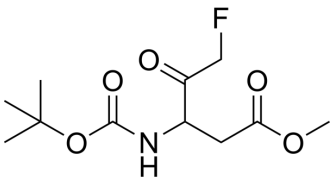
BOC-D-FMK

Cat. No.:	HY-13229
CAS No.:	634911-80-1
Molecular Formula:	$C_{11}H_{18}FNO_5$
Molecular Weight:	263.26
Target:	Caspase; Apoptosis
Pathway:	Apoptosis
Storage:	Pure form -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (379.85 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.7985 mL	18.9926 mL	37.9853 mL
5 mM	0.7597 mL	3.7985 mL	7.5971 mL
10 mM	0.3799 mL	1.8993 mL	3.7985 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: ≥ 2.5 mg/mL (9.50 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (9.50 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (9.50 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Boc-D-FMK is a cell-permeable, irreversible and broad spectrum caspase inhibitor. Boc-D-FMK inhibits apoptosis stimulated by TNF-α with an IC_{50} of 39 μM.

IC_{50} & Target

Caspase

In Vitro

Apoptosis is a pathway of cell death orchestrated by a family of proteases called caspases. Boc-D-fmk inhibits TNFα-stimulated reactive oxygen species (ROS) generation. Boc-D-FMK inhibits apoptosis stimulated by TNF-α with an IC_{50} of 39

	μM[1]. BocD-fmk at 50 μM prevents genistein-induced apoptosis of p815 cells. Confocal microscopy shows that the release of mitochondrial apoptotic factors is inhibited by BocD-fmk[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Boc-D-FMK-fmk effectively attenuates the hepatocyte apoptosis in bile duct-ligated rats and may improve the survival rates after endotoxin challenge[3]. A single injection of Boc-D-FMK results in longterm protection of MNs against root avulsion-induced death for more than 8 weeks and the Boc-D-FMK-treated MNs are able to regenerate their axons into an implanted PN graft and reinnervate the target muscle[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [3]	Rats: Boc-D-FMK is dissolved in DMSO. Male Sprague-Dawley rats group 1 (OBBOC-D) undergo common bile duct ligation and simultaneously treatment with Boc-D-FMK. The first dose of Boc-D-FMK (1.5 mg/kg) is injected into the inferior vena cava immediately after bile duct ligation. Subsequent doses of Boc-DFMK (1.5 mg/kg twice daily) are given intraperitoneally on the first and second postoperative days. The last dose (1.5 mg/kg) is given on the morning of the third postoperative day [3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Exp Clin Cancer Res. 2023 Jun 6;42(1):142.
- Anal Chem. 2017 Sep 19;89(18):9788-9796.
- Sci Rep. 2017 Jun 7;7(1):2929.
- Saudi J Biol Sci. 6 December 2021.
- Exp Cell Res. 2018 Sep 1;370(1):103-115.

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REFERENCES

[1]. Cowburn AS, et al. z-VAD-fmk augmentation of TNF alpha-stimulated neutrophil apoptosis is compound specific and does not involve the generation of reactive oxygen species.

[2]. Yee SB, et al. zVAD-fmk, unlike BocD-fmk, does not inhibit caspase-6 acting on 14-3-3/Bad pathway in apoptosis of p815 mastocytoma cells. Exp Mol Med. 2006 Dec 31;38(6):634-42.

[3]. Sheen-Chen SM, et al. Effect of Boc-D-Fmk on hepatocyte apoptosis after bile duct ligation in rat and survival rate after endotoxin challenge. J Gastroenterol Hepatol. 2008 Aug;23(8 Pt 1):1276-9.

[4]. Chan YM, et al. Inhibition of caspases promotes long-term survival and reinnervation by axotomized spinal motoneurons of denervated muscle in newborn rats. Exp Neurol. 2003 Jun;181(2):190-203.

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