Riviciclib

Cat. No.: HY-16559A

CAS No.: 920113-02-6

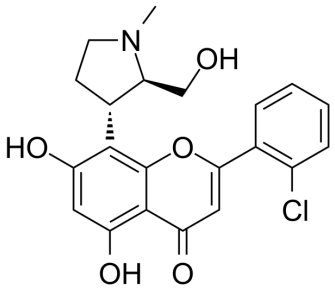
Molecular Formula: $C_{21}H_{20}ClNO_5$

Molecular Weight: 401.84

Target: CDK

Pathway: Cell Cycle/DNA Damage

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Riviciclib (P276-00 free base) 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 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 2.540 μM (IC_{50})	cdk6-cyclin D3 0.396 μM (IC_{50})	CDK9-cyclin H 2.900 μM (IC_{50})	

In Vitro

Riviciclib (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 (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 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 (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 (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 inhibits 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.

Caution: Product has not been fully validated for medical applications. For research use only.

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