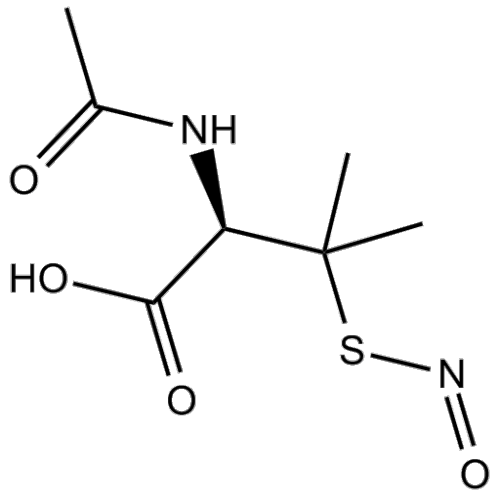
Product Data Sheet

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	OC([C@H](C(C)(C)SN=O)NC(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)

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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.

Caution: Product has not been fully validated for medical applications. For research use only.
Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA

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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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