Data Sheet (Cat.No.T214657)

TargetMol

UK122 hydrochloride

Chemical Properties

CAS No. :

Formula: C17H14ClN3O2

Molecular Weight: 327.77

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	UK122 hydrochloride is a potent and selective inhibitor of urokinase-type plasminogen activator (uPA), exhibiting an IC50 value of 0.2 μM. UK122 hydrochloride demonstrates minimal inhibitory activity against tissue-type plasminogen activator (tPA), plasmin, thrombin, and trypsin, with IC50 values greater than 100 μM for all. As a 4-oxazolidinone analog, UK122 hydrochloride inhibits cancer cell migration and invasion. The selectivity profile of UK122 hydrochloride supports the application of UK122 hydrochloride in research focused on protease biology, extracellular matrix remodeling, tumor cell invasion, and metastatic mechanisms.
Targets(IC50)	Serine Protease
In vitro	Method: UK122 parent compound was tested in an indirect cell-free human urokinase-type plasminogen activator assay based on uPA-mediated plasminogen activation and chromogenic-substrate cleavage. Tissue-type plasminogen activator, plasmin, thrombin and trypsin were included as a selectivity panel. Rresult: UK122 inhibited human uPA with an IC50 of 0.2 μM. The IC50 values against tissue-type plasminogen activator, plasmin, thrombin and trypsin were all greater than 100 μM, indicating more than 500-fold biochemical selectivity for uPA.[1] Method: CFPAC-1 human pancreatic adenocarcinoma cells were exposed to UK122 parent compound. Cell growth and morphology were assessed after treatment for up to 48 h; fibronectin-associated radial migration and Matrigel-coated Boyden-chamber invasion were evaluated after 24 h. Migration and invasion experiments included treatment at 100 μM. Rresult: UK122 showed a cell-growth IC50 greater than 100 μM and did not markedly alter cellular morphology at 100 μM. At 100 μM, it inhibited CFPAC-1 cell migration by approximately 80% and Matrigel invasion by approximately 68%.[1] Method: Stable endoglin-knockdown HTR-8/SVneo human extravillous trophoblast cells, which exhibited increased uPA expression and motility, were treated with 100 μM UK122 and assessed in a cell-migration assay. Rresult: Treatment with 100 μM UK122 markedly inhibited the increased migration of endoglin-knockdown HTR-8/SVneo cells, supporting the involvement of uPA activity in the enhanced motile phenotype. [2] Method: Mouse TSC2-null tumor cells were serum-starved in DMEM containing 0.1% BSA for 24 h and then exposed to 20 nM rapamycin or vehicle for an additional 18 h. Cells were detached, washed and resuspended in medium with or without UK122 before transwell migration and Matrigel-invasion assays. Each condition included three wells

In vitro	and three microscopic fields per well. Rresult: UK122 abolished the rapamycin-induced increases in migration and invasion of TSC2-null tumor cells, supporting dependence of the rapamycin-enhanced invasive phenotype on uPA catalytic activity.[3]
In vivo	Method: Eight-week-old male C57BL/6J mice received 2% dextran sulfate sodium in drinking water for 7 days to induce colitis. UK122 was dissolved in DMSO and saline and administered intraperitoneally at 2 or 4 mg/kg once daily from day 1 to day 7; vehicle controls were included. Eighteen mice were used per treatment group and animals were sacrificed on day 8. Rresult: UK122 dose-dependently reduced colitis severity. At 4 mg/kg, the disease-activity index decreased from 6.00±0.40 to 3.21±0.50 (p=0.0008), and the histological score decreased from 18.21±2.91 to 9.00±1.81 (p=0.0157). Colorectal RANTES decreased from 111.64±35.80 to 25.49±3.86 (p=0.0143).[4]

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	3.0509 mL	15.2546 mL	30.5092 mL
5 mM	0.6102 mL	3.0509 mL	6.1018 mL
10 mM	0.3051 mL	1.5255 mL	3.0509 mL
50 mM	0.061 mL	0.3051 mL	0.6102 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

Zhu M, et al. Identification of a novel inhibitor of urokinase-type plasminogen activator. Mol Cancer Ther. 2007;6 (4):1348-1356.
Mano Y, et al. The Loss of Endoglin Promotes the Invasion of Extravillous Trophoblasts. Endocrinology. 2011;152 (11):4386-4394.
Stepanova V, et al. Urokinase-type plasminogen activator (uPA) is critical for progression of tuberous sclerosis complex 2 (TSC2)-deficient tumors. J Biol Chem. 2017;292(50):20528-20543.
Kida Y, et al. Urokinase-type plasminogen activator blockade ameliorates experimental colitis in mice. Sci Rep. 2023;13(1):2899.