TD52 dihydrochloride

Cat. No.: HY-135699A

Molecular Formula: $C_{24}H_{18}Cl_2N_4$

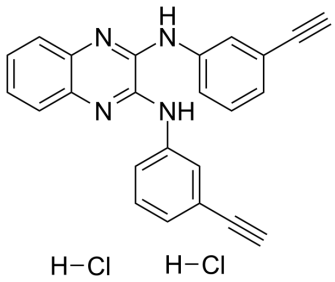
Molecular Weight: 433.33

Target: Akt; Phosphatase; Apoptosis

Pathway: PI3K/Akt/mTOR; Metabolic Enzyme/Protease; 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 : 25 mg/mL (57.69 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3077 mL	11.5386 mL	23.0771 mL
	5 mM	0.4615 mL	2.3077 mL	4.6154 mL
	10 mM	0.2308 mL	1.1539 mL	2.3077 mL

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

BIOLOGICAL ACTIVITY

Description

TD52 dihydrochloride, an Erlotinib (HY-50896) derivative, is an orally active, potent cancerous inhibitor of protein phosphatase 2A (CIP2A) inhibitor. TD52 dihydrochloride mediates the apoptotic effect in triple-negative breast cancer (TNBC) cells via regulating the CIP2A/PP2A/p-Akt signalling pathway. TD52 dihydrochloride indirectly reduced CIP2A by disturbing Elk1 binding to the CIP2A promoter. TD52 dihydrochloride has less p-EGFR inhibition and has potent anti-cancer activity[1].

In Vitro

TD52 dihydrochloride (2-10 μM; 48 hours) shows anti-proliferative ability and induces differential apoptotic effects in these cell lines[1].

TD52 dihydrochloride (5 μM; 48 hours) has minimal effects on p-EGFR or EGFR expression but downregulated CIP2A expression[1].

TD52 dihydrochloride (2.5, 5, 7.5 μM; 48 hours) time-dependently induces apoptosis accompanied with downregulating CIP2A and p-Akt[1].

TD52 dihydrochloride (5 μM; 24 hours) significantly increases the phosphatase activity of PP2A in TNBC cells[1].

TD52 dihydrochloride (5 μM; 48 hours) has no obvious effects on other common RTKs, such as IGFR, PDGFR and VEGFR2[1].

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

In Vivo	TD52 dihydrochloride (10 mg/kg/day; oral gavage; for 52 days) significantly inhibits MDA-MB-468 xenograft tumour size and tumour weight[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Chun-Yu Liu, et al. EGFR-independent Elk1/CIP2A signalling mediates apoptotic effect of an erlotinib derivative TD52 in triple-negative breast cancer cells. Eur J Cancer. 2017 Feb;72:112-123.

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