L-NIL

Cat. No.: HY-12116

CAS No.: 53774-63-3

Molecular Formula: $C_8H_{17}N_3O_2$

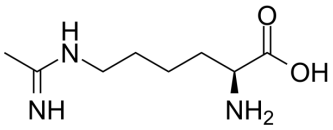
Molecular Weight: 187.24

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 : 50 mg/mL (267.04 mM; Need ultrasonic)

DMSO : < 1 mg/mL (insoluble or slightly soluble)

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	5.3407 mL	26.7037 mL	53.4074 mL
5 mM	1.0681 mL	5.3407 mL	10.6815 mL
10 mM	0.5341 mL	2.6704 mL	5.3407 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (534.07 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

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

IC_{50} & Target

iNOS

In Vitro

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

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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]. 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.

[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]. 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.

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