Z-FA-FMK

Cat. No.: HY-P0109A

CAS No.: 197855-65-5

Molecular Formula: $C_{21}H_{23}FN_2O_4$

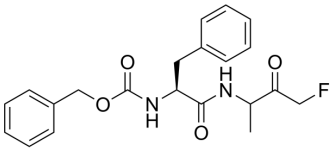
Molecular Weight: 386.42

Target: SARS-CoV; Cathepsin; Apoptosis; Caspase

Pathway: Anti-infection; Metabolic Enzyme/Protease; Apoptosis

Storage: -20°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 250 mg/mL (646.96 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5879 mL	12.9393 mL	25.8786 mL
	5 mM	0.5176 mL	2.5879 mL	5.1757 mL
	10 mM	0.2588 mL	1.2939 mL	2.5879 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (5.38 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (5.38 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.38 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Z-FA-FMK ((1S)-Z-FA-FMK) is a potent Cathepsin B and L inhibitor. Z-FA-FMK blocks the induction of DEVDase activity, DNA fragmentation, and externalization of phosphatidylserine by selective synthetic retinoid-related molecules (RRMs). Z-FA-FMK inhibits apoptosis. Z-FA-FMK inhibits caspase activity and selectively inhibits recombinant effector caspases 2, -3, -6, and -7. Z-FA-FMK is a viral inhibitor. Z-FA-FMK inhibits reovirus replication in a susceptible host[1][2][3].

IC_{50} & Target				
	Cathepsin B	cathepsin L	Caspase-2	Caspase-3
	Caspase-6	Caspase-7		

In Vitro

Z-FA-FMK ((1S)-Z-FA-FMK; 5-100 μM; 1 h; Jurkat cells) reduces levels of DEVDase activity and DNA fragmentation. Z-FA-FMK inhibits the externalization of phosphatidylserine induced by either MX2870-1 or MX781[1].
Z-FA-FMK (100 μM; 1 h; Jurkat cells) inhibits apoptosis. Z-FA-FMK inhibited the induction of DEVDase activity not only by the RRMs but also by other apoptotic insults, including etoposide-, ceramide-, and CD95/Fas receptor-mediated pathways[1].
Z-FA-FMK (0-100 μM; 1 h; Jurkat cells) inhibits caspases 2, -3, -6, and -7 activity through repressed induction of DEVDase activity in Jurkat cells[1].
Z-FA-FMK (0-20 μM; 48 h; HT1080 and mouse embryonic stem cells) blocks reoviral replication and cures cells of a persistent infection with reovirus in vitro[2].
Z-FA-FMK (20 μM; 48 h; HT1080 cells) induces defects in reoviral maturation[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Z-FA-FMK (1 mg/kg; intratumor injection; every 2 d, for 27 d; SCID mice with HT1080 xenograft) blocks reovirus infection in vivo[2].
Z-FA-FMK (8 mg/kg; i.v.; every 2 d, once; male BALB/c mice) markedly lessens the degree of impairment seen in D-GalN/TNF-α-induced kidney injury[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	SCID mice with HT1080 xenograft (6-8 weeks)[2]
Dosage:	1 mg/kg
Administration:	Intratumor injection; every 2 days, for 27 days
Result:	Blocked reovirus replication activity in both tumor and heart tissues.

Animal Model:	Male BALB/c mice[3]
Dosage:	8 mg/kg
Administration:	Intravenous injection; once, 1 hour later, intraperitoneal injection D-GalN (700 mg/kg) and TNF-α (15 μg/kg).
Result:	Decreased in the D-GalN/TNF-α-induced degenerative changes. Decreased in the number of activated caspase-3-positive tubular epithelial cell. Increased in kidney GSH levels, CAT, SOD and GPx activities and decreased in kidney LPO levels, LDH activity, serum AST and ALT activities, uric acid, and urea levels were determined.

CUSTOMER VALIDATION

- Nucleic Acids Res. 2021 Jan 8;49(D1):D1113-D1121.
- Phytomedicine. 2022 Jul;101:154102.

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REFERENCES

[1]. Lopez-Hernandez FJ, et, al. Z-FA-fmk inhibits effector caspases but not initiator caspases 8 and 10, and demonstrates that novel anticancer retinoid-related molecules induce apoptosis via the intrinsic pathway. Mol Cancer Ther. 2003 Mar;2(3):255-63.

[2]. Kim M, et, al. Z-FA-FMK as a novel potent inhibitor of reovirus pathogenesis and oncolysis in vivo. Antivir Ther. 2010;15(6):897-905.

[3]. Gezginci-Oktayoglu S, et, al. Effects of Z-FA.FMK on D-galactosamine/tumor necrosis factor-alpha-induced kidney injury and oxidative stress in mice : effects of Z-FA.FMK on TNF-alpha-mediated kidney injury. Mol Cell Biochem. 2008 Feb;309(1-2):9-20.

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