

## Romidepsin

Catalog No: tcsc0985

Available Sizes

Size: 1mg

Size: 5mg

Size: 10mg

Specifications

CAS No:

128517-07-7

Formula:

 $C_{24}H_{36}N_4O_6S_2$ 

**Pathway:** Epigenetics;Cell Cycle/DNA Damage

Target:

HDAC;HDAC

Purity / Grade:

**Solubility:** 10 mM in DMSO

**Alternative Names:** FK 228;FR 901228;NSC 630176

## **Observed Molecular Weight:**

540.7

**Product Description** 

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Romidepsin is a potent **HDAC1** and **HDAC2** inhibitor with **IC**<sub>50</sub>s of 36 and 47 nM, respectively.

IC50 & Target: IC50: 36 nM (HDAC1), 47 nM (HDAC2)

[1]

*In Vitro:* Romidepsin potently inhibits HDAC1 and HDAC2 ( $IC_{50}$ =36, 47 nM, respectively). Romidepsin has slightly inhibitory effects against HDAC4 and HDAC6 ( $IC_{50}$ =510, 14000 nM, respectively). Romidepsin induces histone acetylation and p21 expression with an EC<sub>50</sub> of 3.0 nM. Romidepsin is more strongly than redFK with EC<sub>50</sub> of 11 nM due to the instability of redFK in HeLa cells<sup>[1]</sup>. Romidepsin is 100 times more potent than TSA and 1,000,000 times more potent than butyrate in inhibiting the proliferation of the A549 cells. Romidepsin causes mitotic arrest, and that the treatment with HDIs causes defects in chromosome segregation in mitosis <sup>[2]</sup>. Romidepsin inhibits the growth of U-937, K562, and CCRF-CEM cells with  $IC_{50}$  values of 5.92 nM, 8.36 nM, and 6.95 nM, respectively<sup>[3]</sup>.

*In Vivo:* In a scid mouse lymphoma model, romidepsin treated mice once or twice a week survive longer than control mice, with median survival times of 30.5 (0.56 mg/kg) and 33 days (0.32 mg/kg), respectively (vs. 20 days in control mice). Remarkably, 2 out of 12 mice treated with romidepsin (0.56 mg/kg once or twice a week) survive past the observation period of 60 days. The apoptotic population of U-937 cells increases to 37.7% after 48 hr of treatment with romidepsin in a time dependent manner. In addition, romidepsin induces G1 and G2/M arrest and the differentiation of U-937 cells to the CD11b(+)/CD14(+) phenotype. Expression of p21(WAF1/Cip1) and gelsolin mRNA increases up to 654- and 152-fold, respectively, after 24 hr of treatment with romidepsin. Romidepsin causes histone acetylation in p21(WAF1/Cip1) promoter regions, including the Sp1-binding sites<sup>[3]</sup>.



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