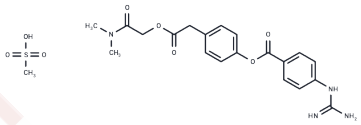
Data Sheet (Cat.No.T2391)

TargetMol

Camostat mesylate

Chemical Properties



CAS No. : 59721-29-8

Formula: C21H26N4O8S

Molecular Weight: 494.52

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	Camostat mesylate (FOY-S980) is a trypsin-like protease inhibitor and inhibits airway epithelial sodium channel (ENaC) function.
Targets(IC50)	SARS-CoV,Serine Protease,Serine/threonin kinase,Sodium Channel
In vitro	Camostat mesilate, at a concentration of 1 mg/g, is effective in preventing pancreatic atrophy and improving exocrine function in rats with DBTC-induced chronic pancreatitis. This compound also inhibits chronic inflammation and fibrosis in the pancreas caused by DBTC. It suppresses the development of pancreatic fibrosis and PSC activation, as well as monocyte infiltration and the expression of MCP-1 in serum and pancreatic tissues. When administered at a dose of 100 mg/kg, Camostat mesilate significantly increases body and pancreatic wet weight, and effectively reduces inflammatory changes and fibrosis in the pancreas by inhibiting gene expression of PAP, p8, and cytokines in chronic pancreatitis. In rats treated with pig serum, Camostat mesilate (1-2 mg/g in diet) notably decreases the levels of hepatic plasmin and TGF-β and inhibits HSC activation and liver fibrosis, without noticeable systemic or local side effects. Additionally, at a dose of 100 μg/kg intratracheally, Camostat prolongs the attenuation of ENaC activity in guinea pig tracheas.
In vivo	At a concentration of 2 mM, Camostat mesilate inhibits the production of MCP-1 and TNF-α in activated rat monocytes and the proliferation of rat pancreatic stellate cells (PSCs) along with MCP-1 production. In the presence of 20 mM Camostat mesilate, there is a notable reduction in blood glucose levels when combined with insulin and administered via the large intestine, effectively decreasing insulin degradation in the rat's small intestine and its homogenate. A 30 μM concentration of Camostat prolongs the decay of ENaC function in a human respiratory epithelial cell model, which can be reversed by adding excess trypsin. At 500 mM, Camostat mesilate inhibits the generation of TGF-β by suppressing plasmin activity and reduces the activity of TGF-beta, thereby blocking the ex vivo activation of hepatic stellate cells (HSC).

Solubility Information

Solubility	DMSO: 250 mg/mL (505.54 mM),Sonication is recommended. H2O: 49.5 mg/mL (100.1 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 2 mg/mL (4.04 mM),Sonication is recommended. *Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.*

Preparing Stock Solutions

	1mg	5mg	10mg
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

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Coote K, et al. J Pharmacol Exp Ther, 2009, 329(2), 764-774.
Zhang S, Gao C, Das T, et al. The spike-ACE2 binding assay: An in vitro platform for evaluating vaccination efficacy and for screening SARS-CoV-2 inhibitors and neutralizing antibodies. Journal of Immunological Methods. 2022: 113244.
Okuno M, et al. Gastroenterology, 2001, 120(7), 1784-1800.
Yamamoto A, et al. Pharm Res, 1994, 11(10), 1496-1500.
Gibo J, et al. Lab Invest, 2005, 85(1), 75-89.
Su SB, et al. Pancreas, 2001, 23(2), 134-140.