Ac-DEVD-CMK TFA

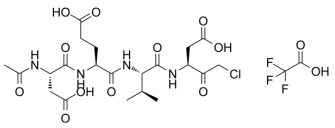
Cat. No.: HY-P0034A

Molecular Formula: $C_{23}H_{32}ClF_3N_4O_{13}$

Molecular Weight: 664.97

Target: Caspase; Apoptosis

Pathway: Apoptosis



Storage: Sealed storage, away from moisture and light, under nitrogen

Powder -80°C 2 years

-20°C 1 year

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

SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (150.38 mM; Need ultrasonic)

H_2O : 50 mg/mL (75.19 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.5038 mL	7.5191 mL	15.0383 mL
	5 mM	0.3008 mL	1.5038 mL	3.0077 mL
	10 mM	0.1504 mL	0.7519 mL	1.5038 mL

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

BIOLOGICAL ACTIVITY

Description

Ac-DEVD-CMK (Caspase-3 Inhibitor III) TFA is a selective and irreversible caspase-3 inhibitor. Ac-DEVD-CMK TFA significantly inhibits apoptosis induced by high levels of glucose or 3,20-dibenzoate (IDB; HY-137295). Ac-DEVD-CMK TFA can be used in a variety of experimental approaches to inhibit apoptosis[1][2][3].

In Vitro

Ac-DEVD-CMK TFA (100 μM; 24 h) inhibits IDB-induced apoptosis[3].

Ac-DEVD-CMK TFA inhibits (10 μM; 36 h) inhibits citrate (10 mM)-induced p21 cleavage and G2/M accumulation in human pharyngeal squamous carcinoma FaDu and Detroit 562 cell lines[4].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[4]

Cell Line:	FaDu and Detroit 562 cell
Concentration:	10 μM; with 10 mM citrate

Incubation Time:	36 h
Result:	Inhibited citrate to induce p21 cleavage.

In Vivo

Ac-DEVD-CMK TFA (Caspase-3 Inhibitor III; 25 mg/kg; IP; single dose; 3 hours post-APAP) significantly attenuates Acetaminophen (APAP; HY-66005)-induced liver injury (AILI)[5].

Ac-DEVD-CMK TFA (25 mg/kg; ip; single dose) significantly attenuates APAP-induced liver injury (AILI) in susceptible Sdc1−/− mice[6].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	$Sdc1^{-/-}$ mice induced by APAP[6]
Dosage:	25 mg/kg
Administration:	IP; single dose, 3 hours post-APAP
Result:	Inhibited GSK3β or caspase3 activity to mitigate liver damage.

REFERENCES

[1]. Lu Cai, et al. Hyperglycemia-induced apoptosis in mouse myocardium: mitochondrial cytochrome C-mediated caspase-3 activation pathway. Diabetes. 2002 Jun;51(6):1938-48.

[2]. M M Mocanu, et al. Caspase inhibition and limitation of myocardial infarct size: protection against lethal reperfusion injury. Br J Pharmacol. 2000 May;130(2):197-200.

[3]. M Blanco-Molina, et al. Ingenol esters induce apoptosis in Jurkat cells through an AP-1 and NF-kappaB independent pathway. Chem Biol. 2001 Aug;8(8):767-78.

[4]. Eon Jeong Nam, et al. Syndecan-1 limits the progression of liver injury and promotes liver repair in acetaminophen-induced liver injury in mice. Hepatology. 2017 Nov;66(5):1601-1615.

[5]. Hung KC, et al. Citrate-Induced p85αPTEN Complex Formation Causes G2/M Phase Arrest in Human Pharyngeal Squamous Carcinoma Cell Lines. Int J Mol Sci. 2019 Apr 29;20(9):2105.

[6]. Nam EJ, et al. Syndecan-1 limits the progression of liver injury and promotes liver repair in acetaminophen-induced liver injury in mice. Hepatology. 2017 Nov;66(5):1601-1615.

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