

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 PKC δ ; it restores PP2A activity by displacing the inhibitory protein SET, thereby inducing ROS - PKC δ - 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].

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

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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 μ M; 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 μ M; 24 h) TFA in canine B-cell lymphoma cell lines is partially mediated by ROS^[7]. OSU-2S (2-4 μ M; 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 PGC1 α 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 μ M; 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 μ M; 24 h) TFA is partially dependent on PKC^[4]. OSU-2S (8 μ M; 4 h) TFA activates PKC in CLL cells^[4]. OSU-2S (8 μ M; 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 μ M; 16-24 h) TFA downregulates TCL1A expression in CLL cells^[4]. OSU-2S (5 μ M; 24 h) TFA selectively kills CLL cells without affecting normal B cells via ROR1-targeted delivery^[4]. OSU-2S (2-8 μ M; 24 h) TFA exhibits potent cytotoxicity against MCL cell lines^[3]. OSU-2S (2-8 μ M; 24 h) TFA induces apoptosis in MCL cell lines^[3]. OSU-2S (4-5 μ M; 24 h) TFA induces CD74 expression on the surface of MCL cells^[3]. OSU-2S (4 μ M; 24 h) TFA enhances the cytotoxicity of Milatuzumab against MCL cells by upregulating CD74^[3]. OSU-2S (2-10 μ M; 48 h) TFA demonstrates anti-proliferative activity against various HCC cell lines^[2]. OSU-2S (1.25-2.5 μ M; 48 h) TFA enhances the anti-proliferative effect of Sorafenib on HCC cells, exhibiting a synergistic effect^[2]. OSU-2S (1.25-2.5 μ M; 48 h) TFA enhances Sorafenib-induced apoptosis in HCC cells^[2]. OSU-2S (1.25-2.5 μ M; 48 h) TFA enhances the anti-HCC effect of Sorafenib via PKC δ and ERK pathways^[2]. OSU-2S (1.25-2.5 μ M; 8-24 h) TFA in combination with Sorafenib inhibits HCC cell migration and invasion^[2]. The activity of OSU-2S (1.25-7.5 μ M) TFA against HCC cells is influenced by p53 status; p53 restoration enhances

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sensitivity^[2]. OSU-2S (1-10 μ M; 24 h) TFA inhibits A549 proliferation, with an IC₅₀ of 4.432 μ M^[1]. OSU-2S (1.5-6 μ M; 14 days) TFA inhibits colony formation in A549 cells^[1]. OSU-2S (1.5-6 μ M; 12-24 h) TFA inhibits A549 cell migration^[1]. OSU-2S (1.5-6 μ M; 24 h) TFA downregulates p-AURKA and S1PR1 protein expression in A549 cells^[1].

References:

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