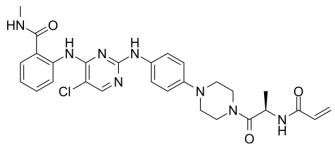
FAK-IN-2

Cat. No.:	HY-144448
CAS No.:	2872588-02-6
Molecular Formula:	$C_{28}H_{31}ClN_8O_3$
Molecular Weight:	563.05
Target:	FAK; Apoptosis
Pathway:	Protein Tyrosine Kinase/RTK; Apoptosis
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 : 100 mg/mL (177.60 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.7760 mL	8.8802 mL	17.7604 mL
	5 mM	0.3552 mL	1.7760 mL	3.5521 mL
	10 mM	0.1776 mL	0.8880 mL	1.7760 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: 2.5 mg/mL (4.44 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (4.44 mM); Clear solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: 2.5 mg/mL (4.44 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

FAK-IN-2 is a potent and orally active focal adhesion kinase (FAK) inhibitor, with anticancer activity (FAK IC_{50}= 35 nM). FAK-IN-2 covalently inhibits the autophosphorylation of FAK in a dose-dependent manner, and inhibits the clone formation and migration of tumor cells, inducing apoptosis[1].

IC_{50} & Target

IC_{50}: 35 nM (FAK)[1]

In Vitro

FAK-IN-2 (compound 11w) (0-5 μM; 72 hours) has high anti-proliferation activities on cancer cell lines, as well as certain

toxicity on normal cell lines[1].
FAK-IN-2 (0-30 nM; 14 days) can remarkably affect HCT-116 cells clone formation in a dose-dependent manner[1].
FAK-IN-2 (10-500 nM; 24 and 48 hours) significantly inhibits the migration of HCT116 cells at both 24 h and 48 h in a dose-dependent manner[1].
FAK-IN-2 (0.001-10 μM; 4 and 24 hours) inhibits the phosphorylation of FAK and its downstream proteins from multiple pathways[1].
FAK-IN-2 (0.01-1 μM; 24 or 48 hours) induces strong cell cycle arrest at the G2/M phase and apoptosis[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay

Cell Line:	Hela, HCT116, MDA-MB-231, H9C2, L929, LO2, HEK293[1]
Concentration:	0-5 μM
Incubation Time:	72 hours
Result:	Showed high anti-proliferation activities on cancer cell lines, as well as certain toxicity on normal cell lines.

Western Blot Analysis

Cell Line:	HCT116 cells[1]
Concentration:	0.001, 0.01, 0.1, 1 and 10 μM
Incubation Time:	4 and 24 hours
Result:	Inhibited the phosphorylation of FAK and its downstream proteins from multiple pathways.

Cell Cycle Analysis

Cell Line:	HCT116 cells[1]
Concentration:	0.01, 0.05, 0.1 and 0.5 μM for 24 hours; 0.01, 0.05, 0.1, 0.3 and 1 μM for 48 hours
Incubation Time:	24 and 48 hours
Result:	Induced strong cell cycle arrest at the G2/M phase and apoptosis.

In Vivo

FAK-IN-2 (5 and 15 mg/kg; 16 days; once daily) has potent antitumor effects in model mice with a dose-dependent manner without significant toxicity[1]Pharmacokinetic Parameters of FAK-IN-2 in male Sprague-Dawley rats[1].

	PO (5 mg/kg)	IV (5 mg/kg)
C_{max} (μg/L)	239.87	2965.27
T_{max} (h)	1.44	0.08
$T_{1/2}$ (h)	4.70	7.57
Clz (L/h/kg)	9.92	2.19
AUC_{0-t} (μg*h/L)	512.75	2439.06

F %	21.02%

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

Animal Model:	Female Balb/C nu/nu mice (HCT116-injected)[1]
Dosage:	5 and 15 mg/kg
Administration:	16 days; once daily
Result:	Displayed potent antitumor effects in a dose-dependent manner without significant toxicity.

REFERENCES

[1]. Chen T, et al. Design, synthesis, and biological evaluation of novel covalent inhibitors targeting focal adhesion kinase. Bioorg Med Chem Lett. 2021;54:128433.

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