Ac-DEVD-CHO

Cat. No.: HY-P1001

CAS No.: 169332-60-9

Molecular Formula: $C_{20}H_{30}N_4O_{11}$

Molecular Weight: 502.47

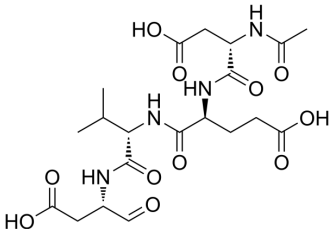
Sequence: N-Acetyl-Asp-Glu-Val-Asp-al

Sequence Shortening: Ac-DEVD-al

Target: Caspase

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

H_2O : ≥ 50 mg/mL (99.51 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.9902 mL	9.9508 mL	19.9017 mL
5 mM	0.3980 mL	1.9902 mL	3.9803 mL
10 mM	0.1990 mL	0.9951 mL	1.9902 mL

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

In Vivo

1. Add each solvent one by one: PBS
Solubility: 100 mg/mL (199.02 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description Ac-DEVD-CHO is a specific Caspase-3 inhibitor with a K_i value of 230 pM.

IC_{50} & Target

Caspase 3 0.23 nM (Ki)	Caspase-8 0.92 nM (Ki)	Caspase-7 1.6 nM (Ki)	Caspase-10 12 nM (Ki)
Caspase-1 18 nM (Ki)	Caspase-6 31 nM (Ki)	Caspase-9 60 nM (Ki)	Caspase-4 132 nM (Ki)

	Caspase-5 205 nM (Ki)	Caspase-2 1710 nM (Ki)

In Vitro	To ascertain the role of caspase-3 in SLNT-induced apoptosis, a? caspase-3 inhibitor (Ac-DEVD-CHO) is used. The addition of Ac-DEVD-CHO significantly prevents SLNT-induced apoptosis (from 32.91±1.21% decreases to 15.88±1.58% while NC and Ac-DEVD-CHO groups are 6.45±0.96%, 7.77±0.79%, respectively)[2]. The apoptosis rates of cells pretreated with zVAD-fmk (5.32%) or Ac-DEVD-CHO (7.43%) decrease obviously after hypericin-mediated PDT treatment[3]. Remarkably, 10 μmol/L Ac-DEVD-CHO partially blocks the effect of SIN-induced apoptosis and reduces the number of apoptotic nuclei. These effects of SIN are blocked by the caspase-3 inhibitor Ac-DEVD-CHO. Camptothecin (4 μM), a positive control, increases caspase-3 activity, which is also blocked by Ac-DEVD-CHO[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Compare with model group, in CI group, the concentrations of serum BUN are decreased significantly at all time points after operation and those of Cr are decreased significantly at 6 hours, then restored to those of the sham group at 12 hours and 24 hours; the concentrations of serum TNF-α, IL-6 are decreased and those of IL-10 are elevated significantly at all time points. [TNF-α (μg/L) 6 hours: 436.2±64.2 vs. 653.6±8.9, 12 hours: 233.4±85.4 vs. 579.7±137.1, 24 hours: 151.0±90.3 vs. 551.0±119.8, IL-6 (μg/L) 6 hours: 1033.2±345.8 vs. 1 595.3±159.4, 12 hours: 366.3±68.3 vs. 1 330.7±249.8, 24 hours: 241.2±208.4 vs. 815.3±572.7, IL-10 (μg/L) 6 hours: 33.6±10.4 vs. 26.6±4.5, 12 hours: 37.2±5.0 vs. 24.5±4.3, 24 hours: 38.3±5.5 vs. 18.2±1.6, all P<0.05]; the renal cell apoptosis rates are decreased significantly at all time points: apoptosis rates 6 hours: (13.9±3.2)% vs. (18.3±1.4)%, 12 hours: (10.5±3.6)% vs. (15.9±3.5)%, 24 hours: (8.4±1.8)% vs.(12.5±2.1)%[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [4]	OCLs are incubated with RANKL and treated with 0.5 mM SIN with or without the specific caspase-3 inhibitor Ac-DEVD-CHO (10 μM) for 24 h. At the end of the treatment, the cells are washed with PBS and are stained for 15 min with 10 μM Hoechst 33258 dye. Images of the staineing cells are captured with a fluorescent microscope. The differences are evaluated by counting the number of cells with apoptotic nuclear condensation in each well[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [5]	One hundred and two male mice are subjected to cecal ligation and puncture or sham operation. The animals are assigned into three equal groups (n=34) according to random number table: sham group, model group, and caspase-3 inhibitor (CI) group. Thirty minutes before CLP, Ac-DEVD-CHO (4 μg/g) is injected subcutaneously in CI group. The levels of blood urea nitrogen (BUN) and creatinine (Cr) are determined, and the concentrations of tumor necrosis factor-α (TNF-α), interleukins (IL-6 and IL-10) are measured by enzyme linked immunosorbent assay (ELISA), the renal cell apoptosis rate is determined by flow cytometry. The 4-day and 7-day survival rates of three groups of mice are observed[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Adv Mater. 2023 Jun;35(23):e2300548.
- Biomaterials. 2022 Sep 29;290:121832.
- J Immunother Cancer. 2024 Mar 1;12(3):e008054.
- J Control Release. 2021 Apr 16;S0168-3659(21)00179-6.
- J Colloid Interface Sci. 29 September 2021.

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[1]. Garcia-Calvo M, et al.nhibition of human caspases by peptide-based and macromolecular inhibitors. J Biol Chem. 1998 Dec 4;273(49):32608-13.

[2]. Jinglin Wang, et al. A polysaccharide from Lentinus edodes inhibits human colon cancer cell proliferation and suppresses tumor growth in athymic nude mice. Oncotarget. 2017 Jan 3; 8(1): 610-623.

[3]. Junping Zhang, et al. Hypericin-mediated photodynamic therapy induces apoptosis of myoloma SP2/0 cells depended on caspase activity in vitro. Cancer Cell Int. 2015; 15: 58

[4]. Long-gang He, et al. Sinomenine induces apoptosis in RAW 264.7 cell-derived osteoclasts in vitro via caspase-3 activation. Acta Pharmacol Sin. 2014 Feb; 35(2): 203-210.

[5]. Liu LX, et al. The effect of caspase-3 inhibitor on the concentrations of serum inflammatory cytokines in sepsis related acute kidney injury induced by peritoneal cavity infection in mice. Zhongguo Wei Zhong Bing Ji Jiu Yi Xue. 2010 Dec;22(12):736-9.

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