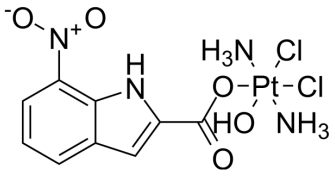
APE1-IN-2

Cat. No.:	HY-151883
CAS No.:	2923433-95-6
Molecular Formula:	$C_9H_{12}Cl_2N_4O_5Pt$
Molecular Weight:	522.21
Target:	Apoptosis; MDM-2/p53
Pathway:	Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

APE1-IN-2 (compound AP1) is a Pt(IV) proagent, targeting a critical BER protein, apurinic/apyrimidinic endonuclease 1 (APE1). APE1-IN-2 shows anticancer activity. APE1-IN-2 induces intracellular accumulation of platinum and activates DNA damage response and apoptosis signals[1].

In Vitro

APE1-IN-2 (compound AP1) can strongly inhibit the growth of malignant cells, including Cisplatin-resistant cancer cells, with up to 18.11 times inhibition compared with Cisplatin (HY-17394)[1].
APE1-IN-2 (500 nM, 24 h) arrests the cell cycle in A549 and MCF7 cells[1].
APE1-IN-2 (10 μM, 24 h) induces p53-dependent apoptosis in A549 cells[1].
APE1-IN-2 (0-250 μM, 72 h) inhibits AP-cutting activity with an IC_{50} of 45.14 ± 17.37 μM[1].
APE1-IN-2 can directly inhibit the AP endonuclease activity of APE1, leading to an interruption of miRNA processing and upregulation of the tumor suppressor PTEN[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	A549 (non-small cell lung cancer), MCF7 (breast cancer), U251 (glioblastoma), A375 (melanoma), PC3 (prostate cancer), and HEP-G2 (hepatocarcinoma) cell lines
Concentration:	
Incubation Time:	72 h
Result:	Demonstrated more potent antiproliferation effects than Cisplatin (HY-17394), with IC_{50} of 0.45 ± 0.03, 0.43 ± 0.03, 4.70 ± 0.14, 0.39 ± 0.03, 5.65 ± 0.21, and 3.53 ± 0.31 μM in A549, MCF7, U251, A375, PC3, and HEP-G2 cell lines, respectively.

Cell Cycle Analysis[1]

Cell Line:	A549 and MCF7 cells
Concentration:	500 nM
Incubation Time:	24 h
Result:	Induced the most severe S-phase arrest in A549 and MCF7 cells.

Cell Proliferation Assay[1]

Cell Line:	A549 cells
Concentration:	10 μM
Incubation Time:	24 h
Result:	Caused apoptosis in approximately 38.7% (22.9% early apoptosis and 15.8% late apoptosis) of cancer cells.

Western Blot Analysis[1]

Cell Line:	A549 and HEK-293T cell lines
Concentration:	0, 16, 40, 100, 250 μM
Incubation Time:	72 h
Result:	Significantly increased the level of p53 by 2.09 ± 0.51-fold. Slightly raised the levelsof p53, γH2A.X, and cl.PARP in HEK-293T. Inhibited AP-cutting activity with an IC_{50} value of 45.14 ± 17.37 μM.

In Vivo

APE1-IN-2 (compound AP1) (2 mg/kg, IP, once every 3 days for 15 days) exhibits an antitumor effect on the A549 xenograft model[1].

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

Animal Model:	BALB/c nude mice (5 week-old, female, 16 ± 2 g of body weight bearing A549 xenograft tumors)[1]
Dosage:	2 mg/kg
Administration:	IP, once every 3 days for 15 days
Result:	Exhibited a 3.86-fold xenograft tumor inhibitory activity compared to Cisplatin. Did not significantly alter the body weight of mice, improving its sufficient safety.

REFERENCES

[1]. Yuan Y, et al. Pt(IV) Prodrug as a Potential Antitumor Agent with APE1 Inhibitory Activity. J Med Chem. 2022 Nov 24;65(22):15344-15357.

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