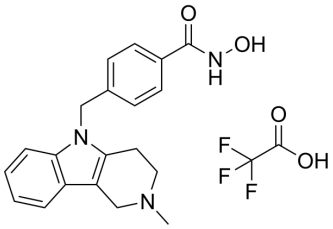
Tubastatin A TFA

Cat. No.:	HY-13271B
CAS No.:	1239262-52-2
Molecular Formula:	$C_{22}H_{22}F_3N_3O_4$
Molecular Weight:	449.42
Target:	HDAC; Autophagy; Apoptosis
Pathway:	Cell Cycle/DNA Damage; Epigenetics; Autophagy; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Tubastatin A (TSA) TFA is a potent and selective?HDAC6?inhibitor with?IC_{50}?of 15 nM in a cell-free assay, and is selective (1000-fold more) against all other isozymes except HDAC8 (57-fold more). Tubastatin A TFA also inhibits HDAC10 and metallo-β-lactamase domain-containing protein?2 (MBLAC2).
IC_{50} & Target	IC50: 15 nM (HDAC6)[1]
In Vitro	Tubastatin A is substantially selective for all 11 HDAC isoforms and maintains over 1000-fold selectivity against all isoforms excluding HDAC8, where it has approximately 57-fold selectivity. In homocysteic acid (HCA) induced neurodegeneration assays, Tubastatin A displays dose-dependent protection against HCA-induced neuronal cell death starting at 5 μM with near complete protection at 10 μM[1].?At 100 ng/mL Tubastatin A increases $Foxp^{3+}$ T-regulatory cells (Tregs) suppression of T cell proliferation in vitro[2].?Tubastatin A treatment in CC12 cells would lead to myotube formation impairment when alpha-tubulin is hyperacetylated early in the myogenic process; however, myotube elongation occurs when alpha-tubulin is hyeperacetylated in myotubes[3].?A recent study indicates that Tubastatin A treatment increases cell elasticity as revealed by atomic force microscopy (AFM) tests without exerting drastic changes to the actin microfilament or microtubule networks in mouse ovarian cancer cell lines, MOSE-E and MOSE-L[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Daily treatment of Tubastatin A at 0.5 mg/kg inhibits HDAC6 to promote Tregs suppressive activity in mouse models of inflammation and autoimmunity, including multiple forms of experimental colitis and fully major histocompatibility complex (MHC)-incompatible cardiac allograft rejection[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Acta Pharm Sin B. 20 May 2022.
- Cell Death Dis. 2022 Oct 21;13(10):888.
- Antioxidants (Basel). 2022 Apr 7;11(4):732.
- Antioxidants. 2020 Jul 9;9(7):599.
- PLoS Pathog. 2021 Sep 20;17(9):e1009940.

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REFERENCES

[1]. de Zoeten EF, et al. Histone deacetylase 6 and heat shock protein 90 control the functions of Foxp3(+) T-regulatory cells. Mol Cell Biol. 2011 May;31(10):2066-78.

[2]. Kyle V. Butler et al. Rational Design and Simple Chemistry Yield a Superior, Neuroprotective HDAC6 Inhibitor, Tubastatin A J. Am. Chem. Soc., 2010, 132 (31), pp 10842-10846

[3]. Di Fulvio S, et al. Dysferlin interacts with histone deacetylase 6 and increases alpha-tubulin acetylation. PLoS One. 2011;6(12):e28563

[4]. Ketene AN, et al. Actin filaments play a primary role for structural integrity and viscoelastic response in cells. Integr Biol (Camb). 2012 May;4(5):540-9.

[5]. Brijmohan AS, et al. HDAC6 Inhibition Promotes Transcription Factor EB Activation and Is Protective in Experimental Kidney Disease. Front Pharmacol. 2018 Feb 1;9:34.

[6]. Severin Lechner, et al. Target deconvolution of HDAC pharmacopoeia reveals MBLAC2 as common off-target. Nat Chem Biol. 2022 Apr 28.

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