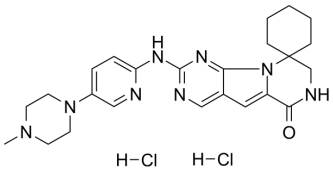
Trilaciclib hydrochloride

Cat. No.:	HY-101467A
CAS No.:	1977495-97-8
Molecular Formula:	$C_{24}H_{32}Cl_2N_8O$
Molecular Weight:	519.47
Target:	CDK
Pathway:	Cell Cycle/DNA Damage
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

H_2O : 25.64 mg/mL (49.36 mM; ultrasonic and adjust pH to 2 with HCl)
DMSO : 1.1 mg/mL (2.12 mM; Need ultrasonic)

Preparing Stock Solutions

Mass / Solvent / Concentration	1 mg	5 mg	10 mg
1 mM	1.9250 mL	9.6252 mL	19.2504 mL
5 mM	0.3850 mL	1.9250 mL	3.8501 mL
10 mM	0.1925 mL	0.9625 mL	1.9250 mL

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

BIOLOGICAL ACTIVITY

Description

Trilaciclib (G1T28) hydrochloride is an orally active CDK4/6 inhibitor with IC_{50} values of 1 nM and 4 nM for CDK4 and CDK6, respectively. Trilaciclib hydrochloride can effectively inhibit tumor cell proliferation and reduce the hematological toxicity caused by chemotherapy. Trilaciclib hydrochloride attenuates apoptosis and myelosuppression induced by 5FU (HY-90006) chemotherapy[1].

IC_{50} & Target

Cdk4/cyclin D1	cdk6/cyclin D3
1 nM (IC_{50})	4 nM (IC_{50})

In Vitro

Trilaciclib hydrochloride (10-1000 nM; 24 h) reversibly modulates the proliferation of mouse and canine bone marrow hematopoietic stem and progenitor cells[1].
Trilaciclib hydrochloride (10-1000 nM; 24 h) can arrest the cell cycle of CDK4/6-dependent cells in the G1 phase, with an EC50 of 30 nM for HS68[1].
Trilaciclib hydrochloride (300 nM; 16 or 48 h) protects CDK4/6 dependent cells (HS68, WM2664) from chemotherapy-induced damage, and attenuates chemotherapy-induced-apoptosis[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	HS68, WM2664 cells
Concentration:	300 nM
Incubation Time:	8h for measure γH2AX foci (DNA damage); 48 hours to measure caspase 3/7 activity (apoptosis).
Result:	Elicited a robust dose-dependent decrease in caspase 3/7 activation suggesting an attenuation of apoptosis. Resulted a dose-dependent decrease in γH2AX foci in all DNA damaging chemotherapies tested (carboplatin, doxorubicin, etoposide, camptothecin).

Cell Cycle Analysis[1]

Cell Line:	HS68, WM2664 cells
Concentration:	10, 30, 100, 300, 1000 nM
Incubation Time:	24 h
Result:	Inhibited only CDK4/6-dependent cells, with EC50 of 30 nM for HS68 cell. Singnificantly decreased the S phase cell numbers, increased the G1 phase cell numbers.

Western Blot Analysis[1]

Cell Line:	HS68, WM2664 cells
Concentration:	10, 30, 100, 300, 1000 nM
Incubation Time:	16 h
Result:	Blocked RB phosphorylation in the RB dependent cell lines by 16 hours post exposure, while the CDK4/6-independent cell line (A2058) exhibits no RB or pRB expression. Suggesting an attenuation of chemotherapy-induced DNA damag

In Vivo

Trilaciclib hydrochloride (50-150 mg/kg; po; single dose) protects mouse bone marrow cells from chemotherapy-induced apoptosis and attenuates chemotherapy-induced myelosuppression in vivo. Trilaciclib hydrochloride at 150 mg/kg reduces HSPC damage induced by 5FU (150 mg/kg; ip) chemotherapy, thereby accelerating blood count recovery after chemotherapy[1].

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

Animal Model:	FVB/n female C57Bl6 mice[1]
Dosage:	50, 100, 150 mg/kg
Administration:	Po; single dose; treated followed 11 or 23 hours later by a single injection of EdU (HY-118411) (100 µg; ip); mouse were euthanized 1 hour after EdU injection.
Result:	Showed a dose-dependent decrease in caspase 3/7 activation. Attenuated chemotherapy-induced myelosuppression in mice.

CUSTOMER VALIDATION

- Department of Biochemistry. 2020 Oct.

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REFERENCES

[1]. Bisi JE, et al. Preclinical Characterization of G1T28: A Novel CDK4/6 Inhibitor for Reduction of Chemotherapy-Induced Myelosuppression. Mol Cancer Ther. 2016 May;15(5):783-93.

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