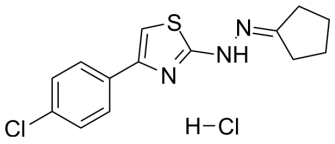
CPTH2 hydrochloride

Cat. No.:	HY-W013274A
CAS No.:	2108899-91-6
Molecular Formula:	$C_{14}H_{15}Cl_2N_3S$
Molecular Weight:	328.26
Target:	Histone Acetyltransferase; Apoptosis
Pathway:	Epigenetics; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	CPTH2 hydrochloride is a potent histone acetyltransferase (HAT) inhibitor. CPTH2 hydrochloride selectively inhibits the acetylation of histone H3 by Gcn5. CPTH2 hydrochloride induces apoptosis and decreases the invasiveness of a clear cell renal carcinoma (ccRCC) cell line through the inhibition of acetyltransferase p300 (KAT3B)[1][2].
IC_{50} & Target	GCN5

In Vitro

CPTH2 (100 μM; 12, 24, 48 hours) hydrochloride causes a decrease in cell proliferation after as early as 12 h with a further significant reduction after 48 h stimulation[1].
CPTH2 (100 μM; 12 or 48 hours) hydrochloride causes a comparable drop of the activity in both cell lines[1].
CPTH2 (100 μM; 48 hours) hydrochloride produces a drastic increase in apoptotic/dead cell population after 48 h[1].
CPTH2 (100 μM; 12, 24, 48 hours) hydrochloride shows a reduced acetylation of both global AcH3 histone and H3AcK18[1].
CPTH2 (100 μM; 24, 48 hours) hydrochloride is capable to counteract invasion and migration of ccRCC-786-O cells in culture [1].
CPTH2 (0.2, 0.5, 1 mM) hydrochloride inhibits the growth of a GCN5 deleted strain and a single catalytic mutant E173H[2].
CPTH2 (0.6, 0.8 mM; for 24 hours) hydrochloride inhibits histone H3 acetylation in yeast cell cultures[2].
CPTH2 hydrochloride inhibits the Gcn5p dependent functional network[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	Papillary thyroid (K1) and clear cell Renal Cell Carcinoma (ccRCC-786-O) cell lines
Concentration:	100 μM
Incubation Time:	12, 24, 48 hours
Result:	Caused a decrease in cell proliferation after as early as 12 h with a further significant reduction after 48 h stimulation.

Cell Viability Assay[1]

Cell Line:	K1 and ccRCC-786-O cell lines
Concentration:	100 μM

Incubation Time:	24 hours (K1 cell) and 48 hours (ccRCC-786-O cell)
Result:	Caused a comparable drop of the activity in both cell lines.

Apoptosis Analysis[1]

Cell Line:	ccRCC-786-O cells
Concentration:	100 μM
Incubation Time:	48 hours
Result:	Produced a drastic increase in apoptotic/dead cell population after 48 h.

Western Blot Analysis[1]

Cell Line:	ccRCC-786-O cells
Concentration:	100 μM
Incubation Time:	12, 24, 48 hours
Result:	Showed a reduced acetylation of both global AcH3 histone and H3AcK18.

REFERENCES

[1]. Cocco E, et al. KAT3B-p300 and H3AcK18/H3AcK14 levels are prognostic markers for kidney ccRCC tumoraggressiveness and target of KAT inhibitor CPTH2. Clin Epigenetics. 2018 Apr 4;10:44.

[2]. Chimenti F, et al. A novel histone acetyltransferase inhibitor modulating Gcn5 network: cyclopentylidene-[4-(4'-chlorophenyl)thiazol-2-yl)hydrazone. J Med Chem. 2009 Jan 22;52(2):530-6.

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

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