

Product Data Sheet

Product Name: N6022
Cat. No.: GC16759

Chemical Properties

Cas. No. 1208315-24-5

Chemical Name 3-[1-(4-carbamoyl-2-methylphenyl)-5-(4-imidazol-1-ylphenyl)pyrrol-2-yl]propanoic acid

SMILES CC1=C(C=CC(=C1)C(=O)N)N2C(=CC=C2C3=CC=C(C=C3)N4C=CN=C4)CCC(=O)O

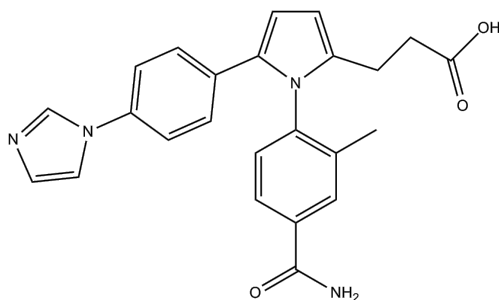
Formula C₂₄H₂₂N₄O₃ M.Wt 414.46

Solubility ≥ 20.7mg/mL in DMSO Storage Store at -20°C

General For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the tips ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Evaluation sample solution : ship with blue ice All other available size: ship with RT , or Condition blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines C2C12 cells (murine myoblast cell line)

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

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C2C12 cells were cultured in Dulbecco's modified Eagle's medium (DMEM) Preparation Method supplemented with 10% fetal bovine serum (FBS) at 37°C, 5% CO₂. C2C12 cells were treated with N6022 at a concentration of 200μM for 72 hours.

Reaction
Conditions 200μM; 72h

Applications N6022 significantly increased the number of mitochondria and induced the accumulation of endoplasmic reticulum vesicles in C2C12 cells. N6022 also upregulated the expression of the mitophagy-related protein PINK1.

Animal experiment [2]:

Animal models C57BL/6J mice (wild-type and GSNOR^{-/-})

Mice were subjected to spinal cord contusion injury at the T9-T10 vertebral level. Preparation Method N6022 (5mg/kg) was administered intravenously via tail vein injection at 2 hours post-injury, followed by daily injections until the study endpoint (up to 4 weeks).

Dosage form 5mg/kg; i.v.; Daily injections for 4 weeks.

Applications N6022 treatment significantly improved locomotor function, reduced pain-like behaviors, and alleviated depressive-like symptoms in SCI mice. N6022 also attenuated blood-spinal cord barrier disruption, edema, and neuroinflammation by inhibiting GSNOR activity, thereby promoting functional recovery.

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References:

- [1] Li J, Wu W, Ji G, et al. L-NAME improves the morphology and necrosis of skeletal muscle by activating PINK1-PARKIN mediated mitophagy in mdx mice. *Brain Dev.* 2025 Aug;47(4):104388.
- [2] Khan M, Qiao F, Islam SMT, et al. GSNOR and ALDH2 alleviate traumatic spinal cord injury. *Brain Res.* 2021 May 1;1758:147335. doi: 10.1016/j.brainres.2021.147335.

Background

N6022 is a selective and reversible inhibitor of S-nitrosogluthathione reductase (GSNOR), with an IC_{50} of 8nM and a K_i of 2.5nM^[1-2]. N6022 exhibits multiple biological functions by binding to the GSNO substrate-binding pocket, thereby increasing intracellular levels of S-nitrosogluthathione (GSNO) and modulating protein S-nitrosylation signaling pathways^[3-4].

In vitro, in an oxygen-glucose deprivation/reperfusion (OGD/R) model using BV2 microglial cells, treatment with N6022 (1–10 μ M) at the onset of reperfusion for 22 hours significantly enhanced cell viability, attenuated the decline in mitochondrial membrane potential, reduced the expression of the pro-apoptotic protein BAX, and upregulated the expression of Bcl-2^[5]. In murine myoblast C2C12 cells treated with N6022 (200 μ M) for 72 hours, N6022 markedly increased mitochondrial numbers and induced the accumulation of endoplasmic reticulum vesicles^[6].

In vivo, in a C57BL/6J mouse model of acetaminophen (APAP)-induced acute liver injury, intraperitoneal administration of N6022 (5mg/kg) at 2 hours post-APAP injection significantly reduced serum levels of liver injury markers AST and ALT, alleviated hepatic necrosis and apoptosis, and suppressed the expression of inflammatory factors^[7]. In a spinal cord injury (SCI) model in C57BL/6J mice, N6022 (5mg/kg) was administered via tail vein injection starting at 2 hours post-injury and continued for 4 weeks. N6022 significantly improved motor function (as assessed by BMS scores), reduced pain-like behaviors, and ameliorated depressive-like behaviors^[8].

References:

- [1] Sun X, Qiu J, Strong SA, et al. Structure-activity relationships of pyrrole based S-nitrosogluthathione reductase inhibitors: pyrrole regioisomers and propionic acid replacement. *Bioorg Med Chem Lett.* 2011 Jun 15;21(12):3671-5.
- [2] Colagiovanni DB, Drolet DW, Langlois-Forget E, et al. A nonclinical safety and pharmacokinetic

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- evaluation of N6022: a first-in-class S-nitrosoglutathione reductase inhibitor for the treatment of asthma. Regul Toxicol Pharmacol. 2012 Feb;62(1):115-24.
- [3] Blonder JP, Mutka SC, Sun X, et al. Pharmacologic inhibition of S-nitrosoglutathione reductase protects against experimental asthma in BALB/c mice through attenuation of both bronchoconstriction and inflammation. BMC Pulm Med. 2014 Jan 10;14:3.
- [4] Khan M, Kumar P, Qiao F, et al. Targeting GSNOR for functional recovery in a middle-aged mouse model of stroke. Brain Res. 2020 Aug 15;1741:146879.
- [5] Duan WL, Ma YP, Wang XJ, et al. N6022 attenuates cerebral ischemia/reperfusion injury-induced microglia ferroptosis by promoting Nrf2 nuclear translocation and inhibiting the GSNOR/GSTP1 axis. Eur J Pharmacol. 2024 Jun 5;972:176553.
- [6] Li J, Wu W, Ji G, et al. L-NAME improves the morphology and necrosis of skeletal muscle by activating PINK1-PARKIN mediated mitophagy in mdx mice. Brain Dev. 2025 Aug;47(4):104388.
- [7] Cui Q, Jiang T, Xie X, et al. S-nitrosylation attenuates pregnane X receptor hyperactivity and acetaminophen-induced liver injury. JCI Insight. 2024 Jan 23;9(2):e172632.
- [8] Khan M, Qiao F, Islam SMT, et al. GSNOR and ALDH2 alleviate traumatic spinal cord injury. Brain Res. 2021 May 1;1758:147335. doi: 10.1016/j.brainres.2021.147335.

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