PKI-166 hydrochloride

Cat. No.: HY-110328

CAS No.: 2230253-82-2

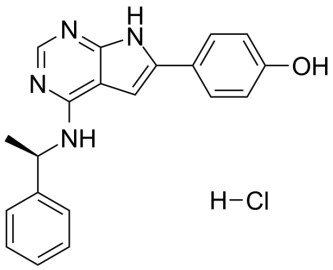
Molecular Formula: $C_{20}H_{19}ClN_4O$

Molecular Weight: 366.84

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

PKI-166 hydrochloride is a potent, selective and orally active EGFR tyrosine kinase inhibitor, with an IC_{50} of 0.7 nM[1].

IC_{50} & Target

IC50: 0.7 nM (EGFR tyrosine kinase)[1]

In Vitro

PKI-166 hydrochloride (0-0.5 μM; 1 hour; pretreatment) inhibits EGFR autophosphorylation in human pancreatic cancer cells [1].

PKI-166 hydrochloride (0.03 μM; 6 days) enhances the cytotoxicity mediated by gemcitabine[1].

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

Western Blot Analysis[1]

Cell Line:	L3.6pl cells
Concentration:	0.01 μM, 0.05 μM, 0.5 μM
Incubation Time:	1 hour
Result:	Inhibited EGFR autophosphorylation in a dose-dependent manner.

Cell Cytotoxicity Assay[1]

Cell Line:	L3.6pl cells
Concentration:	0.03 μM
Incubation Time:	6 days
Result:	Enhanced the cytotoxicity mediated by gemcitabine.

In Vivo

PKI-166 hydrochloride (100 mg/kg; p.o.; daily; for 29 days) inhibits of pancreatic cancer growth[1].

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Animal Model:	Male athymic nude mice with L3.6pl cells xenograft (8-12 weeks)[1]
Dosage:	100 mg/kg

Administration:	Oral administration; daily; for 29 days
Result:	Significantly decreased median tumor volume.

REFERENCES

[1]. Bruns CJ, et al. Blockade of the epidermal growth factor receptor signaling by a novel tyrosine kinase inhibitor leads to apoptosis of endothelial cells and therapy of human pancreatic carcinoma. Cancer Res. 2000 Jun 1;60(11):2926-35.

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