

Product Data Sheet

Product Name: JSH-23
Cat. No.: GC11438

Chemical Properties

Cas. No. 749886-87-1

Chemical Name 4-methyl-1-N-(3-phenylpropyl)benzene-1,2-diamine

SMILES CC1=CC(=C(C=C1)NCCCC2=CC=CC=C2)N

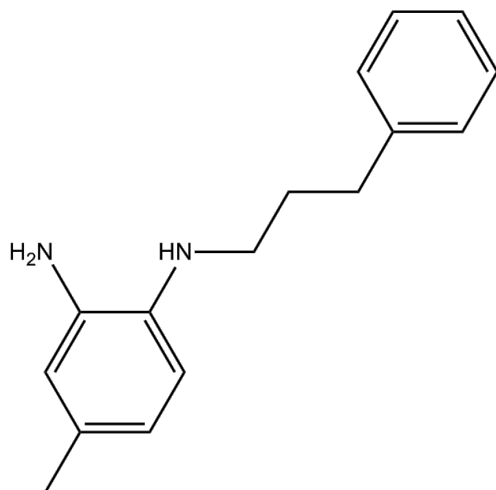
Formula $C_{16}H_{20}N_2$ M.Wt 240.34

Solubility $\geq 24\text{mg/mL}$ in DMSO Storage Store 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



Protocol

Cell experiment [1]:

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

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Cell lines	Normal human lung epithelial cell line BEAS-2B and human lung cancer cell line A549
Preparation Method	Cells were treated with different concentrations of NF- κ B inhibitor JSH-23 (5, 10, 20, 30, 40, and 50 μ M, respectively) for 24 h.
Reaction Conditions	5, 10, 20, 30, 40, and 50 μ M for 24 hours
Applications	With the increase in JSH-23 concentration, the inhibition rate for cell viability was gradually increased, and significant differences existed when the JSH-23 concentration was greater than 20 μ M.

Animal experiment [2]:

Animal models	Male 12-week-old normotensive Wistar rats and Prague hereditary hypertriglyceridemic (HTG) rats
Preparation Method	Mice were given a total dose of either 20 mg/kg of JSH-23 (10 mg/kg 8 h prior to cisplatin injection and 5 mg/kg on days 1 and 2 after cisplatin injection) or a total dose of 40 mg/kg body weight of JSH-23 (20 mg/kg 8 h prior to cisplatin injection and 20 mg/kg on day 1 after cisplatin injection) or vehicle (DMSO).
Dosage form	Intraperitoneal injection, 5, 10, 20 mg/kg

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Applications

JSH-23 (total dose of 40 mg/kg) resulted in a significant decrease in BUN, serum creatinine and serum NGAL. NGAL is an early diagnostic biomarker of cisplatin-induced AKI. JSH-23 had no effect on BUN and serum creatinine in vehicle-treated mice (Fig. 2).

References:

- [1]: Huang L, Li F, Deng P, et al. MicroRNA-223 promotes tumor progression in lung cancer A549 cells via activation of the NF- κ B signaling pathway[J]. Oncology research, 2016, 24(6): 405.
[2]: Ozkok A, Ravichandran K, Wang Q, et al. NF- κ B transcriptional inhibition ameliorates cisplatin-induced acute kidney injury (AKI)[J]. Toxicology letters, 2016, 240(1): 105-113.

Background

JSH-23 exhibited an inhibitory effect on nuclear translocation and NF- κ B transcriptional activity with an IC₅₀ value of 7.1 μ M in lipopolysaccharide (LPS)-stimulated macrophages RAW 264.7 [1].

JSH-23 (1 μ M) monotherapy significantly reduced the chemotactic sensitivity of the cells to SDF1. The co-treatment with cordycepin (10 μ M) and JSH-23 (1 μ M) significantly inhibited the expression of CXCR4 [2].

JSH-23 (1 and 3 mg/kg) treatment significantly reversed the nerve conduction and nerve blood flow deficits seen in diabetic animals [1]. Protein expression studies showed that nuclear translocation of the p65/p50 subunit was inhibited by JSH-23 treatment in the sciatic nerve. The treatment also lowered the elevated IL-6, TNF- α , cyclo-oxygenase (COX-2), and inducible nitric oxide synthase (iNOS) levels/expression [1]. Mice were treated with JSH-23 (20 or 40 mg/kg) which directly affects NF- κ B transcriptional activity. Kidney function, tubular injury (ATN, serum neutrophil gelatinase-associated lipocalin [NGAL], but not apoptosis) and myeloperoxidase (MPO) activity were significantly improved by JSH-23 (40 mg/kg) [3].

References:

- [1]. Kumar A, Negi G, Sharma S S. JSH 23 targets nuclear factor kappa B and reverses various deficits in experimental diabetic neuropathy: effect on neuroinflammation and antioxidant defense [J].

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Diabetes, Obesity and Metabolism, 2011, 13(8): 750-758.

[2]. Guo Z, Chen W, Dai G, et al. Cordycepin suppresses the migration and invasion of human liver cancer cells by downregulating the expression of CXCR4[J]. International journal of molecular medicine, 2020, 45(1): 141-150.

[3]. Ozkok A, Ravichandran K, Wang Q, et al. NF- κ B transcriptional inhibition ameliorates cisplatin-induced acute kidney injury (AKI)[J]. Toxicology letters, 2016, 240(1): 105-113.

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