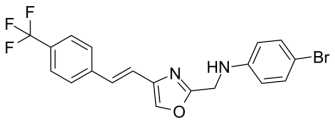
HER2-IN-9

Cat. No.:	HY-143323
Molecular Formula:	$C_{19}H_{14}BrF_3N_2O$
Molecular Weight:	423.23
Target:	EGFR
Pathway:	JAK/STAT Signaling; Protein Tyrosine Kinase/RTK
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

HER2-IN-9 is an orally active HER2 inhibitor, with an IC_{50} value of 0.03 μM. HER2-IN-9 inhibits HER-2 positive breast cancer cells proliferation and migration. HER2-IN-9 can be used in the research of breast cancers[1].

IC_{50} & Target

HER2
0.03 μM (IC_{50})

In Vitro

HER2-IN-9 (24 h) shows anti-proliferation activities against cancer cells (A549, HEPG2, MCF7, SKBR3)[1].
HER2-IN-9 (0-60 nM, 24 h) inhibits the migration of SKBR3 cells[1].
HER2-IN-9 (0-60 nM, 24 h) increases E-cadherin levels and decreases N-cadherin levels in SKBR3 cells[1].
HER2-IN-9 (0-60 nM, 24 h) suppresses the expression of p-HER-2, further inhibits the activation of the EMT signal pathway to inhibit the migration of SKBR3 cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	Cancer cells (A549, HEPG2, MCF7, SKBR3), normal cells (Beas2b, LO2, MCF-0A)
Concentration:	0.05-30 μM approximately
Incubation Time:	24 h
Result:	Inhibited cancer cell proliferation with IC_{50}s of 0.05-12.17 μM. Inhibited noamal cell proliferation with IC_{50}s of 15.4- 26.95 μM.

Western Blot Analysis[1]

Cell Line:	SKBR3 cells
Concentration:	0, 20, 40, 60 nM
Incubation Time:	24 h
Result:	Inhibited HER-2 phosphorylation with no significant change in total HER-2 protein levels. Down-regulated β-catenin, snail, and Vimentin level.

In Vivo

HER2-IN-9 (oral administration, 30 mg/kg, every two days) inhibits tumor growth SKBR3 orthotopic xenograft model[1].

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

Animal Model:	SKBR3 orthotopic xenograft model[1]
Dosage:	30 mg/kg
Administration:	Oral administration, every two days.
Result:	Inhibited the growth of cancer cells in vivo without noticeable toxic effects. Increased the level of cleaved-caspase 3 implicated in cell death pathways (immunohistochemistry assay in tumor).

REFERENCES

[1]. Xin-Yang Li, et al. Synthesis and evaluation of novel HER-2 inhibitors to exert anti-breast cancer ability through epithelial-mesenchymal transition (EMT) pathway. Eur J Med Chem. 2022 Jul 5;237:114325.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA