

Product Data Sheet

Product Name: VT103
Cat. No.: GC63257

Chemical Properties

Cas. No. 2290608-13-6

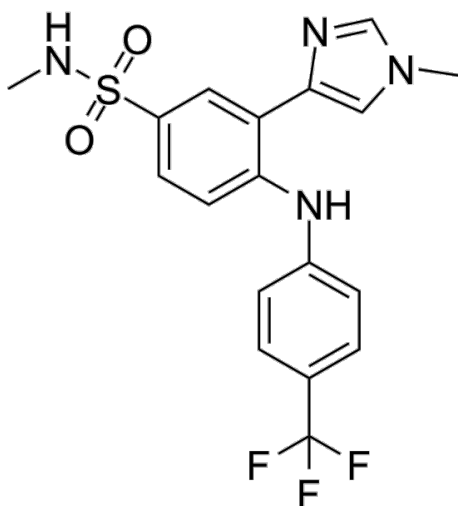
Formula $C_{18}H_{17}F_3N_4O_2S$ M.Wt 410.41

Solubility DMSO : 50 mg/mL (121.83 mM; Need ultrasonic) Storage Store at -20°C

General tips For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

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Cell lines	Neonatal Mice Cardiac Fibroblasts (NMCFs)
Preparation Method	Neonatal Mice Cardiac Fibroblasts (NMCFs) were stimulated with angiotensin II (Ang-II; 100nM). The cells were treated with VT103 (10μM) for 48 hours.
Reaction Conditions	10μM; 48h.
Applications	VT103 reversed the Ang-II-induced increase in α-SMA expression. VT103 also inhibited collagen gel contraction and cell migration.

Animal experiment [2]:

Animal models	Six-week-old female BALB/c-nu mice
Preparation Method	KTOR81 cells (5x10 ⁶) suspended in Matrigel were injected subcutaneously into the backs of the mice. Treatment was initiated when the tumor volume reached 200mm ³ . The mice was treated with VT103 (100mg/kg).
Dosage form	100mg/kg; p.o.; daily administration for 14 days.
Applications	The combination of Dabrafenib and VT103 induced significant tumor shrinkage and showed a sustained response with maintained tumor regression during the 42 days observation period. The combination therapy did not cause significant adverse effects on the body weight of the mice.

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References:

- [1] Song S, Zhang X, Huang Z, et al. TEA domain transcription factor 1 (TEAD1) induces cardiac fibroblasts cells remodeling through BRD4/Wnt4 pathway. *Signal Transduct Target Ther.* 2024 Feb 19;9(1):45.
- [2] Gao X, Chen J, Yin G, et al. Hyperforin ameliorates neuroinflammation and white matter lesions by regulating microglial VEGFR2 /SRC pathway in vascular cognitive impairment mice. *CNS Neurosci Ther.* 2024 Mar;30(3):e14666.

Background

VT103 is a selective TEAD1 protein palmitoylation inhibitor. VT103 inhibits YAP/TAZ-TEAD-mediated gene transcription, blocks TEAD auto-palmitoylation, and disrupts the interaction between YAP/TAZ and TEAD^[1-2]. VT103 can be used in research related to HER2-positive breast cancer, prostate cancer, and triple-negative breast cancer^[3-4].

In vitro, VT103 (10 μ M) was used to treat neonatal mouse cardiac fibroblasts (NMCFs) stimulated with angiotensin II (Ang-II; 100nM) for 48 hours. VT103 reversed the Ang-II-induced increase in α -SMA expression and inhibited collagen gel contraction and cell migration^[5]. VT103 (10 μ M) was also used to treat human coronary artery smooth muscle cells (HCASMCs) overexpressing METTL14 for 24 hours. VT103 inhibited the expression of TEAD1, PDGFRB, CTGF, and Cyr61, restored the expression of contractile markers ACTA2, CNN1, and SM22 α , and attenuated the METTL14 overexpression-enhanced cell viability, proliferation, and migration^[6].

In vivo, VT103 (100mg/kg) combined with Dabrafenib (30mg/kg) was administered orally daily for 14 days to treat female BALB/c-nu mice bearing KTOR81 xenograft tumors. VT103 significantly inhibited tumor growth and maintained sustained tumor regression during a 42-day observation period, without significantly affecting mouse body weight^[7]. VT103 (0.3, 1, 3mg/kg) was administered orally daily for 45 days to treat mice bearing NCI-H226 xenograft tumors. VT103 significantly inhibited tumor growth, and at the 3mg/kg dose, VT103 induced tumor regression without significantly affecting body weight during the treatment period^[8].

References:

- [1] Heinrich T, Peterson C, Schneider R, et al. Optimization of TEAD P-Site Binding Fragment Hit into

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In Vivo Active Lead MSC-4106. J Med Chem. 2022 Jul 14;65(13):9206-9229.

[2] Vaparanta K, Song Z, Farahani I, et al. Phosphoproteomic analysis reveals the diversity of signaling behind ErbB-inhibitor-induced phenotypes. FEBS J. 2025 Dec;292(24):6557-6575.

[3] Zhang S, Tan YQ, Zhang X, et al. TFF3 drives Hippo dependent EGFR-TKI resistance in lung adenocarcinoma. Oncogene. 2025 Mar;44(11):753-768.

[4] Evsen L, Morris PJ, Thomas CJ, et al. Comparative Assessment and High-Throughput Drug-Combination Profiling of TEAD-Palmitoylation Inhibitors in Hippo Pathway Deficient Mesothelioma. Pharmaceuticals (Basel). 2023 Nov 21;16(12):1635.

[5] Song S, Zhang X, Huang Z, et al. TEA domain transcription factor 1(TEAD1) induces cardiac fibroblasts cells remodeling through BRD4/Wnt4 pathway. Signal Transduct Target Ther. 2024 Feb 19;9(1):45.

[6] Wang L, Xia G, Tang Y, et al. METTL14 Promotes Vascular Smooth Muscle Cell Proliferation and Neointima Formation via m6A Methylation TEAD1 mRNA. J Biochem Mol Toxicol. 2025 May;39(5):e70284.

[7] Hosoya K, Ozasa H, Yoshida H, et al. Novel TEAD1 Inhibitor VT103 Enhances Dabrafenib Efficacy in BRAF V600E Mutated Lung Adenocarcinoma via Survivin Downregulation. Cancer Sci. 2025 Jul;116(7):1883-1896.

[8] Tang TT, Konradi AW, Feng Y, et al. Small Molecule Inhibitors of TEAD Auto-palmitoylation Selectively Inhibit Proliferation and Tumor Growth of NF2-deficient Mesothelioma. Mol Cancer Ther. 2021 Jun;20(6):986-998.

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