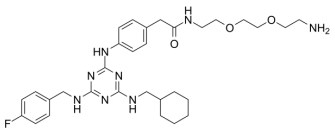
AP-III-a4

Cat. No.:	HY-15858
CAS No.:	1177827-73-4
Molecular Formula:	$C_{31}H_{43}FN_8O_3$
Molecular Weight:	594.72
Target:	Enolase; Apoptosis
Pathway:	Metabolic Enzyme/Protease; Apoptosis
Storage:	4°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (168.15 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	1.6815 mL	8.4073 mL	16.8146 mL
5 mM	0.3363 mL	1.6815 mL	3.3629 mL
10 mM	0.1681 mL	0.8407 mL	1.6815 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 (4.20 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (4.20 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.20 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

AP-III-a4 (ENOblock) is a nonsubstrate analogue enolase inhibitor with an IC_{50} of 0.576 uM. AP-III-a4 can be used for the research of cancer and diabetic[1].

IC_{50} & Target

IC_{50}: 0.576 uM (enolase)[1]

In Vitro

AP-III-a4 (ENOblock) (0-10 μM; 24 h) inhibits HCT116 cell viability in a dose-dependent manner[1].
AP-III-a4 directly binds to enolase and inhibits its activity[1].
AP-III-a4 (0-10 μM; 24 or 48 h) inhibits cancer cell migration and invasion, induces cancer cell apoptosis[1].

AP-III-a4 (10 μM; 24 h) can induce glucose uptake and inhibit phosphoenolpyruvate carboxykinase (PEPCK) expression in hepatocytes and kidney cells[1].

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

Cell Viability Assay[1]

Cell Line:	HCT116
Concentration:	1.25, 2.5, 5 and 10 μM
Incubation Time:	24 h
Result:	Induced higher levels of HCT116 colon cancer cell death in hypoxic conditions compared to normoxia.

Western Blot Analysis[1]

Cell Line:	HCT116
Concentration:	1.25, 2.5, 5 and 10 μM
Incubation Time:	24 h for AKT, 48 h for Bcl-Xl
Result:	Bound to enolase in cell lysate and bound to purified enolase. Decreased the expression of AKT and Bcl-Xl, which are negative regulators of apoptosis.

Cell Invasion Assay[1]

Cell Line:	HCT116
Concentration:	0.156, 0.312, 0.625, 1.25 and 2.5 μM
Incubation Time:	24 h
Result:	Significantly inhibits cancer cell invasion at a treatment concentration of 0.625 μM.

Cell Migration Assay [1]

Cell Line:	HCT116
Concentration:	0.625, 1.25 and 2.5 μM
Incubation Time:	24 h
Result:	Inhibited cell migration dose-dependently.

RT-PCR[1]

Cell Line:	Huh7 and HEK
Concentration:	10 μM
Incubation Time:	24 h
Result:	Induced glucose uptake and inhibited PEPCK expression.

In Vivo

AP-III-a4 (ENOblock) (10 μM; 96 h) inhibits cancer cell metastasis and suppresses the gluconeogenesis regulator PEPCK in zebrafish[1].

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

Animal Model:	The zebrafish cancer cell HCT116 xenograft model[1]
Dosage:	10 μM
Administration:	96 h
Result:	Reduced cancer cell dissemination. Inhibited PEPCK expression and induced glucose uptake. Inhibited adipogenesis and foam cell formation.

CUSTOMER VALIDATION

- Cell Discov. 2020 Aug 18;6:56.
- Gastroenterology. 2024 Jan 24:S0016-5085(24)00064-7.
- Theranostics. 2019 Aug 12;9(20):5769-5783.
- Cancers (Basel). 2020 Jan 29;12(2):311.
- SSRN. 2023 Feb 10.

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REFERENCES

[1]. Da-Woon Jung, et al. A Unique Small Molecule Inhibitor of Enolase Clarifies Its Role in Fundamental Biological Processes.ACS Chem. Biol., 2013, 8 (6), pp 1271–1282

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