Product Data Sheet

Product Name: Nafamostat
Cat. No.: GC17676

Chemical Properties

Cas. No.	81525-10-2		
Chemical Name	(6-carbamimidoylnaphthalen-2-yl) 4-(diaminomethylideneamino)benzoate		
SMILES	C1=CC(=CC=C1C(=O)OC2=CC3=C(C=C2)C=C(C=C3)C(=N)N)N=C(N)N		
Formula	$C_{19}H_{17}N_5O_2$	M.Wt	347.37
Solubility	Soluble in DMSO	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ 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	Nafamostat		

Protocol

Cell experiment [1]:

Cell lines	SW620 cells

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Preparation Method	CRC cells were treated with Nafamostat Mesylate (80μg/mL; Nafamostat Mesylate group), ionizing radiation (ionizing radiation group, IR), both Nafamostat Mesylate (80μg/mL) and ionizing radiation (combination group, IR + Nafamostat Mesylate), or vehicle-only (control group, CTR) for the appropriate time. Cells of the IR and combination groups received 2 or 5Gy IR for the cell proliferation assay, and 5Gy IR for the other analyses. In the combination group, the cells were treated with nafamostat mesilate for three hours before IR.
Reaction Conditions	80μg/mL; 3h
Applications	Nafamostat Mesylate prevents NF-κB activation and induces apoptosis in irradiated colorectal cancer cells.

Animal experiment [2]:

Animal models	Choline deficient ethionine diet mice model
Preparation Method	Two hundred and sixty young female CD-I mice weighing 12-14g were used. They were allowed an ordinary pellet diet and tap water ad libitum prior to the experiments. After an initial 24-hour fast, they were fed a cholinedeficient diet enriched with 0.5% DZ.-ethionine for 24h. They were again fasted for 24h, then fed a regular laboratory diet and tap water ad libitum for the following 3 days. Up to 20 mice were kept in each cage. All the mice fed the choline deficient ethionine (CDE) diet were then divided into the following two groups: (a) control animals (CDE group) - only 0.2mL of saline was injected subcutaneously; (b) Nafamostat Mesylate-treated animals (Nafamostat Mesylate group) - after the beginning of the CDE diet, Nafamostat Mesylate was injected subcutaneously at a dose of 20mg/kg in 0.2mL of saline. In addition to these two groups, normal mice were also used as a pure control group.

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Dosage form	20mg/kg; ip; 5d
Applications	Nafamostat Mesylate inhibited the redistribution of cathepsin B activity and the activation of trypsinogen.

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References:
[1]. Sugano H, Shirai Y, Horiuchi T, et al. Nafamostat mesilate enhances the radiosensitivity and reduces the radiation-induced invasive ability of colorectal cancer cells[J]. Cancers, 2018, 10(10): 386.
[2]. Hirano T, Takeuchi S. A New Protease Inhibitor, Nafamostat Mesilate (FUT-175), Protects Pancreatic Acinar Cells in CDE-Diet-Induced Pancreatitis in Mice[J]. Digestive surgery, 1993, 10(4): 182-188.

Background

Product Data Sheet

Background

Nafamostat is a synthetic serine protease inhibitor [1]. Nafamostat Mesylate reduces inflammatory responses by inhibiting the complement system, reducing cytokine release, and preventing pancreatic enzyme activation [2]. Nafamostat Mesylate is commonly used to treat acute pancreatitis [3-4].

In SW620 cells, Nafamostat Mesylate (80μg/mL; 3h) prevents NF-κB activation and induces apoptosis in irradiated colorectal cancer cells [5]. In MSTO-211H cells, cell viability was significantly reduced after Nafamostat Mesilate (10μM; 48h) treatment [6]. In YCU-L891 and YCU-H891 cells, Nafamostat Mesylate (10μM; 48h) inhibited the proliferation of two HNSCC cell lines [7].

In choline deficient ethionine diet mice model, Nafamostat Mesylate (20mg/kg; ip; 5d) inhibited the redistribution of cathepsin B activity and the activation of trypsinogen [8]. In xenograft pancreatic cancer mice model, Nafamostat Mesylate (30μg/g; ip; 6 weeks) enhances oxaliplatin-induced tumor growth inhibition [9].

References:

[1]. Mellgren K, Skogby M, Friberg L G, et al. The influence of a serine protease inhibitor, nafamostat mesilate, on plasma coagulation, and platelet activation during experimental extracorporeal life support (ECLS)[J]. Thrombosis and haemostasis, 1998, 79(02): 342-347.
[2]. He Q, Wei Y, Qian Y, et al. Pathophysiological dynamics in the contact, coagulation, and complement systems during sepsis: Potential targets for nafamostat mesilate[J]. Journal of intensive medicine, 2024, 4(04): 453-467.
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[4]. Keck T, Balcom J H, Antoniu B A, et al. Regional effects of nafamostat, a novel potent protease and complement inhibitor, on severe necrotizing pancreatitis[J]. Surgery, 2001, 130(2): 175-181.
[5]. Sugano H, Shirai Y, Horiuchi T, et al. Nafamostat mesilate enhances the radiosensitivity and reduces the radiation-induced invasive ability of colorectal cancer cells[J]. Cancers, 2018, 10(10): 386.

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[6]. Sutoh T, Fukuda I, Kimura D, et al. Nafamostat mesilate (FUT-175) inhibits cell growth and invasion of malignant pleural mesothelioma cell line, MSTO-211H[J]. Hirosaki Medical Journal, 2010, 61(1): 19-25.
[7]. Yamashita Y, Ishiguro Y, Sano D, et al. Antitumor effects of Nafamostat mesilate on head and neck squamous cell carcinoma[J]. Auris Nasus Larynx, 2007, 34(4): 487-491.
[8]. Hirano T, Takeuchi S. A New Protease Inhibitor, Nafamostat Mesilate (FUT-175), Protects Pancreatic Acinar Cells in CDE-Diet-lnduced Pancreatitis in Mice[J]. Digestive surgery, 1993, 10(4): 182-188.
[9]. Gocho T, Uwagawa T, Furukawa K, et al. Combination chemotherapy of serine protease inhibitor nafamostat mesilate with oxaliplatin targeting NF-κB activation for pancreatic cancer[J]. Cancer Letters, 2013, 333(1): 89-95.

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