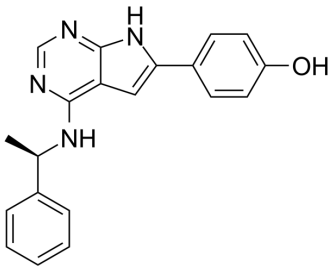
PKI-166

Cat. No.:	HY-117155
CAS No.:	187724-61-4
Molecular Formula:	$C_{20}H_{18}N_4O$
Molecular Weight:	330.38
Target:	EGFR
Pathway:	JAK/STAT Signaling; Protein Tyrosine Kinase/RTK
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 125 mg/mL (378.35 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.0268 mL	15.1341 mL	30.2682 mL
	5 mM	0.6054 mL	3.0268 mL	6.0536 mL
	10 mM	0.3027 mL	1.5134 mL	3.0268 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

PKI-166 is a potent, selective and orally bioavailable 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

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

PKI-166 (0.03μ M; 6 days) enhanced 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 (100 mg/kg; p.o.; daily; day 7-day 35 after xenograft) inhibits of pancreatic cancer growth[1].

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

Animal Model:	Male athymic nude mice with L3.6pl cells xenograft (8–12 weeks)[1]
Dosage:	100 mg/kg
Administration:	Oral administration; daily; from day 7 to day 35 after xenograft
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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