Samuraciclib

Cat. No.: HY-103712

CAS No.: 1805833-75-3

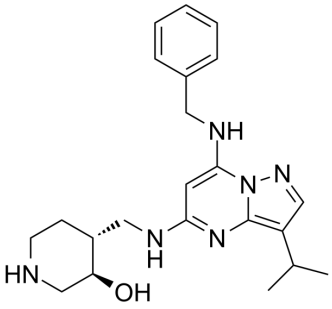
Molecular Formula: $C_{22}H_{30}N_6O$

Molecular Weight: 394.51

Target: CDK; Apoptosis

Pathway: Cell Cycle/DNA Damage; Apoptosis

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



BIOLOGICAL ACTIVITY

Description

Samuraciclib (CT7001) is a potent, selective, ATP-competitive and orally active CDK7 inhibitor, with an IC_{50} of 41 nM. Samuraciclib displays 45-, 15-, 230- and 30-fold selectivity over CDK1, CDK2 (IC_{50} of 578 nM), CDK5 and CDK9, respectively. Samuraciclib inhibits the growth of breast cancer cell lines with GI_{50} values between 0.2-0.3 μM. Samuraciclib has anti-tumor effects[1][2].

IC_{50} & Target

CDK7/CycH/MAT1 41 nM (IC_{50})	CDK2/cycE1 578 nM (IC_{50})	CDK1 1.8 μM (IC_{50})	CDK4 49 μM (IC_{50})
CDK5 9.4 μM (IC_{50})	CDK6 34 μM (IC_{50})	CDK9 1.2 μM (IC_{50})	

In Vitro

Samuraciclib (ICEC0942; 0-10 μM; 24 hours; HCT116 cells) treatment promotes cell apoptosis[1].
Samuraciclib (ICEC0942; 0-10 μM; 24 hours; HCT116 cells) treatment induces cell cycle arrest[1].
Samuraciclib (ICEC0942; 0-10 μM; 0-24 hours; HCT116 cells) treatment inhibits the phosphorylation of PolII CTD in a dose and time dependent manner in HCT116 colon cancer cells. Samuraciclib also inhibits phosphorylation of CDK1, CDK2 and retinoblastoma[1].
Samuraciclib (ICEC0942) inhibits the growth of MCF7, T47D, MDA-MB-231, HS578T, MDA-MB-468, MCF10A and HMEC cells with GI_{50} values of 0.18 μM, 0.32 μM, 0. 33 μM, 0.21 μM, 0.22 μM, 0.67 μM and 1.25 μM, respectively[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Apoptosis Analysis[1]

Cell Line:	HCT116 cells
Concentration:	0 μM, 0.1 μM, 1 μM and 10 μM
Incubation Time:	24 hours
Result:	Induced caspase 3/7 and demonstrated PARP cleavage.

Cell Cycle Analysis[1]

Cell Line:	HCT116 cells
Concentration:	0 μM, 0.01 μM, 0.1 μM, 1 μM and 10 μM

Incubation Time:	24 hours
Result:	Showed accumulation of cells in G2/M.

Western Blot Analysis[1]

Cell Line:	HCT116 cells
Concentration:	0 μM, 0.1 μM, 1 μM and 10 μM
Incubation Time:	0 hour, 4 hours, 8 hours, 16 hours or 24 hours
Result:	PolII CTD phosphorylation was inhibited in a dose and time dependent manner in HCT116 colon cancer cells.

In Vivo

Samuraciclib (ICEC0942; 100 mg/kg; oral gavage; daily; for 14 days; female nu/nu-BALB/c athymic nude mice) treatment inhibits tumor growth by 60% at day 14, and is accompanied by highly significant reductions in PolII Ser2 and Ser5 phosphorylation in PBMCs and in tumors[1].

The combination of Samuraciclib (ICEC0942) and ICI 47699 treatment shows complete growth arrest of estrogen receptor (ER)-positive tumor xenografts[1].

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

Animal Model:	Female nu/nu-BALB/c athymic nude mice (7-week old) with MCF7 cells[1].
Dosage:	100 mg/kg
Administration:	Oral gavage; daily; for 14 days
Result:	At day 14, tumor growth was inhibited by 60%.

CUSTOMER VALIDATION

- Proc Natl Acad Sci U S A. 2019 Jun 25;116(26):12986-12995.
- Cell Death Dis. 2019 Aug 9;10(8):602.
- Int J Mol Sci. 2022 Feb 24;23(5):2493.

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REFERENCES

[1]. Hazel P, et al. Inhibitor Selectivity for Cyclin-Dependent Kinase 7: A Structural, Thermodynamic, and Modelling Study. ChemMedChem. 2017 Mar 7;12(5):372-380.

[2]. Patel H, et al. ICEC0942, an Orally Bioavailable Selective Inhibitor of CDK7 for Cancer Treatment. Mol Cancer Ther. 2018 Jun;17(6):1156-1166.

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

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