## MC1742

®

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Cat. No.: CAS No.: Molecular Formula: Molecular Weight: Target: Pathway: Storage:	HY-110280 1776116-74-5 C <sub>21</sub> H <sub>21</sub> N <sub>3</sub> O <sub>3</sub> S 395.47 HDAC; Apoptosis Cell Cycle/DNA Damage; Epigenetics; Apoptosis 4°C, sealed storage, away from moisture and light * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture	N N N S O H S O H O H
	and light)	

## SOLVENT & SOLUBILITY

	Solvent Mass Concentration	1 mg	5 mg	10 mg
Preparing Stock Solution	1 mM	2.5286 mL	12.6432 mL	25.2864 ml
	5 mM	0.5057 mL	2.5286 mL	5.0573 mL
	10 mM	0.2529 mL	1.2643 mL	2.5286 mL

DescriptionMC1742 is a potent HDAC inhibitor, with IC50s of 0.1 μM, 0.11 μM, 0.02 μM, 0.007 μM, 0.61 μM, 0.04 μM and 0.1 μMHDAC2, HDAC3, HDAC6, HDAC8, HDAC10 and HDAC11, respectively. MC1742 can increase acetyl-H3 and acetyl-tu and inhibits cancer stem cells growth. MC1742 can induce growth arrest, apoptosis, and differentiation in sarcon	MC1742 is a potent HDAC inhibitor, with IC <sub>50</sub> s of 0.1 μM, 0.11 μM, 0.02 μM, 0.007 μM, 0.61 μM, 0.04 μM and 0.1 μM for HDAC1, HDAC2, HDAC3, HDAC6, HDAC8, HDAC10 and HDAC11, respectively. MC1742 can increase acetyl-H3 and acetyl-tubulin levels and inhibits cancer stem cells growth. MC1742 can induce growth arrest, apoptosis, and differentiation in sarcoma CSC <sup>[1]</sup> .				
IC <sub>50</sub> & Target         HDAC1 0.1 μM (IC <sub>50</sub> )         HDAC2 0.11 μM (IC <sub>50</sub> )         HDAC3 0.02 μM (IC <sub>50</sub> )         HDAC6 0.02 μM (IC <sub>50</sub> )           HDAC8 0.61 μM (IC <sub>50</sub> )         HDAC10 0.04 μM (IC <sub>50</sub> )         HDAC11 0.1 μM (IC <sub>50</sub> )         HDAC11 0.1 μM (IC <sub>50</sub> )					
In Vitro MC1742 (compound 1) (0.5 and 2 μM; 24 hours) increases acetylation in a dose-dependent manner, observable as nuclear staining of acetyl-histone H3 <sup>[1]</sup> . MC1742 (0.5, 1 and 2 μM; 24, 48 and 72 hours) significantly induces apoptosis of all CSC cultures <sup>[1]</sup> . MC1742 (0.025-0.5 μM; 14 days) enhances bone nodule formation in a significant dose-dependent manner <sup>[1]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only. Immunofluorescence	MC1742 (compound 1) (0.5 and 2 μM; 24 hours) increases acetylation in a dose-dependent manner, observable as punctuate nuclear staining of acetyl-histone H3 <sup>[1]</sup> . MC1742 (0.5, 1 and 2 μM; 24, 48 and 72 hours) significantly induces apoptosis of all CSC cultures <sup>[1]</sup> . MC1742 (0.025-0.5 μM; 14 days) enhances bone nodule formation in a significant dose-dependent manner <sup>[1]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only. Immunofluorescence				

Cell Line:	Sarcoma cancer stem cells <sup>[1]</sup>	
Concentration:	0.5 and 2 μM	
Incubation Time:	24 hours	
Result:	Increased acetylation in a dose-dependent manner, observable as punctuate nuclear staining of acetyl-histone H3.	
Apoptosis Analysis		
Cell Line:	Sarcoma cancer stem $\operatorname{cells}^{[1]}$	
Concentration:	0.5, 1 and 2 μM	
Incubation Time:	24, 48 and 72 hours	
Result:	Significantly induced apoptosis of all CSC cultures.	
Cell Differentiation Assa	у	
Cell Line:	Sarcoma cancer stem cells <sup>[1]</sup>	
Concentration:	0.025, 0.05, 0.1 and 0.5 μM	
Incubation Time:	14 days	
Result:	Successfully enhanced bone nodule formation in a significant dose-dependent manner.	

## REFERENCES

[1]. Di Pompo G, et al. Novel histone deacetylase inhibitors induce growth arrest, apoptosis, and differentiation in sarcoma cancer stem cells. J Med Chem. 2015;58(9):4073-4079.

## Caution: Product has not been fully validated for medical applications. For research use only.

 Tel: 609-228-6898
 Fax: 609-228-5909
 E-mail: tech@MedChemExpress.com

 Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA