AMXT-1501 tetrahydrochloride

Cat. No.: HY-124617A

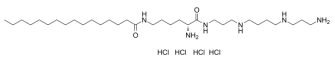
Molecular Formula: $C_{32}H_{72}Cl_4N_6O_2$

Molecular Weight: 715

Target: Apoptosis

Pathway: Apoptosis

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



SOLVENT & SOLUBILITY

In Vitro

H_2O : 83.33 mg/mL (116.55 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.3986 mL	6.9930 mL	13.9860 mL
	5 mM	0.2797 mL	1.3986 mL	2.7972 mL
	10 mM	0.1399 mL	0.6993 mL	1.3986 mL

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

In Vivo

1. Add each solvent one by one: PBS
Solubility: 50 mg/mL (69.93 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

AMXT-1501 tetrahydrochloride is an orally active polyamine transport inhibitor. AMXT1501 blocks tumor growth in immunocompetent mice but not in athymic nude mice lacking T cells[1]. Combination of DFMO and AMXT□1501 induces caspase□3 mediated apoptosis in NB cell lines[2].

IC_{50} & Target

Polyamine transport[1]

In Vitro

AMXT-1501 tetrahydrochloride (0.39-50 μM; 48 hours) treatment exhibits cytotoxicity against this panel of NB cell lines (BE(2)-C, SMS-KCNR and SH-SY5Y cells), with IC_{50} values of 17.72 μM for SMS-KCNR, 17.69 μM for BE(2)-C, and 14.13 μM for SH-SY5Y[2].

BE(2)□C, SMS□KCNR and SH□SY5Y cells are exposed to AMXT-1501 tetrahydrochloride (2.5 μM) and DFMO (2.5 mM) alone or in combination (AMXT-1501 tetrahydrochloride 2.5 μM + DFMO 2.5 mM). After 96 hours exposure to AMXT-1501 tetrahydrochloride or DFMO does not significantly alter the level of noncleaved PARP, cleaved PARP and cleaved caspase 3, whereas cells treated with the combination of AMXT-1501 tetrahydrochloride with DFMO decrease the amount of noncleaved PARP and increase the amount of cleaved PARP and cleaved caspase 3[2].

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

Cell Viability Assay[2]

Cell Line:	BE(2)□C, SMS□KCNR and SH□SY5Y cells
Concentration:	0.39 μM, 1 μM, 3.1 μM, 10 μM, 31 μM, 50 μM
Incubation Time:	48 hours
Result:	AMXT-1501 tetrahydrochloride exhibited cytotoxicity against this panel of NB cell lines.

Western Blot Analysis[2]

Cell Line:	BE(2)□C, SMS□KCNR and SH□SY5Y cells
Concentration:	2.5 μM
Incubation Time:	72 hours
Result:	Combination treatment with DFMO decreased the amount of noncleaved PARP and increased the amount of cleaved PARP and cleaved caspase 3 in all three cell lines.

In Vivo

AMXT-1501 tetrahydrochloride (3 mg/kg; subcutaneous injection; every day; 28 days) alone is sufficient to delay EAE onset moderately,but fails to protect animals from reaching the endpoint. However, the combination of DFMO and AMXT-1501 tetrahydrochloride are sufficient to deplete T cell polyamine pool, and consequently suppress T cell proliferation and effector function in vivo[3].

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Animal Model:	C57BL/6 (WT) and ODC knockout strain (ODC cKO) mice bearing experimental autoimmune encephalomyelitis (EAE) model [3]
Dosage:	3 mg/kg
Administration:	Subcutaneous injection; every day; 28 days
Result:	Displayed a delayed disease onset initially, but eventually proceeded with pathologic development and reached the endpoint.

REFERENCES

[1]. Candace S Hayes, et al. Polyamine-blocking therapy reverses immunosuppression in the tumor microenvironment. Cancer Immunol Res. 2014 Mar;2(3):274-85.

[2]. Ruohan Wu, et al. De novo synthesis and salvage pathway coordinately regulate polyamine homeostasis and determine T cell proliferation and function. Sci Adv. 2020 Dec 16;6(51):eabc4275.

[3]. Katherine Samal, et al. AMXT-1501, a novel polyamine transport inhibitor, synergizes with DFMO in inhibiting neuroblastoma cell proliferation by targeting both ornithine decarboxylase and polyamine transport. Int J Cancer. 2013 Sep 15;133(6):1323-33.

Caution: Product has not been fully validated for medical applications. For research use only.

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