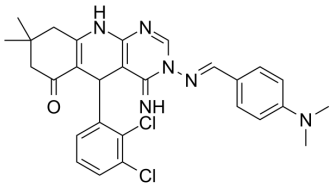
EGFR-IN-60

Cat. No.:	HY-147826
CAS No.:	2699877-43-3
Molecular Formula:	$C_{28}H_{28}Cl_2N_6O$
Molecular Weight:	535.47
Target:	EGFR; Apoptosis
Pathway:	JAK/STAT Signaling; Protein Tyrosine Kinase/RTK; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

EGFR-IN-60 (Compound 7d) shows obvious inhibition of $EGFR^{WT}$, $EGFR^{T790M}$, $EGFR^{L858R}$ and JAK3 with IC_{50}s of 83, 26, 53, and 69 nM, respectively. EGFR-IN-60 potently inhibits the growth of H1975 cells harboring $EGFR^{T790M}$ mutation (IC_{50}=1.32 μM) over A431 cells overexpressing $EGFR^{WT}$ (IC_{50}=4.96 μM). EGFR-IN-60 exhibits good oral absorption, potent and safe antitumor activity. EGFR-IN-60 induces cell death through apoptosis supported by increased Bax/Bcl-2 ratio[1].

IC_{50} & Target

EGFR	$EGFR^{L858R}$	$EGFR^{T790M}$	JAK3
0.083 μM (IC_{50})	0.053 μM (IC_{50})	0.026 μM (IC_{50})	0.069 μM (IC_{50})

In Vitro

EGFR-IN-60 (compound 7d) (3.25-88.46 μM, 48 hours) shows well antitumor activity against hepatocellular (HepG2), colorectal (HCT-116) and breast (MCF-7) cancer cells[1].

EGFR-IN-60 (compound 7d) (0.49-86.4 μM, 48 hours) shows cytotoxic activity against cancer cells[1].

EGFR-IN-60 (compound 7d) (0-5.27 μM, 24 hours) induces an increase in G2/M phase cells and induces apoptosis in HepG2, HCT-116, and MCF-7 cell lines[1].

EGFR-IN-60 (compound 7d) (0 μM, 10 μM, 24 hours) can induce apoptosis through up-regulation of Bax and down-regulation of Bcl-2[1].

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

Cell Proliferation Assay[1]

Cell Line:	Hepatocellular (HepG2), Colorectal (HCT-116), Breast (MCF-7) cancer cells
Concentration:	3.25-88.46 μM
Incubation Time:	48 hours
Result:	Inhibited HepG2 cells, HCT-116 cells, MCF-7 cells with IC_{50}values of 4.46 μM, 5.27 μM and 3.25 μM respectively.

Cell Cytotoxicity Assay[1]

Cell Line:	Overexpress $EGFR^{WT}$ human epidermoid carcinoma cells (A431), Mutant $EGFR^{T790M}$ cells NSCLC (H1975), Lung fibroblast cells (WI38), Amnion epithelial cells (WISH)
Concentration:	0.49-86.4 μM

Incubation Time:	48 hours
Result:	Showed cytotoxic activity against A431, H1975, WI38, WISH with IC_{50} value of 4.96 μM, 1.32 μM, 64.27 μM, 46.38 μM respectively.

Cell Cycle Analysis[1]

Cell Line:	HepG2, HCT-116, MCF-7
Concentration:	0 μM, 3.25 μM, 4.46 μM, 5.27 μM
Incubation Time:	24 hours
Result:	Resulted in an increase in the percentage of G2/M phase cells from 14.09% to 25.66% , from 15.87% to 38.51%, from 10.95% to 41.60% in HepG2, HCT-116, MCF-7 cell lines respectively.

Apoptosis Analysis[1]

Cell Line:	HepG2, HCT-116, MCF-7
Concentration:	3.25 μM, 4.46 μM, 5.27 μM
Incubation Time:	24 hours
Result:	Induced more apoptosis in MCF-7 cells comparing with HepG2 and HCT-116 cells.

Western Blot Analysis[1]

Cell Line:	HepG2, HCT-116, MCF-7
Concentration:	0 μM, 10 μM
Incubation Time:	24 hours
Result:	Showed the levels of pro-apoptotic protein Bax upgrading by 5.71, 8.15 and 16.51 fold and the levels of anti-apoptotic protein Bcl-2 down-regulating by 0.72, 0.53 and 0.31 fold in HepG2, HCT-116, MCF-7, respectively.

REFERENCES

[1]. Mennatallah A Shaheen, et al. Design, synthesis and biological evaluation of new series of hexahydroquinoline and fused quinoline derivatives as potent inhibitors of wild-type EGFR and mutant EGFR (L858R and T790M). Bioorg Chem. 2020 Dec;105:104274.

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