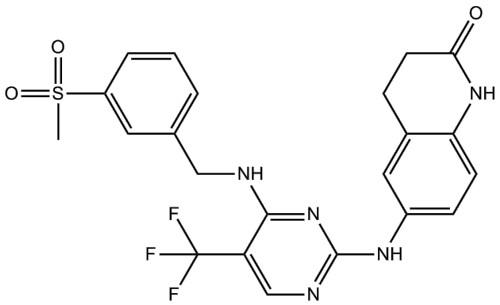
Product Data Sheet

Product Name: PF-573228
Cat. No.: GC11107

Chemical Properties

Cas. No.	869288-64-2		
Chemical Name	6-[[4-[(3-methylsulfonylphenyl)methylamino]-5-(trifluoromethyl)pyrimidin-2-yl]amino]-3,4-dihydro-1H-quinolin-2-one		
SMILES	CS(=O)(=O)C1=CC=CC(=C1)CNC2=NC(=NC=C2C(F)(F)F)NC3=CC4=C(C=C3)NC(=O)CC4		
Formula	$C_{22}H_{20}F_3N_5O_3S$	M.Wt	491.49
Solubility	≥ 166.6mg/mL in DMSO	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C 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



Protocol

Cell experiment [1]:	
Cell lines	H2596 cells (human malignant pleural mesothelioma cell line), MiaPaca 2 cells and Capan 1 cells (human pancreatic ductal adenocarcinoma cell lines)

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Preparation Method	H2596, MiaPaca 2 and Capan 1 cells were cultured in RPMI 1640 medium supplemented with 10% FBS, L-glutamine and 1% penicillin-streptomycin at 37°C, 5% CO_2. Cells were treated with PF-573228 at 1-10μM for 48 or 72h. After treatment, cell viability, apoptosis, cell cycle was measured.
Reaction Conditions	1-10μM; 24-72h
Applications	PF-573228 decreased cell viability in H2596, MiaPaca 2 and Capan 1 cells in a dose-dependent manner. PF-573228 arrested H2596, MiaPaca 2 and Capan 1 cells in G2/M phase. PF-573228 reduced cleaved PARP levels and decreased Cyclin D1 protein in H2596, MiaPaca 2 and Capan 1 cells. PF-573228 inhibited phosphorylation of FAK at Tyr397 and downstream paxillin phosphorylation. PF-573228 decreased anchorage-independent colony number and colony size in H2596, MiaPaca 2 and Capan 1 cells.
Animal experiment [2]:	
Animal models	Male NKG mice (6 weeks old, 18-20g) bearing subcutaneous xenografts of MGC803-CPZ-OENC or MGC803-CPZ-OE gastric cancer cells
Preparation Method	When tumor volume reached ~100mm³, mice were randomly assigned to four groups (CPZ-OENC control, CPZ-OENC+PF-573228, CPZ-OE control, CPZ-OE+PF-573228, n=5 per group). Treatment groups received intraperitoneal injections of 5mg/kg PF-573228 (dissolved in DMSO) 5 days per week for 35 days; control groups received DMSO only. Tumor length and width were measured every 3 days to calculate volume ($L \times W^2/2$); mice were euthanized on day 35 and tumors excised for final volume/weight recording.
Dosage form	5mg/kg; i.p.; 5 days/week for 35 days

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Applications	PF-573228 inhibited subcutaneous xenograft tumor growth in both MGC803-CPZ-OENC and MGC803-CPZ-OE models. PF-573228 produced a greater reduction in tumor volume and weight in the CPZ-OE group than in the CPZ-OENC group.

References:

[1] Kanteti R, Mirzapoiazova T, Riehm JJ, et al. Focal adhesion kinase a potential therapeutic target for pancreatic cancer and malignant pleural mesothelioma. Cancer Biol Ther. 2018;19(4):316-327.

[2] Luo R, Huang W, Wang H, et al. CPZ promotes the proliferation of gastric cancer by activating the Wnt pathway through binding with DVL2. Discover Oncol. 2025;16:2085.

Background

PF-573228 is an ATP-competitive focal adhesion kinase (FAK) inhibitor (IC_{50}=4nM). PF-573228 binds to the FAK kinase domain and inhibits FAK autophosphorylation at Tyr397. PF-573228 disrupts the formation of the FAK-Src complex and downstream signaling for haptotaxis, cell migration, and focal adhesion turnover. PF-573228 can be used in studies related to tumors (such as small cell lung cancer, endocrine-resistant breast cancer, glioblastoma), cardiac fibrosis, skin fibrosis/hypertrophic scars[1-4].

In vitro, co-treatment of HepG2, Huh7, and MHCC97H cells with PF-573228 and 2.5μM Sorafenib for 1-48 hours inhibited Sorafenib-induced upregulation of FAK phosphorylation at Tyr397. PF-573228 synergized with Sorafenib to reduce HCC cell viability and suppress colony formation[5]. Treatment of H2596 cells, MiaPaca 2 cells, and Capan 1 cells with 1-10μM PF-573228 for 24-72 hours dose-dependently reduced cell viability, induced cell cycle arrest, and increased the proportion of apoptotic cells[6]. Treatment of ARK-1 cells with 10μM PF-573228 for 1 hour reduced FAK phosphorylation levels at Tyr397. Treatment of ARK-1 cell-formed 3D spheroids with 10μM PF-573228 for 48 hours significantly reduced spheroid perimeter, decreased Cyclin D1 levels, and reduced cell migration speed and cumulative migration distance[7].

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In vivo, NKG mice bearing subcutaneous xenografts of MGC803-CPZ-OENC or MGC803-CPZ-OE cells reaching approximately 100mm³ received daily intraperitoneal injections of 5mg/kg PF-573228 for 5 days per week for 35 consecutive days. PF-573228 inhibited xenograft tumor growth, with a more pronounced inhibitory effect in the CPZ-OE group compared to the CPZ-OENC group[8]. C57B/6 mice received a single intratracheal instillation of 3U/kg Bleomycin Sulfate to establish a pulmonary fibrosis model, followed by daily intraperitoneal injections of 50mg/kg PF-573228 for 21 consecutive days. PF-573228 reduced lung tissue fibrosis, decreased whole-lung Fibronectin and procollagen-1 protein levels, and reduced α-SMA expression[9]. On day 3 after optic nerve transection, Sprague-Dawley rats received a single intravitreal injection of 25mM PF-573228 (4μL). PF-573228 reduced the density of Fluoro-Gold retrograde-labeled retinal ganglion cells in the retinal ganglion cell layer and decreased the number of surviving injured retinal ganglion cells[10].

References:

[1] Slack-Davis JK, Martin KH, Tilghman RW, et al. Cellular characterization of a novel focal adhesion kinase inhibitor. J Biol Chem. 2007 May 18;282(20):14845-52.

[2] Maher S, Bayachou M, Fu P, et al. Focal adhesion kinase activation is involved in contractile stimulation-induced detrusor muscle contraction in mice. Eur J Pharmacol. 2023 Aug 5;952:175807.

[3] Tsang VSK, Riccio F, Wilson AS, et al. A human iPSC-based neural spheroid platform for modelling glioblastoma infiltration using high-content imaging. Sci Rep. 2025 Dec 13;16(1):1223.

[4] Zhang H, Schaefer A, Wang Y, et al. Gain-of-Function *RHOA* Mutations Promote Focal Adhesion Kinase Activation and Dependency in Diffuse Gastric Cancer. Cancer Discov. 2020 Feb;10(2):288-305.

[5] Feng J, Ye L, Chen C, et al. RhoE downregulation leads to enhanced cholesterol biosynthesis and sorafenib resistance in hepatocellular carcinoma. J Biol Chem. 2025 Dec;301(12):110918.

[6] Kanteti R, Mirzapoiazova T, Riehm JJ, et al. Focal adhesion kinase a potential therapeutic target for pancreatic cancer and malignant pleural mesothelioma. Cancer Biol Ther. 2018;19(4):316-327.

[7] Lopez-Mejia IC, Pijuan J, Navaridas R, et al. Oxidative stress-induced FAK activation contributes to uterine serous carcinoma aggressiveness. Mol Oncol. 2023;17(1):98-118.

Caution: Product has not been fully validated for medical applications. For research use only.
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[8] Luo R, Huang W, Wang H, et al. CPZ promotes the proliferation of gastric cancer by activating the Wnt pathway through binding with DVL2. Discover Oncol. 2025;16:2085.

[9] Zhao XK, Cheng Y, Cheng ML, et al. Focal Adhesion Kinase Regulates Fibroblast Migration via Integrin beta-1 and Plays a Central Role in Fibrosis. Sci Rep. 2016 Jan 14;6:19276.

[10] D'Onofrio PM. Integrin Activation and Focal Adhesion Kinase in the Survival and Regeneration of Injured Adult Rat CNS Neurons. MSc Thesis, University of Toronto. 2014.

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