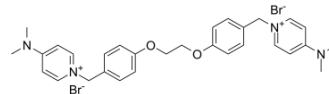


EB-3D

Cat. No.:	HY-115463		
CAS No.:	1839150-63-8		
Molecular Formula:	C ₃₀ H ₃₆ Br ₂ N ₄ O ₂		
Molecular Weight:	644.44		
Target:	AMPK; Apoptosis		
Pathway:	Epigenetics; PI3K/Akt/mTOR; Apoptosis		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (77.59 mM; Need ultrasonic)

Concentration	Mass		
	1 mg	5 mg	10 mg
1 mM	1.5517 mL	7.7587 mL	15.5173 mL
5 mM	0.3103 mL	1.5517 mL	3.1035 mL
10 mM	0.1552 mL	0.7759 mL	1.5517 mL

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

BIOLOGICAL ACTIVITY

Description

EB-3D is a potent and selective choline kinase α (ChoK α) inhibitor, with an IC₅₀ of 1 μ M for ChoK α 1. EB-3D exerts effects on ChoK α expression, AMPK activation, apoptosis, endoplasmic reticulum stress and lipid metabolism. EB-3D exhibits a potent antiproliferative activity in a panel of T-leukemia cell lines. Anti-cancer activity^{[1][2][3]}.

IC₅₀ & Target

IC₅₀: 1 μ M; K_d: 0.7 μ M^[3]

In Vitro

EB-3D displays (0-100 μ M; 72 hours) excellent antiproliferative activity against a wide cohort of T-leukemic cell lines, with GI₅₀s 13 values in the nanomolar range^[1].

EB-3D (1.25-5 μ M; 24 hours) induced apoptosis in leukemia cell lines^[1].

EB-3D (0.5-1 μ M; 24 hours) induces a G₀/G₁ arrest that lead to apoptosis^[1].

EB-3D (0.3 μ M; 48 hours) shows a first spike of activation of AMPK α after 30 minutes and a later increase in the phosphorylation of T172^[1].

EB-3D (1-40 μ M; 48 hours) inhibits cell growth in HepG2 cells with a GI₅₀ of 14.55 μ M^[2].

EB-3D induces deregulation of the AMPK-mTOR pathway and apoptosis in leukemia T-cells^[1].

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

Cell Proliferation Assay^[1]

Cell Line:	JURKAT, CCRF-CEM, HSB-2, MOLT-16, DNA-41, LOUCY, PEER, ALL-SIL cells
Concentration:	0.001, 0.01, 0.1, 1, 10, 100 μ M
Incubation Time:	72 hours
Result:	Inhibited JURKAT, CCRF-CEM, HSB-2, MOLT-16, DNA-41, LOUCY, PEER, and ALL-SIL cells growth with GI ₅₀ s of 136.2, 478.8, 17.7, 0.9, 60.6, 200, 265, and 132 nM, respectively.

Apoptosis Analysis^[1]

Cell Line:	Jurkat, CCRF-CEM and HSB-2 cells
Concentration:	1.25, 2.5, 5 μ M
Incubation Time:	24 hours
Result:	Induced apoptosis in leukemia cell lines.

Cell Cycle Analysis^[1]

Cell Line:	Jurkat, CCRF-CEM and HSB-2 cells
Concentration:	0.5, 1 μ M
Incubation Time:	24 hours
Result:	Induces cell cycle arrest in G ₀ /G ₁ phase.

Western Blot Analysis^[1]

Cell Line:	Jurkat cells
Concentration:	0.3 μ M
Incubation Time:	48 hours
Result:	Showed a first spike of activation of AMPK α after 30 minutes of treatment and a later increase in the phosphorylation of T172. The increase in S79 phosphorylation of its main target ACC (acetyl-coenzyme A (CoA) carboxylase), followed the same pattern. This rapid activation of AMPK, in turn induced a consequent reduction in mTOR phosphorylation that is visible already at 30' and that becomes amplified at longer time probably due to the interruption of feedback loops that are characteristic of mTOR connecting pathways.

In Vivo

EB-3D (1 mg/kg; i.p.; every other day) impairs mammary tumor growth in syngeneic orthotopic E0771-C57BL/6 mouse model [4].

EB-3D (2.5 mg/kg; every other day for 4 weeks) shows a reduction of the number of spontaneous lung macro- and micrometastasis^[4].

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

Animal Model:	E0771-C57BL/6 mice ^[4]
Dosage:	i.p.; every other day for 4 weeks
Administration:	2.5 mg/kg

Result:	A reduction of the number of spontaneous lung macro- and micrometastasis.
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REFERENCES

- [1]. Mariotto E, et al. EB-3D a novel choline kinase inhibitor induces deregulation of the AMPK-mTOR pathway and apoptosis in leukemia T-cells. *Biochem Pharmacol.* 2018 Sep;155:213-223.
- [2]. Sola-Leyva A, et al. Choline kinase inhibitors EB-3D and EB-3P interferes with lipid homeostasis in HepG2 cells. *Sci Rep.* 2019 Mar 25;9(1):5109.
- [3]. Schiaffino-Ortega S, et al. Design, synthesis, crystallization and biological evaluation of new symmetrical biscationic compounds as selective inhibitors of human Choline Kinase $\alpha 1$ (ChoK $\alpha 1$).
- [4]. Mariotto E, et al. Choline Kinase Alpha Inhibition by EB-3D Triggers Cellular Senescence, Reduces Tumor Growth and Metastatic Dissemination in Breast Cancer. *Cancers (Basel).* 2018;10(10):391. Published 2018 Oct 22.
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Caution: Product has not been fully validated for medical applications. For research use only.

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