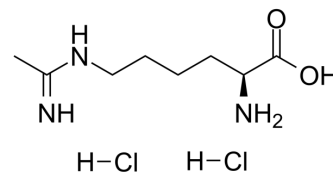


L-NIL dihydrochloride

Cat. No.:	HY-12118
CAS No.:	159190-45-1
Molecular Formula:	C ₈ H ₁₉ Cl ₂ N ₃ O ₂
Molecular Weight:	260.16
Target:	NO Synthase
Pathway:	Immunology/Inflammation
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	L-NIL dihydrochloride is an inducible NO synthase inhibitor, with an IC ₅₀ of 3.3 μM for miNOS ^{[1][2][3]} .								
IC₅₀ & Target	IC ₅₀ : 3.3 μM (mouse inducible NO synthase), 92 μM (rat brain constitutive NO synthase) ^[3] .								
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 ^[2] . 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.								
	<table border="1"> <tr> <td>Animal Model:</td> <td>Adult male Balb/c (20-25 g)^[1].</td> </tr> <tr> <td>Dosage:</td> <td>10 and 30 mg/kg.</td> </tr> <tr> <td>Administration:</td> <td>Intraperitoneally at the end of CLP and at 6 h after sepsis induction.</td> </tr> <tr> <td>Result:</td> <td>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.</td> </tr> </table>	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.
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CUSTOMER VALIDATION

- J Adv Res. 5 March 2022.

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REFERENCES

- [1]. Consuelo Pasten, et al. I-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]. W M Moore, 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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Caution: Product has not been fully validated for medical applications. For research use only.

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