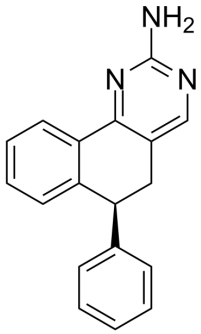
ARQ 069

Cat. No.:	HY-101544
CAS No.:	1314021-57-2
Molecular Formula:	$C_{18}H_{15}N_3$
Molecular Weight:	273.33
Target:	FGFR
Pathway:	Protein Tyrosine Kinase/RTK
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

ARQ 069, an analog of ARQ 523, inhibits FGFR in an enantiospecific manner. ARQ 069 targets the unphosphorylated, inactive forms of FGFR1/FGFR2 kinases (IC_{50}s of 0.84 μM and 1.23 μM, respectively). ARQ 069 inhibits FGFR1/FGFR2 autophosphorylation (IC_{50}s of 2.8 and 1.9 μM, respectively) through a mechanism in a non-ATP competitive dependent manner[1].

IC_{50} & Target

unphosphorylated FGFR1	unphosphorylated FGFR2	FGFR1 autophosphorylation	FGFR2 autophosphorylation
0.84 μM (IC_{50})	1.23 μM (IC_{50})	2.8 μM (IC_{50})	1.9 μM (IC_{50})

In Vitro

ARQ 069 (3.8-60 μM; for 2 hours) reduces the degree of phosphorylation of FGFR (predominantly FGFR2) in a concentration-dependent manner, without decreasing β-actin[1].
ARQ 069 shows an affinity for FGFR2 of 5.2 μM[1].
ARQ 069 inhibits FGFR phosphorylation in Kato III cells with an IC_{50} of 9.7 μM[1].
ARQ 069 targets the inactive forms of FGFR1 and FGFR2 kinases and inhibits their enzymatic activity. When ARQ 069 is preincubated with either phosphorylated FGFR1 or FGFR2, the potency of ARQ 069 in inhibiting Pyk2 phosphorylation is markedly reduced, with IC_{50} values determined to be greater than 30 and 24.8 μM for FGFR1 and FGFR2, respectively. ARQ 069 exhibits at least a 20-fold preference for binding to the unphosphorylated, inactive forms of FGFR1 and FGFR2[1].
ARQ 068 is the R-enantiomer, and ARQ 069 is the S-enantiomer[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	Kato III human gastric carcinoma cells
Concentration:	3.8, 7.5, 15, 30, 60 μM
Incubation Time:	For 2 hours
Result:	Reduced the degree of phosphorylation of FGFR (predominantly FGFR2) in a concentration-dependent manner, without decreasing β-actin.

REFERENCES

[1]. Eathiraj S, et al. A novel mode of protein kinase inhibition exploiting hydrophobic motifs of autoinhibited kinases: discovery of ATP-independent inhibitors of fibroblast growth factor receptor. J Biol Chem. 2011 Jun 10;286(23):20677-87.

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