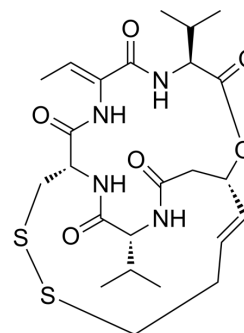


## Romidepsin

Cat. No.:	HY-15149
CAS No.:	128517-07-7
Molecular Formula:	C <sub>24</sub> H <sub>36</sub> N <sub>4</sub> O <sub>6</sub> S <sub>2</sub>
Molecular Weight:	540.7
Target:	HDAC; Apoptosis
Pathway:	Cell Cycle/DNA Damage; Epigenetics; Apoptosis
Storage:	Powder -20°C 3 years

\* The compound is unstable in solutions, freshly prepared is recommended.



### SOLVENT & SOLUBILITY

#### In Vitro

DMSO : ≥ 100 mg/mL (184.95 mM)  
 \* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	1.8495 mL	9.2473 mL	18.4945 mL
	5 mM	0.3699 mL	1.8495 mL	3.6989 mL
	10 mM	0.1849 mL	0.9247 mL	1.8495 mL

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

#### In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline  
 Solubility: ≥ 2.08 mg/mL (3.85 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)  
 Solubility: 2.08 mg/mL (3.85 mM); Suspended solution; Need ultrasonic
- Add each solvent one by one: 10% DMSO >> 90% corn oil  
 Solubility: ≥ 2.08 mg/mL (3.85 mM); Clear solution

### BIOLOGICAL ACTIVITY

#### Description

Romidepsin (FK 228) is a Histone deacetylase (HDAC) inhibitor with anti-tumor activities. Romidepsin (FK 228) inhibits HDAC1, HDAC2, HDAC4, and HDAC6 with IC<sub>50</sub>s of 36 nM, 47 nM, 510 nM and 1.4 μM, respectively<sup>[1]</sup>. Romidepsin (FK 228) is produced by Chromobacterium violaceum, induces cell G2/M phase arrest and apoptosis<sup>[2]</sup>.

#### IC<sub>50</sub> & Target

HDAC1 36 nM (IC <sub>50</sub> )	HDAC2 47 nM (IC <sub>50</sub> )	HDAC4 510 nM (IC <sub>50</sub> )	HDAC6 14000 nM (IC <sub>50</sub> )
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#### In Vitro

Romidepsin (0-72 hours; 0-80 nM) inhibits proliferation of HCC cells in dose-dependent manner<sup>[2]</sup>.

Romidepsin (0-48 hours; 0-60 nM) leads to a time- and dose-dependent induction of cell cycle arrest in the G2/M phase in HCC cells<sup>[2]</sup>.

Romidepsin (0-48 hours; 0-60 nM) promotes apoptosis in HCC cells, increases c-caspase-3, c-caspase-9, and c-PARP protein expression<sup>[2]</sup>.

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

#### Cell Proliferation Assay<sup>[2]</sup>

Cell Line:	HCC cells
Concentration:	0 nM; 10 nM; 20 nM; 30 nM; 40 nM; 50 nM; 60 nM; 70 nM; 80 nM
Incubation Time:	0 hours; 12 hours; 24 hours; 48 hours; 72 hours
Result:	Inhibited HCC cells proliferation.

#### Cell Cycle Analysis<sup>[2]</sup>

Cell Line:	HCC cells
Concentration:	0 nM; 15 nM; 30 nM; 60 nM
Incubation Time:	12 hours; 24 hours; 48 hours
Result:	Caused a G2/M arrest.

#### Western Blot Analysis<sup>[2]</sup>

Cell Line:	HCC cells
Concentration:	0 nM; 15 nM; 30 nM; 60 nM
Incubation Time:	12 hours; 24 hours; 48 hours
Result:	Increased c-caspase-3, c-caspase-9, and c-PARP expression in HCC cells.

#### In Vivo

Romidepsin (intraperitoneal injection; 0.5 and 1 mg/kg; every 3 day; 21 days) inhibited the tumor growth, reveals a higher expression of p-cdc25C, ki67, c-caspase-3 and c-PARP, and a lower expression of Ki-67 in Romidepsin treated tumors<sup>[2]</sup>.

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

Animal Model:	Nude mice with Huh7 cells <sup>[2]</sup>
Dosage:	0.5 and 1 mg/kg
Administration:	Intraperitoneal injection; 0.5 and 1 mg/kg; every 3 day; 21 days
Result:	Suppressed tumor growth in mouse xenograft models.

#### CUSTOMER VALIDATION

- Cancer Cell. 2023 Mar 13;41(3):602-619.e11.
- Theranostics. 2021 Mar 20;11(11):5605-5619.
- Cancer Res. 2020 Oct 15;80(20):4426-4438.
- Cancer Res. 2016 Dec 1;76(23):7001-7011.

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- EBioMedicine. 2022 Dec 31;87:104420.

See more customer validations on [www.MedChemExpress.com](http://www.MedChemExpress.com)

## REFERENCES

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- [1]. Furumai R, et al. FK228 (depsipeptide) as a natural prodrug that inhibits class I histone deacetylases. *Cancer Res.* 2002 Sep 1;62(17):4916-21.
- [2]. Sun WJ, et al. Romidepsin induces G2/M phase arrest via Erk/cdc25C/cdc2/cyclinB pathway and apoptosis induction through JNK/c-Jun/caspase3 pathway in hepatocellular carcinoma cells. *Biochem Pharmacol.* 2017 Mar 1;127:90-100.
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**Caution: Product has not been fully validated for medical applications. For research use only.**

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