increased the ischemic-to-normal limb blood flow (INBF) ratio on day 14 and enhanced microvessel density in the ischemic area^[7]. Continuous intracerebroventricular infusion of SNAP (0.1μM; 2μL) over 6 days in P2X7R^{+/+} and P2X7R^{-/-} mice significantly elevated S-nitrosylation of protein disulfide isomerase (SNO-PDI) in the hippocampus and enhanced phosphorylation of NF-κB p65 at serine 276 in microglia^[8].

References:

- [1] Salas E, Moro MA, Askew S, et al. Comparative pharmacology of analogues of S-nitroso-N-acetyl-DL-penicillamine on human platelets. *Br J Pharmacol.* 1994 Aug;112(4):1071-6.
- [2] Pravdic D, Vladic N, Cavar I, et al. Effect of nitric oxide donors S-nitroso-N-acetyl-DL-penicillamine, spermine NONOate and propylamine propylamine NONOate on intracellular pH in cardiomyocytes. *Clin Exp Pharmacol Physiol.* 2012 Sep;39(9):772-8.

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- [3] Mang CF, Kilbinger H. Modulation of acetylcholine release in the guinea-pig trachea by the nitric oxide donor, S-nitroso-N-acetyl-DL-penicillamine (SNAP). Br J Pharmacol. 2000 Sep;131(1):94-8.
- [4] Lu J, Katano T, Okuda-Ashitaka E, et al. Involvement of S-nitrosylation of actin in inhibition of neurotransmitter release by nitric oxide. Mol Pain. 2009 Sep 29;5:58.
- [5] Ioannidis I, de Groot H. Cytotoxicity of nitric oxide in Fu5 rat hepatoma cells: evidence for co-operative action with hydrogen peroxide. Biochem J. 1993 Dec 1;296 (Pt 2)(Pt 2):341-5.
- [6] Hirano S. In vitro and in vivo cytotoxic effects of nitric oxide on metastatic cells. Cancer Lett. 1997 May 1;115(1):57-62.
- [7] Yeh JN, Yip HK, Shao PL, et al. Combination of melatonin-delivered endothelial progenitor cells with S-nitroso-N-acetyl-DL-penicillamine for improving critical limb ischemia in the rat. Am J Transl Res. 2024 Sep 15;16(9):5020-5037.
- [8] Kim JE, Wang SH, Lee DS, et al. Protein disulfide isomerase integrates toll-like receptor 4 and P2X7 receptor signaling pathways during lipopolysaccharide-induced neuroinflammation. Sci Rep. 2025 Mar 6;15(1):7906.

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