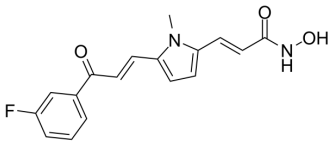
MC1568

Cat. No.:	HY-16914
CAS No.:	852475-26-4
Molecular Formula:	$C_{17}H_{15}FN_2O_3$
Molecular Weight:	314.31
Target:	HDAC
Pathway:	Cell Cycle/DNA Damage; Epigenetics
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 18.5 mg/mL (58.86 mM; Need ultrasonic and warming)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.1816 mL	15.9079 mL	31.8157 mL
	5 mM	0.6363 mL	3.1816 mL	6.3631 mL
	10 mM	0.3182 mL	1.5908 mL	3.1816 mL

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

In Vivo

1. Add each solvent one by one: 17% Polyethylene glycol 12-hydroxystearate in saline
   Solubility: 2.5 mg/mL (7.95 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: 1 mg/mL (3.18 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

MC1568 is a selective class II (IIa) histone deacetylas (HDAC II) inhibitor, used for cancer research.

IC_{50} & Target

HDAC

In Vitro

MC1568 arrests myogenesis by decreasing myocyte enhancer factor 2D (MEF2D) expression, by stabilizing the HDAC4–HDAC3–MEF2D complex, and paradoxically, by inhibiting differentiation-induced MEF2D acetylation[1]. MC1568 and MC1575 inhibits IL-8 levels and cell proliferation in either unstimulated or PMA-stimulated melanoma cells. They acts by suppressing c-Jun binding to the IL-8 promoter, recruitment of histones 3 and 4, RNA polymerase II and TFIIB to the c-Jun promoter, and c-Jun expression[2]. MC1568 interferes with the RAR- and PPARγ-mediated differentiation-inducing signaling pathways. In F9 cells, this inhibitor specifically blocks endodermal differentiation. In 3T3-L1 cells, MC1568 attenuates PPAR

	γ-induced adipogenesis[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	MC1568 shows an apparent tissue-selective HDAC inhibition. In skeletal muscle and heart, MC1568 inhibits the activity of HDAC4 and HDAC5 without affecting HDAC3 activity, thereby leaving MEF2–HDAC complexes in a repressed state[1]. MC1568 increases mortality and lesion volume and did not improve functional outcome. In addition, MC1568 decreases microtubule associated protein 2, phosphorylated neurofilament heavy chain and myelin basic protein immunoreactivity in the periinfarct cortex[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	For proliferation studies, 15 ×10^3 cells are seeded onto 24-well plates in RPMI-1640 medium supplemented with 10% heat-inactivated fetal bovine serum, 3 mM L-glutamine, 2% penicillin/streptomycin. After 24 h, untreated or HDACis-treated cells are incubated with either vehicle alone or PMA (50 ng/mL) for 6 h, and cell proliferation is evaluated by MTT assay and by cell number counting[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [4]	Adult male Wistar rats (n=15-17/group) are subjected to 2 h MCAO and orally gavaged with MC1568 (a selective class IIa HDAC inhibitor), SAHA (a non-selective HDAC inhibitor), or vehicle-control for 7 days starting 24 h after MCAO. A battery of behavioral tests is performed. Lesion volume measurement and immunohistochemistry are performed 28 days after MCAO [4] MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Cell Physiol. 2018 Jan;233(1):673-687.
- J Ethnopharmacol. 2023 Feb 8;116240.
- Cell Signal. 2020 Dec;76:109786.
- J Mol Med (Berl). 2019 Aug;97(8):1183-1193.
- Tissue Barriers. 2021 May 6;1911195.

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REFERENCES

[1]. Nebbioso A, et al. Selective class II HDAC inhibitors impair myogenesis by modulating the stability and activity ofHDAC-MEF2 complexes. EMBO Rep. 2009 Jul;10(7):776-82.

[2]. Venza I, et al. Class II-specific histone deacetylase inhibitors MC1568 and MC1575 suppress IL-8 expression in human melanoma cells. Pigment Cell Melanoma Res. 2013 Mar;26(2):193-204.

[3]. Nebbioso A, et al. HDACs class II-selective inhibition alters nuclear receptor-dependent differentiation. J Mol Endocrinol. 2010 Oct;45(4):219-28.

[4]. Kassis H, et al. Class IIa histone deacetylases affect neuronal remodeling and functional outcome after stroke. Neurochem Int. 2016 Jun;96:24-31.

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

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