MDMX/MDM2-IN-2

Cat. No.: HY-149250

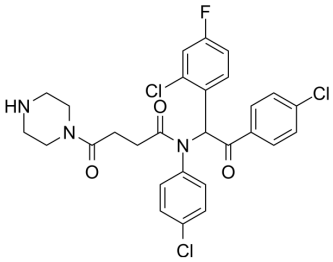
Molecular Formula: $C_{28}H_{25}Cl_3FN_3O_3$

Molecular Weight: 576.87

Target: MDM-2/p53; Apoptosis

Pathway: Apoptosis

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

MDMX/MDM2-IN-2 is a potent p53-MDM2/MDMX dual inhibitors with K_is of 0.23 μM and 2.45 μM for MDM2 and MDMX, respectively. MDMX/MDM2-IN-2 inhibits the binding of p53 and MDM2 proteins. MDMX/MDM2-IN-2 restores the function of p53 and enables cell cycle arrest and apoptosis. MDMX/MDM2-IN-2 inhibits cell migration and invasion. MDMX/MDM2-IN-2 has antitumor activity[1].

IC_{50} & Target

Ki: 0.23 μM (MDM2) and 2.45 μM (MDMX)[1]

In Vitro

MDMX/MDM2-IN-2 demonstrates moderate anti-proliferative activities against HCT116 and SH-SY5Y cells (IC_{50}=0.68 μM and 0.54 μM, respectively). MDMX/MDM2-IN-2 possesses low cytotoxicity on normal human lung epithelial BEAS-2B cells and LO2 liver cells (IC_{50}=17.96 μM and 15.93 μM, respectively)[1].

MDMX/MDM2-IN-2 (0.6-2.4 μM; 48 h) induces apoptosis of HCT116 and SH-SY5Y cells[1].

MDMX/MDM2-IN-2 (0.6-2.4 μM; 48 h) arrests the cell cycle in G1 phase[1].

MDMX/MDM2-IN-2 (0.6-2.4 μM; 48 h) increases the levels of p53 and its downstream targets, MDM2, MDMX, p21 and cleaved-caspase3[1].

MDMX/MDM2-IN-2 (0.4-0.8 μM) dramatically inhibits colony formation, migration and invasion of HCT116 and SH-SY5Y cells[1].

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

Apoptosis Analysis[1]

Cell Line:	HCT116 and SH-SY5Y cells
Concentration:	0.6, 1.2, 2.4 μM
Incubation Time:	48 h
Result:	The percentages of apoptotic HCT116 and SH-SY5Y cells were 13.63% and 15.69% with 0.6 μM. The percentage of apoptotic cells correspondingly increased to 37.6% and 40.8% with 2.4 μM.

Cell Cycle Analysis[1]

Cell Line:	HCT116 and SH-SY5Y cells

Concentration:	0.6, 1.2, 2.4 μM
Incubation Time:	48 h
Result:	There was an increase in the percentage of cancer cells at the G1 phase. Meanwhile, the percentage of G2 phase cells was relatively decreased.

Western Blot Analysis[1]

Cell Line:	HCT116 and SH-SY5Y cells
Concentration:	0.6, 1.2, 2.4 μM
Incubation Time:	48 h
Result:	Increased the levels of p53 and its downstream targets, MDM2, MDMX, p21 and cleaved-caspase3.

Cell Migration Assay [1]

Cell Line:	HCT116 and SH-SY5Y cells
Concentration:	0.4, 0.6, 0.8 μM
Incubation Time:	48 h
Result:	Significantly inhibited the migration and invasion in a dose-dependent manner.

REFERENCES

[1]. Hui-Juan Luo, et al. Structure-based discovery of novel α-aminoketone derivatives as dual p53-MDM2/MDMX inhibitors for the treatment of cancer. Eur J Med Chem. 2023 Apr 5;252:115282.

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