Product Data Sheet

Product Name: OSU-2S TFA
Cat. No.: GC81880

Chemical Properties

Cas. No.

Formula	$C_{23}H_{38}F_3NO_4$	M.Wt	449.55
Solubility	DMSO: 100 mg/mL (222.44 mM; Need ultrasonic)	Storage	Store at 4°C, sealed storage, away from moisture and light
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

Background

OSU-2S TFA is a non-immunosuppressive derivative of Fingolimod hydrochloride (FTY720). It is an orally active, blood-brain barrier-permeable allosteric activator of PP2A that also activates PKCδ; it restores PP2A activity by displacing the inhibitory protein SET, thereby inducing ROS - PKCδ - caspase-3 -dependent apoptosis, c-Myc degradation, cell cycle arrest, and differentiation. OSU-2S TFA is used in research on diseases such as leukemia, liver cancer, lymphoma, and lung cancer [1] [2] [3] [4] [5] [6] [7].

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In Vivo, OSU-2S (2.5–10 mg/kg; intraperitoneal injection; every other day for 35 days) TFA inhibits tumor growth in an A549 xenograft model, is well tolerated, and downregulates Ki-67 and VEGF[1].

In Vitro, OSU-2S (2-6 μM; 24 h) TFA exhibits concentration-dependent cytotoxicity against canine B-cell lymphoma cell lines (CLBL-1 and 17–71)[7]. The cytotoxicity of OSU-2S (3 μM; 24 h) TFA in canine B-cell lymphoma cell lines is partially mediated by ROS[7]. OSU-2S (2-4 μM; 24 h) TFA induces ROS production and mediates cytotoxicity in primary cells from canine spontaneous B-cell lymphoma[7]. OSU-2S (24 h) TFA downregulates PGC1α and inhibits mitochondrial biogenesis via PP2A-dependent FOXO1 dephosphorylation[6]. OSU-2S TFA reduces mitochondrial mass and ATP production, and inhibits the expression of genes associated with mitochondrial biogenesis[6]. OSU-2S (2 μM; 24 h) TFA induces apoptosis in primary CLL cells[4]. OSU-2S TFA activates PP2A in CLL cells[4]. OSU-2S (5 h) TFA induces SHP1 S591 phosphorylation via a PKC-dependent mechanism[4]. OSU-2S (5-24 h) TFA -induced pSHP1 S591 levels correlate with CLL cell death [4]. The cytotoxicity of OSU-2S (2 μM; 24 h) TFA is partially dependent on PKC[4]. OSU-2S (8 μM; 4 h) TFA activates PKC in CLL cells[4]. OSU-2S (8 μM; 16 h) TFA extensively regulates gene expression in CLL cells, affecting processes such as cell growth, proliferation, death, and the cell cycle[4]. OSU-2S (8 μM; 16-24 h) TFA downregulates TCL1A expression in CLL cells[4]. OSU-2S (5 μM; 24 h) TFA selectively kills CLL cells without affecting normal B cells via ROR1-targeted delivery[4]. OSU-2S (2-8 μM; 24 h) TFA exhibits potent cytotoxicity against MCL cell lines[3]. OSU-2S (2-8 μM; 24 h) TFA induces apoptosis in MCL cell lines[3]. OSU-2S (4-5 μM; 24 h) TFA induces CD74 expression on the surface of MCL cells[3]. OSU-2S (4 μM; 24 h) TFA enhances the cytotoxicity of Milatuzumab against MCL cells by upregulating CD74[3]. OSU-2S (2-10 μM; 48 h) TFA demonstrates anti-proliferative activity against various HCC cell lines[2]. OSU-2S (1.25-2.5 μM; 48 h) TFA enhances the anti-proliferative effect of Sorafenib on HCC cells, exhibiting a synergistic effect[2]. OSU-2S (1.25-2.5 μM; 48 h) TFA enhances Sorafenib-induced apoptosis in HCC cells[2]. OSU-2S (1.25-2.5 μM; 48 h) TFA enhances the anti-HCC effect of Sorafenib via PKCδ and ERK pathways[2]. OSU-2S (1.25-2.5 μM; 8-24 h) TFA in combination with Sorafenib inhibits HCC cell migration and invasion[2]. The activity of OSU-2S (1.25-7.5 μM) TFA against HCC cells is influenced by p53 status; p53 restoration enhances

sensitivity[2]. OSU-2S (1-10 μM; 24 h) TFA inhibits A549 proliferation, with an IC_{50} of 4.432 μM[1]. OSU-2S (1.5-6 μM; 14 days) TFA inhibits colony formation in A549 cells[1]. OSU-2S (1.5-6 μM; 12-24 h) TFA inhibits A549 cell migration[1]. OSU-2S (1.5-6 μM; 24 h) TFA downregulates p-AURKA and S1PR1 protein expression in A549 cells[1].

References:

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[4]. Mani R, et al. Tumor antigen ROR1 targeted drug delivery mediated selective leukemic but not normal B-cell cytotoxicity in chronic lymphocytic leukemia. Leukemia. 2015 Feb;29(2):346-55.
[5]. Xie Z, et al. Pharmacokinetics and Tolerability of the Novel Non-immunosuppressive Fingolimod Derivative, OSU-2S, in Dogs and Comparisons with Data in Mice and Rats. AAPS J. 2020 Jul 16;22(4):92.
[6]. Khalil SM, et al. Protein Phosphatase 2A Activation Dependent Down Regulation of PGC1-Alpha and FOXO1 Phosphorylation with OSU-2S in Human Lymphoma Cells. Blood. 2019;134(Supplement_1):3977.
[7]. Mani R, et al. Non-immunosuppressive FTY720-derivative OSU-2S mediates reactive oxygen species-mediated cytotoxicity in canine B-cell lymphoma. Vet Comp Oncol. 2017 Sep;15(3):1115-1118.

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