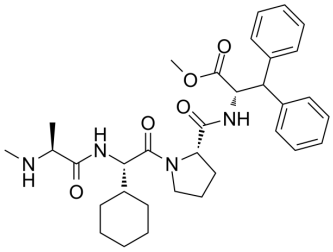
MV1

Cat. No.:	HY-113534
CAS No.:	1001600-54-9
Molecular Formula:	$C_{33}H_{44}N_4O_5$
Molecular Weight:	576.73
Target:	IAP; Apoptosis
Pathway:	Apoptosis
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 125 mg/mL (216.74 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.7339 mL	8.6696 mL	17.3391 mL
5 mM	0.3468 mL	1.7339 mL	3.4678 mL
10 mM	0.1734 mL	0.8670 mL	1.7339 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 (3.61 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (3.61 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (3.61 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

MV1 is an antagonist of IAP (inhibitor of apoptosis protein), leads to protein knockdown of HaloTag-fused proteins when combined with HaloTag ligand[1].

In Vitro

MV1 (0.08-20 μM; 24 h) inhibits the growth of EVSAT cells[1].

MV1 (5 μM; 0-60 min) treatment causes rapid loss of c-IAP1 and c-IAP2 in MDA-MB-231 cells[1].

MV1 (5 μM; 1 h) treatment inducing degradation of c-IAP1 and c-IAP2 is dependent on proteasomal machinery but not on

caspase activation[1].

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

Cell Viability Assay[1]

Cell Line:	EVSAT cells
Concentration:	0.08-20 μM
Incubation Time:	24 hours
Result:	Showed IC_{50} value of 5 μM for EVSAT cells.

Western Blot Analysis[1]

Cell Line:	MDA-MB-231 cells
Concentration:	5 μM
Incubation Time:	0-60 min
Result:	Showed the decreasement of c-IAP1 and 2 protein levels by as early as two minutes following exposure.

REFERENCES

[1]. Eugene Varfolomeev, et al. IAP antagonists induce autoubiquitination of c-IAPs, NF-kappaB activation, and TNFalpha-dependent apoptosis. Cell. 2007 Nov 16;131(4):669-81.

[2]. Tomoshige S, et al. Efficient protein knockdown of HaloTag-fused proteins using hybrid molecules consisting of IAP antagonist and HaloTag ligand. Bioorg Med Chem. 2016 Jul 15;24(14):3144-8.

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