AP-III-a4 hydrochloride

Cat. No.: HY-15858A

CAS No.: 2070014-95-6

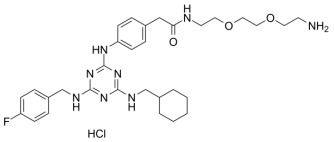
Molecular Formula: $C_{31}H_{44}ClFN_8O_3$

Molecular Weight: 631

Target: Enolase; Apoptosis

Pathway: Metabolic Enzyme/Protease; Apoptosis

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 2 years; -20°C, 1 year (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 53 mg/mL (83.99 mM)

H_2O : 12.5 mg/mL (19.81 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.5848 mL	7.9239 mL	15.8479 mL
	5 mM	0.3170 mL	1.5848 mL	3.1696 mL
	10 mM	0.1585 mL	0.7924 mL	1.5848 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 (3.30 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (3.30 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (3.30 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

AP-III-a4 (ENOblock) hydrochloride is a nonsubstrate analogue enolase inhibitor with an IC_{50} of 0.576 uM. AP-III-a4 hydrochloride 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.
- SSRN. 2023 Feb 10.
- Research Square Print. November 14th, 2022

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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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