L-NIL hydrochloride

Cat. No.: HY-12117

CAS No.: 150403-89-7

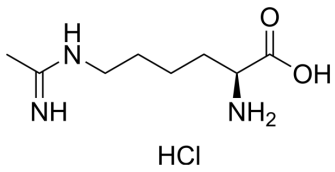
Molecular Formula: $C_8H_{18}ClN_3O_2$

Molecular Weight: 223.7

Target: NO Synthase

Pathway: Immunology/Inflammation

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (447.03 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	4.4703 mL	22.3514 mL	44.7027 mL
	5 mM	0.8941 mL	4.4703 mL	8.9405 mL
	10 mM	0.4470 mL	2.2351 mL	4.4703 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: 2.5 mg/mL (11.18 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (11.18 mM); Clear solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: 2.5 mg/mL (11.18 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

L-NIL hydrochloride is an inducible NO synthase inhibitor, with an IC_{50} of 3.3 μM for mouse inducible NOS[1][2][3].

IC_{50} & Target

IC50: 3.3 μM (mouse inducible NO synthase), 92 μM (rat brain constitutive NO synthase)[1].

In Vitro

L-NIL produces a concentration-dependent inhibition of both the mouse inducible NOS and the rat brain constitutive NOS (rcNOS) and is considerably more potent for mouse inducible NOS. The IC_{50} values for L-NIL with mouse inducible NOS and rcNOS are 3.3 and 92 pM, respectively, indicating that L-NIL is 28-fold more selective for mouse inducible NOS. In addition, L-NIL has approximately 6-fold greater potency for mouse inducible NOS than either L-NMA or L-NNA[3].

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

In Vivo

L-NIL (10 and 30 mg/kg, IP) prevents the inflammation, oxidative stress and autophagy induced by renal IR in mice[1].

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

Animal Model:	Adult male Balb/c (20-25 g)[1].
Dosage:	10 and 30 mg/kg.
Administration:	Intraperitoneally at the end of CLP and at 6 h after sepsis induction.
Result:	Led to a negligible increase in plasma NGAL compared to sham mice. Led to a significant decrease in both TLR4 and IL1β protein contents and clusterin transcript. Showed an increase in NFAT5 mRNA levels, as compared with mice treated with vehicle. Promoted a decrease in AR protein expression, as compared with animals treated with vehicle.

CUSTOMER VALIDATION

- Nat Biomed Eng. 2023 Mar;7(3):281-297.
- J Adv Res. 5 March 2022.
- Cancer Lett. 2023 Jul 29;216330.
- Free Radic Biol Med. 2023 Mar 3;S0891-5849(23)00100-4.

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REFERENCES

[1]. Consuelo Pasten, et al. l-NIL prevents the ischemia and reperfusion injury involving TLR-4, GST, clusterin, and NFAT-5 in mice. Am J Physiol Renal Physiol. 2019 Apr 1;316(4):F624-F634.

[2]. Sharon Angela Tanuseputero, et al. Intravenous Arginine Administration Downregulates NLRP3 Inflammasome Activity and Attenuates Acute Kidney Injury in Mice with Polymicrobial Sepsis. Mediators Inflamm. 2020 May 11;2020:3201635.

[3]. Moore WM, et al. L-N6-(1-iminoethyl)lysine: a selective inhibitor of inducible nitric oxide synthase. J Med Chem. 1994 Nov 11;37(23):3886-8.

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