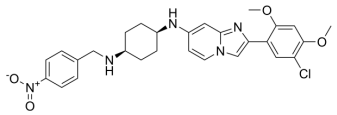
OTS193320

Cat. No.:	HY-122182
CAS No.:	2093401-33-1
Molecular Formula:	$C_{28}H_{30}ClN_5O_4$
Molecular Weight:	536.02
Target:	Histone Methyltransferase; Apoptosis
Pathway:	Epigenetics; Apoptosis
Storage:	Powder -20°C 3 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 62.5 mg/mL (116.60 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.8656 mL	9.3280 mL	18.6560 mL
	5 mM	0.3731 mL	1.8656 mL	3.7312 mL
	10 mM	0.1866 mL	0.9328 mL	1.8656 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: ≥ 6.25 mg/mL (11.66 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 6.25 mg/mL (11.66 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

OTS193320, a imidazo[1,2-a]pyridine compound, is a SUV39H2 methyltransferase activity inhibitor. OTS193320 decreases global histone H3 lysine 9 tri-methylation levels in breast cancer cells and triggers apoptotic cell death. Combination of OTS193320 with Doxorubicin (DOX; HY-15142A) results in reduction of γ-H2AX levels as well as cancer cell viability compared to a single agent OTS193320 or DOX[1].

IC_{50} & Target

SUV39H2/KMT1B

In Vitro

OTS193320 (0.125-0.5 μM; 24 hours) has growth inhibitory effect on breast cancer cell lines. OTS193320 exhibits a high inhibitory effect against SUV39H2 enzymatic activity (IC_{50}=22.2 nM) and a growth suppressive effect of SUV39H2-positive A549 lung cancer cells (IC_{50}=0.38 μM)[1].
OTS193320 (0.5 μM; 48 hours) induces apoptosis in breast cancer cells[1].

OTS193320 (0.125-0.5 μM; 24 hours) causes attenuation of H3K9me3 levels in a dose-dependent manner[1].
OTS193320 sensitizes breast cancer cells to doxorubicin via attenuation of γ-H2AX. Combination of OTS193320 and doxorubicin (DOX) significantly attenuates cancer cell viability in vitro, compared to single agent treatment of either drug[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	MCF-7, SK-BR-3, ZR-75-1, T-47D, MDA-MB-231, BT-20 breast cancer cell lines
Concentration:	0-1 μM
Incubation Time:	72 hours
Result:	Had Growth inhibitory effect on MCF-7, SK-BR-3, ZR-75-1, T-47D, MDA-MB-231, and BT-20 breast cancer cell lines with IC_{50} values from 0.41 to 0.56 μM, respectively.

Apoptosis Analysis[1]

Cell Line:	MDA-MB-231 and BT-20 cells
Concentration:	0.5 μM
Incubation Time:	48 hours
Result:	Showed an increase in the number of cells at early- and late-stage apoptosis.

Western Blot Analysis[1]

Cell Line:	MDA-MB-231 and BT-20 cells
Concentration:	0.125, 0.25, 0.5 μM
Incubation Time:	24 hours
Result:	Caused attenuation of H3K9me3 levels in a dose-dependent manner.

REFERENCES

[1]. Theodore Vougiouklakis, et al. Development of novel SUV39H2 inhibitors that exhibit growth suppressive effects in mouse xenograft models and regulate the phosphorylation of H2AX. Oncotarget. 2018 Aug 7;9(61):31820-31831.

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