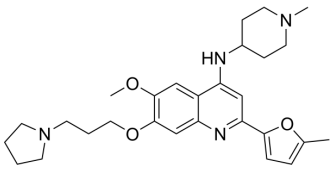
CM-272

Cat. No.:	HY-101925
CAS No.:	1846570-31-7
Molecular Formula:	$C_{28}H_{38}N_4O_3$
Molecular Weight:	478.63
Target:	Histone Methyltransferase; DNA Methyltransferase; Apoptosis
Pathway:	Epigenetics; 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 (261.16 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.0893 mL	10.4465 mL	20.8930 mL
5 mM	0.4179 mL	2.0893 mL	4.1786 mL
10 mM	0.2089 mL	1.0446 mL	2.0893 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: ≥ 2.08 mg/mL (4.35 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CM-272 is a first-in-class, potent, selective, substrate-competitive and reversible dual G9a/DNA methyltransferases (DNMTs) inhibitor with antitumor activities. CM-272 inhibits G9a, DNMT1, DNMT3A, DNMT3B and GLP with IC_{50}s of 8 nM, 382 nM, 85 nM, 1200 nM and 2 nM, respectively. CM-272 inhibits cell proliferation and promotes apoptosis, inducing IFN-stimulated genes and immunogenic cell death[1].

IC_{50} & Target

G9a 8 nM (IC_{50})	EHMT1/GLP/KMT1D 2 nM (IC_{50})	DNMT1 382 nM (IC_{50})	DNMT3A 85 nM (IC_{50})
DNMT3B 1200 nM (IC_{50})			

In Vitro

CM-272 (100-1000 nM; 12-72 hours; CEMO-1, MV4-11 and OCI-Ly10 cell lines) treatment inhibits cell proliferation in a dose-

and time-dependent manner[1].
CM-272 (100-1000 nM; 24 hours; CEMO-1, MV4-11 and OCI-Ly10 cell lines) treatment blocks cell cycle progression[1].
CM-272 (100-1000 nM; 12-72 hours; CEMO-1, MV4-11, and OCI-Ly10 cell lines) treatment induces apoptosis in ALL, AML and DLBCL cell lines in a dose- and time-dependent manner[1].
CM-272 after 48 h of treatment CEMO-1 acute lymphoblastic leukaemia (ALL) cell line, MV4-11 acute myeloid leukaemia (AML) cell line, and OCI-Ly10 diffuse large B-cell lymphoma (DLBCL) cell line, the GI_{50} values of 218 nM, 269 nM and 455 nM, respectively, and is associated with a decrease in global levels of H3K9me2 and 5mC[1].
The therapeutic activity of CM-272 relies on the early activation of the type I IFN response in tumor cells, potentially leading to the induction of cell-autonomous immunogenic death in tumor cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	CEMO-1, MV4-11 and OCI-Ly10 cell lines
Concentration:	125 nM, 250 nM, 500 nM (CEMO-1 cells); 135 nM, 270 nM, 540 nM (MV4-11 cells); 100 nM, 400 nM, 1000 nM (OCI-Ly10 cells)
Incubation Time:	12 hours, 24 hours, 48 hours and 72 hours
Result:	Inhibited cell proliferation in a dose- and time-dependent manner.

Cell Cycle Analysis[1]

Cell Line:	CEMO-1, MV4-11 and OCI-Ly10 cell lines
Concentration:	125 nM, 250 nM, 500 nM (CEMO-1 cells); 135 nM, 270 nM, 540 nM (MV4-11 cells); 100 nM, 400 nM, 1000 nM (OCI-Ly10 cells)
Incubation Time:	24 hours
Result:	Blocked cell cycle progression.

Apoptosis Analysis[1]

Cell Line:	CEMO-1, MV4-11 and OCI-Ly10 cell lines
Concentration:	125 nM, 250 nM, 500 nM (CEMO-1 cells); 135 nM, 270 nM, 540 nM (MV4-11 cells); 100 nM, 400 nM, 1000 nM (OCI-Ly10 cells)
Incubation Time:	12 hours, 24 hours, 48 hours and 72 hours
Result:	Induced apoptosis in ALL, AML and DLBCL cell lines in a dose- and time-dependent manner.

In Vivo

CM-272 (2.5 mg/kg; intravenous injection; daily; for 28 days; female Rag2 $^{/}$ γc $^{/}$ mice) treatment significantly prolongs survival of CEMO-1 cells xenogeneic models[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Female BALB/Ca-Rag2$^{-/-}$γc$^{-/-}$ mice (6–8-week-old) with CEMO-1 cells[1]
Dosage:	2.5 mg/kg
Administration:	Intravenous injection; daily; for 28 days
Result:	Induced a statistically significant increase in overall survival (OS) in mice.

CUSTOMER VALIDATION

- Biochim Biophys Acta Gen Subj. 2023 Jun 23;130417.

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REFERENCES

[1]. San José-Enériz E, et al. Discovery of first-in-class reversible dual small molecule inhibitors against G9a and DNMTs in hematological malignancies. Nat Commun. 2017 May 26;8:15424.

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