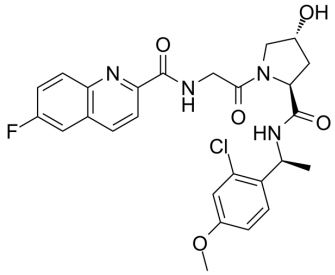
CAM833

Cat. No.:	HY-150147
CAS No.:	2758364-02-0
Molecular Formula:	$C_{26}H_{26}ClFN_4O_5$
Molecular Weight:	528.96
Target:	RAD51; Apoptosis
Pathway:	Cell Cycle/DNA Damage; 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 : 125 mg/mL (236.31 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.8905 mL	9.4525 mL	18.9050 mL
	5 mM	0.3781 mL	1.8905 mL	3.7810 mL
	10 mM	0.1891 mL	0.9453 mL	1.8905 mL

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

BIOLOGICAL ACTIVITY

Description

CAM833 is a potent orthosteric inhibitor of the interaction between BRCA2 and RAD51 with a K_d of 366 nM against the ChimRAD51 protein. CAM833 also inhibits RAD51 oligomerization. CAM833 increases the progression of G2/M-arrested cells into apoptosis[1].

IC_{50} & Target

K_d: 355 nM (ChimRAD51, measured by FP), 366 nM (ChimRAD51, measured by ITC)[1]

In Vitro

CAM833 (3.125-50 μM; 24 h) causes a concentration-dependent decrease in RAD51 foci and subsequent increase in DNA damage[1].

CAM833 (25 μM) inhibits RAD51 molecular clustering at DNA damage sites and suppresses extended RAD51 filament assembly[1].

CAM833 (0-50 μM) inhibits DNA repair by homologous recombination[1].

CAM833 (20 μM; 0-72 h) potentiates radiation-induced cell-cycle arrest and increases apoptosis over time in HCT116 cells[1].

CAM833 (0.1-100 μM; 96 h) causes a dose-dependent growth inhibition of multiple cancer-derived human cell lines that is enhanced when combined with ionizing radiation[1].

CAM833 (20 μM; 96 h) potentiates the growth suppressive effect of PARP1 inhibition in BRCA2 wild-type cells[1].

CAM833 (96 h) alone inhibits the growth of HCT116 colon carcinoma cells with a GI_{50} (50% growth inhibition) of 38 μM, when combined with 3 Gy IR, CAM833 suppresses the growth of HCT116 cells with a GI_{50} of 14 μM[1].
The quinoline of CAM833 occupies a hotspot, the Phe-binding pocket on RAD51 and the methyl of the substituted α-methylbenzyl group occupies the Ala-binding pocket[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Cycle Analysis[1]

Cell Line:	HCT116 cells
Concentration:	20 μM
Incubation Time:	0-72 h
Result:	In the control the percentage of cells in the apoptotic subG1 fraction remains below 5% throughout, in the compound-treated cells this rises progressively to peak at 15% at 48 hours.

REFERENCES

[1]. Scott DE, et al. A small-molecule inhibitor of the BRCA2-RAD51 interaction modulates RAD51 assembly and potentiates DNA damage-induced cell death. Cell Chem Biol. 2021 Jun 17;28(6):835-847.e5.

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