Riviciclib hydrochloride

Cat. No.: HY-16559

CAS No.: 920113-03-7

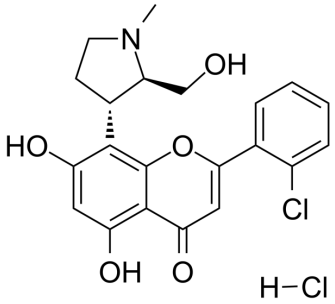
Molecular Formula: $C_{21}H_{21}Cl_2NO_5$

Molecular Weight: 438.3

Target: CDK; Apoptosis

Pathway: Cell Cycle/DNA Damage; Apoptosis

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (114.08 mM; Need ultrasonic)
H_2O : 25 mg/mL (57.04 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.2815 mL	11.4077 mL	22.8154 mL
5 mM	0.4563 mL	2.2815 mL	4.5631 mL
10 mM	0.2282 mL	1.1408 mL	2.2815 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: ≥ 2.08 mg/mL (4.75 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (4.75 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.75 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Riviciclib hydrochloride (P276-00) is a potent cyclin-dependent kinase (CDK) inhibitor, which inhibits CDK9-cyclinT1, CDK4-cyclin D1, and CDK1-cyclinB with IC_{50}s of 20 nM, 63 nM, and 79 nM, respectively[1][2].Riviciclib hydrochloride (P276-00) shows antitumor activity on cisplatin-resistant cells[3].

IC_{50} & Target

CDK9- Cyclin T1 0.020 μM (IC_{50})	cdk4-cyclin D1 0.063 μM (IC_{50})	CDK1-Cyclin B 0.079 μM (IC_{50})	cdk2-cyclin A 0.224 μM (IC_{50})
cdk2-cyclin E	cdk6-cyclin D3	CDK9-cyclin H	

	2.500 μM (IC_{50})	0.396 μM (IC_{50})	2.900 μM (IC_{50})

In Vitro

Riviciclib hydrochloride (1.5-5 μM; 72 hours) shows no detectable cells in G1 and G2 in promyelocytic leukemia cells and arrest of cells in G1 in synchronized human non-small cell lung carcinoma (H-460) and human normal lung fibroblast (WI-38) cells[3].

Riviciclib hydrochloride (3-24 hours; 1.5 μM) reduces cyclin D1, Cdk4, and Rb levels in H-460 cells. Rb (retinoblastoma) phosphorylation at Ser^{780} decrease at 3 h[2].

Riviciclib hydrochloride shows activity in human cancer cell lines, such as colon carcinoma, osteosarcomal, cervical carcinoma, and bladder carcinoma cells[2].

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

Cell Cycle Analysis[3]

Cell Line:	Promyelocytic leukemia cells (HL-60 cells), non-small cell carcinoma (H-460) cells, human normal lung fibroblast (WI-38) cells
Concentration:	1.5, 5 μM
Incubation Time:	72 hours
Result:	Showed apoptosis at the end of 24 h and no detectable cells were present in G1 and G2 in HL-60 cells. Caused an exclusive G1 arrest of synchronous population of cancerous cells H-460 cells and normal cells WI-38.

Western Blot Analysis[2]

Cell Line:	H-460 cells; MCF-7 cells
Concentration:	1.5 μM
Incubation Time:	3, 6, 9, 12, 24 hours
Result:	Reduced cyclin D1, Cdk4, and Rb levels in H-460 cells. Rb (retinoblastoma) phosphorylation at Ser^{780} decrease at 3 h. Decreased protein levels of cyclin D1 and Cdk4 levels staring at 6 and 9 h in MCF-7 cells, respectively, and accompanied by a decrease in phosphorylation of Rb at Ser^{780} from 6 h onward, followed by reduced Rb levels at 24 h.

In Vivo

Riviciclib hydrochloride (administered i.p.; 35 kg/mg daily for 10 days, in human xenograft mode with severe combined immunodeficient mice) shows significant inhibition in the growth of human colon carcinoma HCT-116 xenograft[3].

Riviciclib hydrochloride (administered via i.p.; 50 mg/kg once daily; 30 mg/kg twice daily for 18 treatments, in human xenograft mode with severe combined immunodeficient mice) significantly inhibited growth[3].

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

Animal Model:	Human xenograft mode with HCT-116 tumor model (severe combined immunodeficient mice)[3]
Dosage:	35 mg/kg
Administration:	Administered i.p.; daily for 10 days
Result:	Given 35 mg/kg showed significant inhibition in the growth.

Animal Model:	Human xenograft model with H-460 tumor xenograft (severe combined immunodeficient mice)[3]

Dosage:	50 mg/kg; 30 mg/kg
Administration:	Administered i.p.; 50 mg/kg once daily for 20 days; Administered i.p.; 30 mg/kg twice daily for 18 treatments
Result:	Given 50 mg/kg and 30 mg/kg twice daily significantly inhibited growth.

CUSTOMER VALIDATION

- Elife. 2020 Dec 7;9:e61405.
- Int J Mol Sci. 2022 Feb 24;23(5):2493.

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REFERENCES

[1]. Roskoski R Jr,Cyclin-dependent protein kinase inhibitors including palbociclib as anticancer drugs.Pharmacol Res. 2016 May;107:249-275.

[2]. Joshi KS, et al. In vitro antitumor properties of a novel cyclin-dependent kinase inhibitor, P276-00. Mol Cancer Ther. 2007 Mar;6(3):918-25.

[3]. Joshi KS,et al. P276-00, a novel cyclin-dependent inhibitor induces G1-G2 arrest, shows antitumor activity on cisplatin-resistant cells and significant in vivo efficacy in tumor models. Mol Cancer Ther. 2007 Mar;6(3):926-34.

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