Nimesulide

Cat. No.: HY-B0363

CAS No.: 51803-78-2

Molecular Formula: $C_{13}H_{12}N_2O_5S$

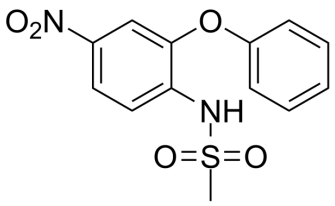
Molecular Weight: 308.31

Target: COX

Pathway: Immunology/Inflammation

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (324.35 mM)

H_2O : < 0.1 mg/mL (insoluble)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.2435 mL	16.2174 mL	32.4349 mL
5 mM	0.6487 mL	3.2435 mL	6.4870 mL
10 mM	0.3243 mL	1.6217 mL	3.2435 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 (8.11 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.11 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Nimesulide is a selective COX-2 inhibitor, with IC_{50}s of 70 nM-70 μM in a time-dependent manner, but it shows no effect on COX-1 (IC_{50} >100 μM). Nimesulide has potent anti-inflammatory, analgesic and antipyretic properties.

IC_{50} & Target

COX-2

0.07-70 μM (IC_{50})

In Vitro

Nimesulide is a selective COX-2 inhibitor, with IC_{50}s of 70 nM-70 μM in a time-dependent manner, but it shows weak effect on COX-1 (IC_{50} >100 μM)[1]. Nimesulide (10 μM) effectively decreases VEGF in endometrium cancer cells, and shows no effect

	on that in normal cells. Nimesulide (10 and 50 μM) dramatically decreases MCP-1 levels in normal cell, and such an effect is also observed with 10 μM in cancer cells. In addition, Nimesulide (50 μM) potently affects IL-8 level in normal cells, but causes no changes in cancer cells[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Nimesulide (3 and 10 mg/kg, i.p.) effectively blocks fever induced by i.p. injection of LPS in rats. Nimesulide (3 mg/kg, i.p.) potently reduces fever response induced by IL-1β, IL-6 or TNF-α, but does not prevent the initial rise in the febrile response induced by arachidonic acid. Nimesulide also significantly reduces PGE2 levels and PGF2α levels in the cerebrospinal fluid of the LPS-stimulated animals, and inhibits the increase in plasma TNF-α by 97%[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [3]	5×10^5 viable cells are carefully dislodged with sterile Pasteur pipettes, transferred into new flasks and incubated with two different doses of Nimesulide (10 and 50 μM) for another 24 h. Incubations for different doses of Nimesulide are repeated three times. The culture supernatant is then collected and stored in small aliquots at -70°C until studied. VEGF, MCP-1 and IL-8 concentrations are determined by sandwich quantitative enzyme immunoassay (ELISA) using commercial kits[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Rats[2] In the initial experiments, rats are pre-treated with intraperitoneal injections of 1, 3 or 10 mg/kg doses of Nimesulide, diluted in a 5% cremophor vehicle, or 2 mg/kg of indomethacin diluted in tris(hydroximetyl)-aminomethane-HCl (TRIS), pH 8.2, 30 min prior to an i.p. injection of LPS (50 μg/kg). Control animals receive the appropriate vehicle plus saline (1 mL/200 g, i.p.). The dose of 3 mg/kg of Nimesulide is chosen for the remaining experiments. In another set of experiments, rats are pretreated with an i.p. injection of Nimesulide (3 mg/kg) or indomethacin (2 mg/kg), diluted in the appropriate vehicles, 30 min prior to an i.c.v. injection (2 μL over 1 min) of IL-1β (3.12 ng), IL-6 (300 ng), TNF-α (250 ng), arachidonic acid (50 μg), MIP-1α (500 ng), PGE2 (250 ng), PGF2α (250 ng), CRF (2 μg) or ET-1 (1 pmol). Control animals receive the appropriate vehicles (1 mL/200 g, i.p.) and sterile saline (2 μL over 1 min, i.c.v.). All the drugs are injected between 10:00 and 11:00 AM to avoid circadian rhythm variations[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Hazard Mater. 2015 May 30;289:18-27.
- J Ethnopharmacol. 2023 Mar 9;309:116357.
- Chem-Biol Interact. 2021, 109425.
- J Phys Chem Solids. 2017 October;109:117-123.
- Biotechnol Bioeng. 2021 Sep 3.

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REFERENCES

[1]. Vago T, et al. Effect of nimesulide action time dependence on selectivity towards prostaglandin G/H synthase/cyclooxygenase activity. Arzneimittelforschung. 1995 Oct;45(10):1096-8.

[2]. Werner MF, et al. Nimesulide-induced antipyresis in rats involves both cyclooxygenase-dependent and independent mechanisms. Eur J Pharmacol. 2006 Aug 14;543(1-3):181-9.

[3]. Genç S, et al. The effect of COX-2 inhibitor, nimesulide, on angiogenetic factors in primary endometrial carcinoma cell culture. Clin Exp Med. 2007 Mar;7(1):6-10.

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