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Camostat Mesilate

Technical Data

Molecular Weight	494.52	**Storage**	3 years	-20°C	powder
Formula	$C_{20}H_{22}N_4O_5.CH_4O_3S$		2 years	-80°C	in solvent
CAS No.	59721-29-8	**Synonyms**	FOY-305		
Chemical Name	Benzeneacetic acid, 4-[[4-[(aminoiminomethyl)amino]benzoyl]oxy]-, 2-(dimethylamino)-2-oxoethyl ester, methanesulfonate (1:1)				
Solubility (25°C) *	In vitro	DMSO	99 mg/mL (200.19 mM)		
		Water	10 mg/mL (20.22 mM)		
		Ethanol	Insoluble		
	In vivo（should be freshly prepared each time）				

* <1 mg/ml means slightly soluble or insoluble.
* Please note that Selleck tests the solubility of all compounds in-house, and the actual solubility may differ slightly from published values. This is normal and is due to slight batch-to-batch variations.

Preparing Stock Solutions

Volume / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.0222 mL	10.1108 mL	20.2216 mL
5 mM	0.4044 mL	2.0222 mL	4.0443 mL
10 mM	0.2022 mL	1.0111 mL	2.0222 mL
50 mM	0.0404 mL	0.2022 mL	0.4044 mL

Biological Activity

Description	Camostat is a trypsin-like protease inhibitor, inhibits airway epithelial sodium channel (ENaC) function with **IC50** of 50 nM, less potent to trpsin, prostasin and matriptase.
Targets	epithelial sodium channel (ENaC) [1] 50 nM
In vitro	Camostat (30 μM) prolongs attenuation of ENaC function in human airway epithelial cell models that is reversible upon the addition of excess trypsin. [1] Camostat mesilate (500 mM) inhibits generation of TGF-beta by suppressing plasmin activity and reduces the activity of TGF-beta, which blocks in vitro activation of HSCs. [2] Camostat mesilate (20 mM) combined with insulin results a significant hypoglycemic effect following large intestinal administration. Camostat mesilate (20 mM) is effective in reducing insulin degradation in both small and large intestinal homogenates of rats. [3] Camostat mesilate (2 mM) inhibits MCP-1 and TNF- production in activated rat monocytes. Camostat mesilate (2 mM) inhibits proliferation and MCP-1 production of cultured rat PSCs. [4]
In vivo	Camostat (100 μg/kg i.t.) induces a potent and prolonged attenuation of ENaC activity in the guinea pig trachea. [1] Camostat mesilate (1-2 mg/g of diet) markedly attenuats an increase in hepatic plasmin and TGF-beta levels, HSC activation, and hepatic fibrosis without apparent systemic or local side effects in porcine serum–treated rats. [2] Camostat mesilate (1 mg/g) prevents pancreatic atrophy and improves pancreatic exocrine function of rat chronic pancreatitis induced by DBTC. Camostat mesilate (1 mg/g) inhibits chronic inflammation and pancreatic fibrosis induced by DBTC. Camostat mesilate (1 mg/g) inhibits the development of pancreatic fibrosis and PSCs activation in the pancreas induced by DBTC. Camostat mesilate (1 mg/g) suppresses monocytes infiltration and inhibits MCP-1 expression both in serum and in pancreatic tissue. [4] Camostat mesilate (100 mg/kg) significantly increases the body weight and pancreatic wet weight, and it significantly inhibits inflammatory changes and fibrosis of the pancreas through the suppression of gene expressions of PAP, p8, and cytokines in rat chronic pancreatitis. [5]
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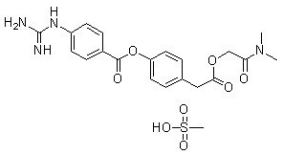
Protocol *(Only for Reference)*

Kinase Assay: [1]

Binding assay	Enzymes are diluted into the appropriate activity buffer (prostasin, matriptase, and trypsin: 15 mM HEPES, pH 7.4, 25 mM NaCl, and 0.5% CHAPS; furin: 15 mM HEPES, pH 7.6, 25 mM NaCl, 1 mM $CaCl_2$, 1 mM β-mercaptoethanol, and 0.5% CHAPS; HNE: 15 mM HEPES, pH 7.6, 150 mM NaCl, and 0.75% CHAPS), added to compound solutions, and incubated at room temperature for 20 min. Fluorogenic substrates are mixed with activity buffer and added to the enzyme/compound solution after the 20-min incubation. Fluorescence emission is quantified on a Gemini plate reader at 37°C. Excitation wavelength is 380 nm, emission wavelength is 450 nm, and cut-off is 435 nm. Ki values are determined through analysis of progression curves using BioKin PlateKi.

Animal Study: [2]

Chemical Structure



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Animal Models	male Wistar rats
Formulation	Saline
Dosages	2 mg/g
Administration	Diet

References:

[1] Coote K, et al. J Pharmacol Exp Ther, 2009, 329(2), 764-774.
[2] Okuno M, et al. Gastroenterology, 2001, 120(7), 1784-1800.
[3] Yamamoto A, et al. Pharm Res, 1994, 11(10), 1496-1500.
[4] Gibo J, et al. Lab Invest, 2005, 85(1), 75-89.
[5] Su SB, et al. Pancreas, 2001, 23(2), 134-140.