Elacridar hydrochloride

Cat. No.: HY-50880

CAS No.: 143851-98-3

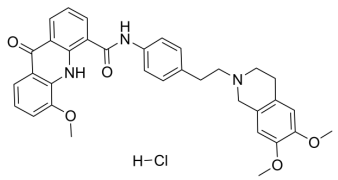
Molecular Formula: $C_{34}H_{34}ClN_3O_5$

Molecular Weight: 600.1

Target: P-glycoprotein; BCRP

Pathway: Membrane Transporter/Ion Channel

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 25 mg/mL (41.66 mM; ultrasonic and warming and heat to 60°C)
H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

Preparing Stock Solutions

Mass / Solvent / Concentration	1 mg	5 mg	10 mg
1 mM	1.6664 mL	8.3319 mL	16.6639 mL
5 mM	0.3333 mL	1.6664 mL	3.3328 mL
10 mM	0.1666 mL	0.8332 mL	1.6664 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 (3.47 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Elacridar hydrochloride (GF120918A) is an orally active P-glycoprotein (P-gp) and breast cancer resistance protein (BCRP) inhibitor. Elacridar hydrochloride can be used to examine the influence of efflux transporters on agent distribution to brain and it can be used for the research of cancer[1][2].

In Vitro

Elacridar hydrochloride (0.001-1 μM; 2 h) inhibits cell viability of 786-O cells[2].
Elacridar hydrochloride (5 μM; 24 h) affects P-glycoprotein and ABCG2 protein expression levels in MCF-7 and 786-O cell lines [2].
Elacridar hydrochloride (5 μM; 24 h) affects ^{99m}Tc-MIBI intracellular accumulation in MCF-7 and 786-O cells[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[2]

Cell Line:	786-O cells

Concentration:	2.5 and 5 μM
Incubation Time:	2 hours
Result:	Dose-dependently inhibited cell viability of 786-O cells and showed better inhibitory effect with sunitnib adding.

Western Blot Analysis[2]

Cell Line:	MCF-7, Caki-1, and 786-O cell lines
Concentration:	5 μM
Incubation Time:	24 hours
Result:	Dreased P-glycoprotein protein expression level in 786-O cells and increased ABCG2 protein expression level in Caki-1 cells.

Cell Viability Assay[2]

Cell Line:	MCF-7 and 786-O cell lines
Concentration:	5 μM
Incubation Time:	24 hours
Result:	Dose-dependently increased ^{99m}Tc-MIBI intracellular accumulation in MCF-7 and 786-O cells.

In Vivo

Elacridar hydrochloride (100 mg/kg; i.p. once) shows different distribution in brain and plasma[1].
Plasma Pharmacokinetic Parameters of Elacridar hydrochloride in mice[1].

	Mice PO 100 mg/kg	Mice IP 100 mg/kg	Mice IV 2.5 mg/kg
CL/F (ml/min)	2.05	33.2	0.46
Vd/F (liter)	3.5	12.3	0.17
$t_{1/2}$ (h)	20	4.3	4.4
$AUC_{0\text{-}inf}$ (μg·min/ml)	1460	90.3	161.4
F	0.22	0.013	1

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

Animal Model:	FVB wild-type mice[1]
Dosage:	100 mg/kg
Administration:	Intraperitoneal injection; 100 mg/kg once
Result:	Showd a higher concertration in brain than plasma except at 4 h after administration.

CUSTOMER VALIDATION

- Cell Metab. 2024 Jan 2:S1550-4131(23)00465-5.
- Sci Adv. 2023 Oct 20;9(42):eabp9530.
- Clin Cancer Res. 2018 Jan 15;24(2):383-394.
- Mol Psychiatry. 2023 Oct 16.
- Cell Death Dis. 2021 Jul 27;12(8):742.

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REFERENCES

[1]. Sane R, et al. Brain distribution and bioavailability of elacridar after different routes of administration in the mouse. Drug Metab Dispos. 2012 Aug;40(8):1612-9.

[2]. Sato H, et al. Elacridar enhances the cytotoxic effects of sunitinib and prevents multidrug resistance in renal carcinoma cells. Eur J Pharmacol. 2015 Jan 5;746:258-66.

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