

# **Product** Data Sheet

# S-Nitroso-N-acetyl-DL-penicillamine

Cat. No.:HY-121526CAS No.:67776-06-1Molecular Formula: $C_7H_{12}N_2O_4S$ Molecular Weight:220.25Target:NO Synthase

Pathway: Immunology/Inflammation

Storage: Powder -20°C 3 years 4°C 2 years

\* The compound is unstable in solutions, freshly prepared is recommended.

#### **SOLVENT & SOLUBILITY**

In Vitro

DMSO: 250 mg/mL (1135.07 mM; Need ultrasonic)  $H_2O: 11.11 \text{ mg/mL}$  (50.44 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent Mass Concentration	1 mg	5 mg	10 mg
	1 mM	4.5403 mL	22.7015 mL	45.4030 mL
	5 mM	0.9081 mL	4.5403 mL	9.0806 mL
	10 mM	0.4540 mL	2.2701 mL	4.5403 mL

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

In Vivo

- 1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: ≥ 2.08 mg/mL (9.44 mM); Clear solution
- 2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE- $\beta$ -CD in saline) Solubility:  $\geq$  2.08 mg/mL (9.44 mM); Clear solution

## **BIOLOGICAL ACTIVITY**

S-Nitroso-N-acetyl-DL-penicillamine (SNAP) is a nitric oxide donor and acts as a stable inhibitor of platelet aggregation<sup>[1][2]</sup>

In Vitro

S-Nitroso-N-acetyl-DL-penicillamine (10 mM; 8 hours) induces toxicity of about 80% after 6 hours under normoxic conditions by releasing nitric oxide (NO)<sup>[1]</sup>.

?S-Nitroso-N-acetyl-DL-penicillamine has a half-time about 6 hours in in isolated rat ventricular myocytes<sup>[3]</sup>.

?S-Nitroso-N-acetyl-DL-penicillamine (100  $\mu$ M; 30 minutes) causes sustained decrease in the basal pHi in isolated rat ventricular myocytes<sup>[3]</sup>.

 $\label{eq:mce} \mbox{MCE has not independently confirmed the accuracy of these methods. They are for reference only.}$ 

Cell Viability Assay<sup>[1]</sup>

	Cell Line:	Rat liver sinusoidal endothelial cells	
	Concentration:	2 mM, 5 mM, 10 mM	
	Incubation Time:	2 hours, 4 hours, 6 hours, 8 hours	
	Result:	Exhibited cytotoxicity against cultivated endothelial cells.	
In Vivo	SNAP (100 $\mu$ M, 300 $\mu$ M) causes small but significant increases of the electrically evoked [ $^3$ H]-acetylcholine release in guineapig tracheal [ $^4$ ].		

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

### **CUSTOMER VALIDATION**

• Hepatol Commun. 2023 Dec 22;8(1):e0350.

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#### **REFERENCES**

- [1]. E. Salas, 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]. Ioannidis I, et al. Enhanced release of nitric oxide causes increased cytotoxicity of S-nitroso-N-acetyl-DL-penicillamine and sodium nitroprusside under hypoxic conditions. Biochem J. 1996 Sep 15;318 (Pt 3):789-95.
- [3]. Pravdic D, 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.
- [4]. Mang CF, et al. 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.

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

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