Nafamostat mesylate

Cat. No.: HY-B0190A

CAS No.: 82956-11-4

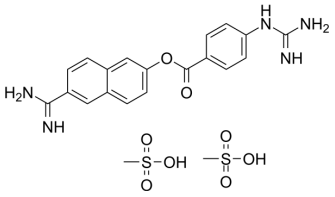
Molecular Formula: $C_{21}H_{25}N_5O_8S_2$

Molecular Weight: 539.58

Target: Flavivirus; TNF Receptor; NF-κB; Apoptosis; Ser/Thr Protease

Pathway: Anti-infection; Apoptosis; NF-κB; Metabolic Enzyme/Protease

Storage: 4°C, sealed storage, away from moisture and light
* In solvent : -80°C, 1 year; -20°C, 6 months (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (92.66 mM; Need ultrasonic)
H_2O : 33.33 mg/mL (61.77 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.8533 mL	9.2665 mL	18.5329 mL
5 mM	0.3707 mL	1.8533 mL	3.7066 mL
10 mM	0.1853 mL	0.9266 mL	1.8533 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 (4.63 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (4.63 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.63 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Nafamostat mesylate (FUT-175), an anticoagulant, is a synthetic serine protease inhibitor. Nafamostat mesylate has anticancer and antivirus effect. Nafamostat mesylate induce apoptosis by up-regulating the expression of tumor necrosis factor receptor-1 (TNFR1). Nafamostat mesylate can be used in the development of the pathological thickening of the arterial wall[1][2][3][4].

IC_{50} & Target

I-kappaBalpha

In Vitro

Nafamostat mesylate (10-80 μg/mL, 3-48 h) inhibits NF-κB activity by blocking IκBα phosphorylation in MDAPanc-28 cells[1].
Nafamostat mesylate (80 μg/mL, 24-48 h) induces apoptosis by up-regulating the expression of tumor necrosis factor receptor-1 (TNFR1) in MDAPanc-28 cells[1].
Nafamostat mesylate (0.1-10 μM, 24 h) has suppressive effect on invasiveness in Panc-1 cells[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	MDAPanc-28 cells
Concentration:	80 μg/mL
Incubation Time:	24 h, 48 h
Result:	Substantially reduced the cell viability of MDAPanc-28 cells at both 24 hours and 48 hours.

Cell Invasion Assay[2]

Cell Line:	Inhibited NF-κB DNA-binding activity and the degradation of IκBα in a dose-dependent manner as well as in a time-dependent manner. Inhibited NF-κB DNA-binding activity and the degradation of IκBα in a dose-dependent manner as well as in a time-dependent manner. Panc-1 cells
Concentration:	0.1 μM, 1 μM, 10 μM
Incubation Time:	24 h
Result:	Observed significant inhibition in Panc-1-Try clones at concentrations as low as 0.1 mM.

Western Blot Analysis[1]

Cell Line:	MDAPanc-28 cells
Concentration:	10 μg/mL, 20 μg/mL ,40 μg/mL, 80 μg/mL
Incubation Time:	3 h, 8 h, 24 h, 48 h
Result:	Inhibited NF-κB DNA-binding activity and the degradation of IκBα in a dose-dependent manner as well as in a time-dependent manner. Inhibited phosphorylation of IκBα in a time-dependent manner.

In Vivo

Nafamostat mesylate (10 mg/kg, Intraperitoneal injection, once a day for 18 days) exhibits favourable antiviral effects against Zika virus (ZIKV) infection in A129 mice[3].
Nafamostat mesylate (0.5-2.0 mg/mL (dissolved in saline), Intraperitoneal injection, once a day for 7 consecutive days) has inhibitory effect on neointimal formation after balloon injury of the rat carotid wall[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	A129 mice[3]
Dosage:	10 mg/kg
Administration:	Intraperitoneal injection (i.p.)
Result:	Exhibit delayed lethality and improved survival (40%).

Animal Model:	Balloon injury of the rat carotid wall [4]
Dosage:	0.5 mg/mL, 1 mg/mL, 2 mg/mL (dissolved in saline)
Administration:	Intraperitoneal injection (i.p.)
Result:	Showed smaller ratios of the neointima/medial area. Showed positive but reduced immunoreactivity of the cells in the neointimal.

CUSTOMER VALIDATION

- Cell Res. 2020 Mar;30(3):269-271.
- Nucleic Acids Res. 2021 Jan 8;49(D1):D1113-D1121.
- Nat Chem Biol. 2022 Jun 8.
- Antiviral Res. 2023 Apr 17;105606.
- Cells. 2022, 11(3), 319.

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REFERENCES

[1]. Uwagawa T, et al. Mechanisms of synthetic serine protease inhibitor (FUT-175)-mediated cell death [J]. Cancer: Interdisciplinary International Journal of the American Cancer Society, 2007, 109(10): 2142-2153.

[2]. Tajima H, et al. Enhanced invasiveness of pancreatic adenocarcinoma cells stably transfected with cationic trypsinogen cDNA [J]. International journal of cancer, 2001, 94(5): 699-704.

[3]. Yan Y, et al. Nafamostat mesylate as a broad-spectrum candidate for the treatment of flavivirus infections by targeting envelope proteins [J]. Antiviral research, 2022, 202: 105325.

[4]. Sawada M, et al. Prevention of neointimal formation by a serine protease inhibitor, FUT-175, after carotid balloon injury in rats [J]. Stroke, 1999, 30(3): 644-650.

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