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L-NAME hydrochloride

Cat. No.: HY-18729A

CAS No.: 51298-62-5

Molecular Formula: $C_7H_{16}ClN_5O_4$

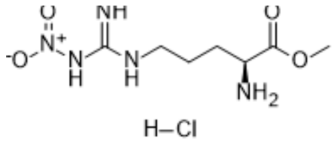
Molecular Weight: 269.69

Target: NO Synthase

Pathway: Immunology/Inflammation

Storage: -20°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (370.80 mM; Need ultrasonic)

DMSO : 100 mg/mL (370.80 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.7080 mL	18.5398 mL	37.0796 mL
5 mM	0.7416 mL	3.7080 mL	7.4159 mL
10 mM	0.3708 mL	1.8540 mL	3.7080 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 140 mg/mL (519.11 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

L-NAME hydrochloride inhibits NOS with an IC_{50} of 70 μM. L-NAME is a precursor to NOS inhibitor L-NOARG which has an IC_{50} value of 1.4 μM.

IC_{50} & Target

IC50: 70 μM (NOS)[1]

In Vitro

L-arginine analogues are widely used inhibitors of nitric oxide synthase (NOS) activity, with N^{w}-nitro-L-arginine methyl ester (L-NAME) being at the head[2]. Freshly dissolved L-NAME is a 50 fold less potent inhibitor of purified brain NOS (mean IC_{50}= 70 μM) than L-NOARG (IC_{50}= 1.4 μM), but the apparent inhibitory potency of L-NAME approached that of L-NOARG upon prolonged incubation at neutral or alkaline pH. HPLC analyses reveal that NOS inhibition by L-NAME closely correlated with hydrolysis of the drug to L-NOARG[1].

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

In Vivo

L-NAME hydrochloride can be used in animal modeling to construct cardiovascular and cerebrovascular disease models.

L-NAME infusion significantly decreases NKT-leukocyte level, tumor-necrosis factor (TNF)-alpha production by T-splenocytes and macrophages, and IFNγ production by T-leukocytes, monocytes, and T-splenocytes, as well as increased interleukin-6 production by T-leukocytes and monocytes and nitrate/nitrite production by T-leukocytes[3]. There is increasing evidence that nitric oxide may be involved in learning and memory. l-NAME produces a task-dependent impairment of fear extinction, and implies that nitric oxide signaling is involved in memory process of certain fear extinction tasks[4]. Chronic L-NAME administration induces cardiac hypertrophy in rodent models. Six weeks L-NAME administration induces significant cardiac hypertrophy compared to control hearts[5].

b>L-NAME hydrochloride is a classic hypertension modeling agent that decreases nitric oxide (NO) release with an inhibition competence in endothelial nitric oxide synthase (eNOS) in animals. Rats and mice are generally used as animal models[6][7].

Dose reference for L-NAME hydrochloride induction[6][7]:
(1) Model animal: Swiss Webster male mice
Hypertension Model: 400 mg/kg/day, i.p, 7 day
(2) Model animals: Male Sprague-Dawley (SD) rats
Hypertension Model: 40 mg/kg, drinking water, 5 weeks

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

Animal Model:	Male Sprague-Dawley (SD) rats [7]
Dosage:	40 mg/kg, 5 weeks
Administration:	drinking water
Result:	Induced hypertension with body weight loss and high blood pressure.

CUSTOMER VALIDATION

- J Extracell Vesicles. 2023 May;12(5):e12328.
- Arterioscler Thromb Vasc Biol. 2020 Jul;40(7):1705-1721.
- Cell Commun Signal. 2021 Mar 18;19(1):35.
- Front Med. 2021 Apr 13;8:634882.
- JCI Insight. 2021 Sep 22;6(18):e133690.

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REFERENCES

[1]. V A Peotta, et al. Cardiovascular neural reflexes in L-NAME-induced hypertension in mice. Hypertension. 2001, 38, 3.

[2]. Xiaofei Li, et al. Fucoidan from Undaria pinnatifida prevents vascular dysfunction through PI3K/Akt/eNOS-dependent mechanisms in the l-NAME-induced hypertensive rat model. Food Funct. 2016, 7, 5.

[3]. Pfeiffer S, et al. Inhibition of nitric oxide synthesis by NG-nitro-L-arginine methyl ester (L-NAME): requirement forbioactivation to the free acid, NG-nitro-L-arginine. Br J Pharmacol. 1996 Jul;118(6):1433-40.

[4]. Kopincová J, et al. L-NAME in the cardiovascular system - nitric oxide synthase activator? Pharmacol Rep. 2012;64(3):511-20.

[5]. Lo HC, et al. The Nitric Oxide Synthase Inhibitor NG-Nitro-L-Arginine Methyl Ester Diminishes the Immunomodulatory Effects of Parental Arginine in Rats with Subacute

Peritonitis. PLoSOne. 2016 Mar 23;11(3):e0151973.

[6]. Luo H, et al. Effect of nitric oxide synthase inhibitor L-NAME on fear extinction in rats: a task-dependent effect. Neurosci Lett. 2014 Jun 20;572:13-8.

[7]. Ocsan RJ, et al. Chronic NG-nitro-l-arginine methyl ester (L-NAME) administration in C57BL/6J mice induces a sustained decrease in c-kit positive cells during development of cardiac hypertrophy. J Physiol Pharmacol. 2013 Dec;64(6):727-36.

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