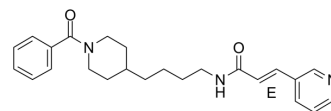


## (E)-Daporinad

<b>Cat. No.:</b>	HY-50876		
<b>CAS No.:</b>	658084-64-1		
<b>Molecular Formula:</b>	C <sub>24</sub> H <sub>29</sub> N <sub>3</sub> O <sub>2</sub>		
<b>Molecular Weight:</b>	391.51		
<b>Target:</b>	NAMPT; Autophagy		
<b>Pathway:</b>	Metabolic Enzyme/Protease; Autophagy		
<b>Storage:</b>	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	2 years
		-20°C	1 year



### SOLVENT & SOLUBILITY

#### In Vitro

DMSO : ≥ 50 mg/mL (127.71 mM)  
 H<sub>2</sub>O : < 0.1 mg/mL (insoluble)  
 \* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	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

Please refer to the solubility information to select the appropriate solvent.

#### In Vivo

- Add each solvent one by one: 20% SBE-β-CD in saline  
Solubility: 4 mg/mL (10.22 mM); Clear solution; Need ultrasonic
- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline  
Solubility: ≥ 2.5 mg/mL (6.39 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)  
Solubility: ≥ 2.5 mg/mL (6.39 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil  
Solubility: ≥ 2.5 mg/mL (6.39 mM); Clear solution

### BIOLOGICAL ACTIVITY

#### Description

(E)-Daporinad (FK866) is an effective inhibitor of nicotinamide phosphoribosyltransferase (NMPRTase; Nampt) with an IC<sub>50</sub> of 0.09 nM.

<b>IC<sub>50</sub> &amp; Target</b>	IC50: 0.09 nM (NMPRTase)
<b>In Vitro</b>	Nampt inhibition with (E)-Daporinad (FK866) induces significant NAD <sup>+</sup> intracellular reduction and selectively kills MM cells. (E)-Daporinad (FK866)-induced cell death is associated with inhibition of Nampt activity, rather than protein expression, and higher NAD <sup>+</sup> baseline levels in MM cells than normal PBMCs confer (E)-Daporinad (FK866) sensitivity. (E)-Daporinad (FK866) abrogates the survival advantage conferred by the bone marrow microenvironment <sup>[1]</sup> . (E)-Daporinad (FK866) prevents the [Ca <sup>2+</sup> ] <sub>i</sub> increase induced by different mitogens and reduces the Ca <sup>2+</sup> content of TG-responsive Ca <sup>2+</sup> stores in Jurkat and in activated PBLs. (E)-Daporinad (FK866) reduces the Ca <sup>2+</sup> content of TG-responsive Ca <sup>2+</sup> stores in Jurkat cells but not in Bcl2-Jurkat cells <sup>[2]</sup> . Inhibition of NAMPT by (E)-Daporinad (FK866), or inhibition of SIRT by nicotinamide decreases proliferation and triggered death of 293T cells involving the p53 acetylation pathway <sup>[3]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.
<b>In Vivo</b>	(E)-Daporinad (FK866) (30 mg/kg, i.p.) decreases the tumor burden in CB17-SCID mice, and the tumor tissue demonstrates a significant decrease in ERK phosphorylation and proteolytic cleavage of LC3 <sup>[1]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## PROTOCOL

<b>Cell Assay</b> <sup>[1]</sup>	MM1S cells (2×10 <sup>4</sup> cells/well) are cultured for 72 and 96 hours in BMSC-coated 96-well plates in the presence or absence of drug. DNA synthesis is measured by ( <sup>3</sup> H)-thymidine uptake, with ( <sup>3</sup> H)-thymidine added (0.5 μCi/well) during the last 8 hours of cultures. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
<b>Animal Administration</b> <sup>[1]</sup>	CB17-SCID mice (28-35 days old) are irradiated (200 cGy), and then inoculated subcutaneously in the right flank with 3×10 <sup>6</sup> MM1S cells in 100 μL RPMI 1640. After detection of tumor (2 weeks after the injection), 7 mice are treated intraperitoneally with either vehicle or (E)-Daporinad (FK866) (30 mg/kg body weight) twice a day for 4 days, repeated weekly over 3 weeks. Caliper measurements of the longest perpendicular tumor diameters are performed twice a week to estimate the tumor volume using the following formula: length×width <sup>2</sup> ×0.5. Tumor growth inhibition (TGI) is calculated. Animals are killed when tumors reach 2 cm <sup>3</sup> or the mice appear moribund. Survival is evaluated from the first day of treatment until death. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## CUSTOMER VALIDATION

- Sci Adv. 2023 Apr 14;9(15):eadf8522.
- Hepatology. 2022 Jul 11.
- Cell Death Differ. 2024 Jan 5.
- Redox Biol. 2024 Jan 3;69:103030.
- Acta Pharmacol Sin. 2023 Jun 5.

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## REFERENCES

- [1]. Cea M, et al. Targeting NAD<sup>+</sup> 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.
- [2]. Magnone M, et al. NAD<sup>+</sup> levels control Ca<sup>2+</sup> store replenishment and mitogen-induced increase of cytosolic Ca<sup>2+</sup> by Cyclic ADP-ribose-dependent TRPM2 channel gating in human T lymphocytes. J Biol Chem. 2012 Jun 15;287(25):21067-81.

**Caution: Product has not been fully validated for medical applications. For research use only.**

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