

Product Data Sheet

Product Name: L-NAME hydrochloride

Cat. No.: GA11233

Chemical Properties

Cas. No. 51298-62-5

Chemical Name methyl (2S)-2-amino-5-[[amino(nitramido)methylidene]amino]pentanoate;hydrochloride

SMILES COC(=O)C(CCCN=C(N)N[N+](=O)[O-])N.Cl

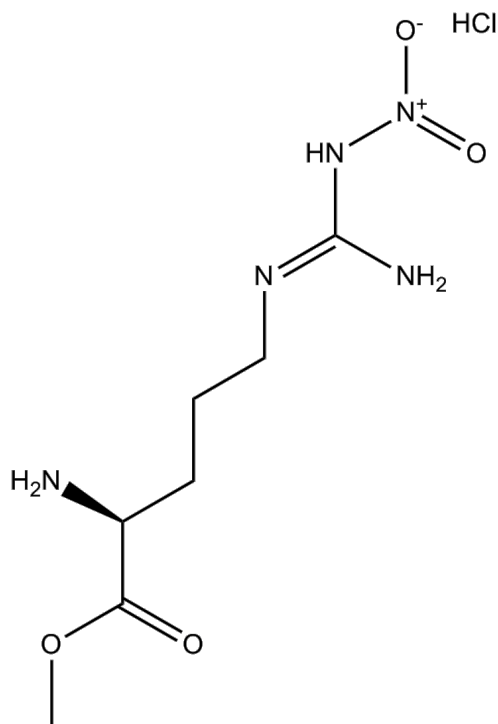
Formula $C_7H_{15}N_5O_4 \cdot HCl$ M.Wt 269.7

Solubility $\geq 27\text{mg/mL}$ in Water, $\geq 23\text{ mg/mL}$ in DMSO Storage Stored 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



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

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Protocol

Cell experiment [1]:	
Cell lines	HeLa cells (human cervical cancer cell line)
Preparation Method	HeLa cells were maintained in DMEM supplemented with 10% FBS at 37°C, 5% CO ₂ . HeLa cells were pre-treated with L-NAME hydrochloride at 500μM for 24h prior to and during 100μM cadmium chloride exposure for up to 24h, after which nitrotyrosine formation, HSP27 levels, cell viability, ROS/peroxynitrite, NO bioavailability, lipid peroxidation and cell death were assessed.
Reaction Conditions	500μM; 24h pre-treatment
Applications	L-NAME hydrochloride delayed cadmium-induced nitrotyrosine formation in HeLa cells. L-NAME hydrochloride partially prevented cadmium-induced reduction of intracellular HSP27 levels. L-NAME hydrochloride attenuated cadmium-induced decrease in cell viability. L-NAME hydrochloride mitigated cadmium-induced necrotic cell death and reduced apoptotic cell death at 3h. L-NAME hydrochloride reduced cadmium-elevated ROS and peroxynitrite signals and blunted the cadmium-driven drop in NO bioavailability.
Animal experiment [2]:	
Animal models	Male adult Swiss Webster mice (28-34g)

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Preparation Method	Mice were intraperitoneally administered L-NAME hydrochloride at 400µg/g daily for 7 days (control group received saline 13µL/g i.p. daily). On day 7, mice were anesthetized with urethane (1mg/g i.p.), instrumented with femoral artery and vein catheters, and baseline mean arterial pressure (MAP) and heart rate (HR) were recorded. Phenylephrine (1µg/g IV, 10s infusion), sodium nitroprusside (1µg/g IV, 10s infusion), phenylbiguanide (40ng/g IV bolus) and potassium cyanide (0.26µg/g IV bolus) were administered to test baroreflex, Bezold-Jarisch reflex and chemoreflex; hearts were collected for heart weight/body weight ratio at endpoint.
Dosage form	400µg/g; i.p.; daily for 7 days
Applications	L-NAME hydrochloride administration increased baseline MAP under anesthesia without changing HR. L-NAME hydrochloride reduced body weight gain throughout the 7-day treatment. L-NAME hydrochloride did not change heart weight/body weight ratio. L-NAME hydrochloride enhanced baroreflex sensitivity to phenylephrine-induced pressor challenge. L-NAME hydrochloride attenuated phenylbiguanide-induced Bezold-Jarisch reflex. L-NAME hydrochloride attenuated potassium cyanide-induced chemoreflex. L-NAME hydrochloride attenuated phenylephrine-induced pressor response.
References: [1] Alvarez-Olmedo DG, Biaggio VS, Koumbadinga GA, et al. Recombinant heat shock protein 27 (HSP27/HSPB1) protects against cadmium-induced oxidative stress and toxicity in human cervical cancer cells. <i>Cell Stress Chaperones</i> . 2017;22(3):357-369. [2] Peotta VA, Vasquez EC, Meyrelles SS. Cardiovascular neural reflexes in L-NAME-induced hypertension in mice. <i>Hypertension</i> . 2001 Sep;38(3 Pt 2):555-559.	

Background

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L-NAME hydrochloride is a non-selective, competitive nitric oxide synthase (NOS) inhibitor ($IC_{50}=70\mu M$). L-NAME hydrochloride inhibits nNOS ($K_i=15nM$), eNOS ($K_i=39nM$) and iNOS ($K_i=4.4\mu M$) activities. L-NAME hydrochloride blocks the conversion of L-arginine to nitric oxide and citrulline, thereby reducing endogenous NO production, causing vasoconstriction, increased vascular tone and elevated blood pressure, and affecting cGMP generation, immune cytokine secretion and neural signal transduction. L-NAME hydrochloride can be used in research related to hypertension, endothelial dysfunction, cardiac hypertrophy, kidney injury, diabetes and its complications, neuroscience and learning memory, inflammation and immune regulation^[1-4].

In vitro, treatment with L-NAME hydrochloride ($100\mu M$) combined with ephrin-A1-Fc ($1\mu g/ml$) for 6h or 24h in human umbilical vein endothelial cells inhibited ephrin-A1-induced endothelial cell migration and tube formation, and blocked ephrin-A1-stimulated NO production^[5]. Pre-treatment with L-NAME hydrochloride ($500\mu M$) for 24h followed by co-treatment with $100\mu M$ cadmium chloride in HeLa cells delayed cadmium-induced protein nitration, prevented the decrease in intracellular HSP27 levels, improved cell viability, and reduced necrotic death and apoptosis^[6]. Pre-treatment with L-NAME hydrochloride ($1000\mu M$) for 1h followed by co-treatment with $100\mu M$ bezafibrate for 48h in mouse osteoblast-like MC3T3-E1 cells inhibited bezafibrate-induced enhancement of cell viability, and reversed bezafibrate-induced increases in alkaline phosphatase (ALP) activity and ALP, BMP-2, Runx-2 mRNA expression^[7].

In vivo, pregnant BALB/c mice received daily oral administration of L-NAME hydrochloride ($4.464mg/30g$) from gestational day 5 to day 15, L-NAME hydrochloride induced a preeclampsia-like phenotype, decreased splenic $CD4^+CD25^+$ regulatory T cell proportion, increased $CD11b^+IL-6^+$ and $CD11b^+IL-1\beta^+$ cell proportions, and reduced relative TGF- β expression within Tregs^[8]. Swiss Webster mice received daily intraperitoneal injections of L-NAME hydrochloride at $400\mu g/g$ for 7 consecutive days, L-NAME hydrochloride induced a hypertensive phenotype, elevated baseline mean arterial pressure, did not change heart rate, and reduced body weight^[9]. Male Sprague Dawley rats received oral L-NAME hydrochloride at $40mg/kg/day$ in drinking water for 5 consecutive weeks, L-NAME hydrochloride elevated systolic blood pressure, diastolic blood pressure and mean blood pressure, slowed body weight gain, and caused thoracic aorta medial thickening and decreased lumen/media ratio^[10].

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- [2] Lipton HL, Hao Q, Hyman A. L-NAME enhances pulmonary vasoconstriction without inhibiting EDRF-dependent vasodilation. J Appl Physiol (1985). 1992 Dec;73(6):2432-9.
- [3] Sung JH, Jo YS, Kim SJ, et al. Effect of Lutein on L-NAME-Induced Hypertensive Rats. Korean J Physiol Pharmacol. 2013 Aug;17(4):339-45.
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