L-NAME hydrochloride

Cat. No.:	HY-18729A	
CAS No.:	51298-62-5	
Molecular Formula:	C ₇ H ₁₆ CIN ₅ O ₄	O NH O
Molecular Weight:	269.69	
Target:	NO Synthase	H H NH ₂
Pathway:	Immunology/Inflammation	H–Cl
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

	Concentration	1 mg	5 mg	10 mg		
Preparing Stock Solutions	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 so	Please refer to the solubility information to select the appropriate solvent.					
1. Add each solvent	1. Add each solvent one by one: PBS					
	Please refer to the sol	Stock Solutions 5 mM 10 mM Please refer to the solubility information to select the approximation to select the approximation to select the approximation to select the approximation to select the solubility information to select the approximation to select the solubility information to select the solubi	Stock Solutions 5 mM 0.7416 mL 10 mM 0.3708 mL Please refer to the solubility information to select the appropriate solvent.	Stock Solutions 5 mM 0.7416 mL 3.7080 mL 10 mM 0.3708 mL 1.8540 mL Please refer to the solubility information to select the appropriate solvent. 1. Add each solvent one by one: PBS		

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.			

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L-NAME infusion significantly decreases NKT-leukocyte level, tumor-necrosis factor (TNF)-alpha production by Tsplenocytes 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. I-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].

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PROTOCOL

Animal Administration ^{[3][5]} Rats: The purpose is to investigate dose effects of chronically infused NOS inhibitor, LNAME on the anabolism, inflammatory responses, and arginine metabolism in parenterally fed rats with cecal puncture-induced subacute peritonitis. Male Wistar rats (8-9 weeks old), initially weighing 250 g, are used in the study. Rats are divided into 4 groups and are administered total parenteral nutrition solutions with 0, 5 (low dose), 25 (medium dose), or 50 (high dose) mg/kg per day of L-NAME for 7 days [3].

Mice: 12-20 week old C57BL/6J mice (5 per group) are administered L-NAME (0.325mg/mL) in the drinking water. Hearts are excised at 1-day, 2-days, 5-days, 2-weeks or 6-weeks; or controls which received no L-NAME. Ventricular cross-sectional wall thickness and individual cardiac myocytes cross-sectional area and cardiomyocyte/nuclear ratio to determine cardiac hypertrophy. Immuno-histochemical staining for c-kit, sca-1 and BCRP undertaken^[5].

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

CUSTOMER VALIDATION

- J Extracell Vesicles. 2023 May;12(5):e12328.
- Arterioscler Thromb Vasc Biol. 2020 Jul;40(7):1705-1721.
- JCI Insight. 2021 Sep 22;6(18):e133690.
- Int J Mol Sci. 2023 Mar 7.
- Nutrients. 2022, 14(23), 5025

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REFERENCES

[1]. 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.

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

[3]. 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.

[4]. 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.

[5]. 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.

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

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