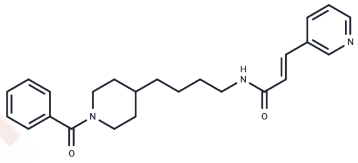
Data Sheet (Cat.No.T2644)

TargetMol

(E)-Daporinad

Chemical Properties



CAS No. : 658084-64-1

Formula: C24H29N3O2

Molecular Weight: 391.51

Storage: Store at low temperature
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	(E)-Daporinad (FK866) is a highly specific, non-competitive small molecule inhibitor of nicotinamide phosphoribosyltransferase (NAMPT) with potential anti-tumor and anti-angiogenic activities with an IC50 value of 0.09 nM.
Targets(IC50)	Autophagy,NAMPT,Transferase
In vitro	**METHODS**: Hepatocytes were pretreated with (E)-Daporinad（FK866）(100 nM, 30 minutes) and then challenged with GaIN/LPS (G/L, 1 mg/mL /30 ng/mL, 24 hours) to observe whether (E)-Daporinad（FK866）could alleviate GaIN/LPS-induced hepatotoxicity. **RESULTS** Primary hepatocytes pretreated with (E)-Daporinad（FK866）showed a significant increase in autophagic activity after GaIN/LPS injury. (E)-Daporinad（FK866）could improve liver injury induced by GaIN/LPS and ConA. Its protective mechanism may involve its ability to induce autophagy by inhibiting JNK. [1]
In vivo	**METHODS**: (E)-Daporinad（FK866）(10 mg/kg, intraperitoneal injection) was administered to mice 24, 12, and 0.5 hours before treatment with GaIN/LPS and ConA to test the potential effect of (E)-Daporinad（FK866）on ALF in mice. **RESULTS** (E)-Daporinad（FK866）treatment reduced the mortality of mice treated with GaIN/LPS or ConA. (E)-Daporinad（FK866）treatment before and after treatment attenuated GaIN/LPS and ConA-induced ALF in mice, and (E)-Daporinad（FK866）pretreatment resulted in a better response to GaIN/LPS or ConA challenge. [1]
Kinase Assay	High Throughput Screening: FITC-MBM1 at 15 nM and menin at 150 nM in the FP buffer are mixed and incubated for 1h in the dark at room temperature. For point screening, the 0.2 μL of each compound (20 μM final concentration, 1% DMSO) is added to 20 μL of the aliquot of the protein-peptide mixture and incubated on 384-well plates in the dark at room temperature for 1h. In confirmation screening, the serial dilution plates with compounds in DMSO are prepared and used to titrate the menin-FITC-MBM1 complex. Change in fluorescence polarization is monitored at 525 nm after excitations at 495 nm using the PHERAstar microplate reader (BMG) and applied to determine IC50 values with the Origin 7.0 program.
Cell Research	For MTT assays, 0.5 × 106 cells/mL is plated in triplicate on 96-well plates. APO866 (0.01 nM-100 nM) is added in 50 μL of culture medium, with culture medium alone serving as control. After 72 or 96 hours of incubation, 15 μL of dye solution is added to each well and cells are incubated for an additional 4 hours. Stop solution (100 μL/well) is added

Cell Research	for 1 hour and the absorbance is read at 570 nm on a spectrophotometer. For trypan blue dye exclusion staining, 0.5 × 105 cells/well is grown in 6-well plates with 1 mL media in the absence or presence of APO866 for 96 hours. Cells from each sample are incubated with 10 μL trypan blue solution (at a 1:1 ratio [vol/vol] for 1 minute). Cell survival is determined by calculating proportion of live (unstained) cells. (Only for Reference)

Solubility Information

Solubility	DMSO: 257.5 mg/mL (657.71 mM),Sonication is recommended. Ethanol: 72 mg/mL (183.9 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+90% Saline: 0.1 mg/mL (0.26 mM),Solution. *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	2.5542 mL	12.7711 mL	25.5421 mL
5 mM	0.5108 mL	2.5542 mL	5.1084 mL
10 mM	0.2554 mL	1.2771 mL	2.5542 mL
50 mM	0.0511 mL	0.2554 mL	0.5108 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

Guo E, et al. FK866 attenuates acute hepatic failure through c-jun-N-terminal kinase (JNK)-dependent autophagy. Sci Rep. 2017 May 19;7(1):2206.

Xu B, Zhang P, Tang X, et al. Metabolic Rewiring of Kynurenine Pathway during Hepatic Ischemia-Reperfusion Injury Exacerbates Liver Damage by Impairing NAD Homeostasis. Advanced Science. 2022: 2204697.

Liu H Y, Wang F H, Liang J M, et al. Targeting NAD metabolism regulates extracellular adenosine levels to improve the cytotoxicity of CD8+ effector T cells in the tumor microenvironment of gastric cancer. Journal of Cancer Research and Clinical Oncology. 2022: 1-14

Cea M,et al. Targeting NAD+ salvage pathway induces autophagy in multiple myeloma cells via mTORC1 and extracellular signal-regulated kinase (ERK1/2) inhibition. Blood. 2012 Oct 25;120(17):3519-29.

Magnone M, et al. NAD+ levels control Ca2+ store replenishment and mitogen-induced increase of cytosolic Ca2+ by Cyclic ADP-ribose-dependent TRPM2 channel gating in human T lymphocytes. J Biol Chem. 2012 Jun 15;287(25): 21067-81.

Thakur BK,et al. Inhibition of NAMPT pathway by FK866 activates the function of p53 in HEK293T cells. Biochem Biophys Res Commun. 2012 Aug 3;424(3):371-7.

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