

Product Data Sheet

Product Name: STAT3-IN-1

Cat. No.: GC37688

Chemical Properties

Cas. No. 2059952-75-7

SMILES COC1=CC(OC)=CC(/C=C/C2=CC=C(NC(/C=C/C3=CC(OC)=C(OC)C(OC)=C3)=O)C=C2)=C1

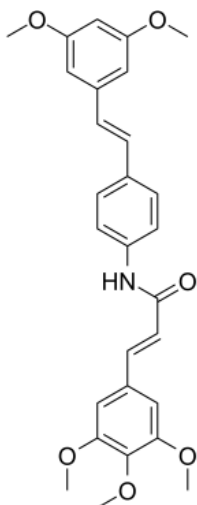
Formula $C_{28}H_{29}NO_6$ M.Wt 475.53

Solubility DMSO: 125 mg/mL (262.86 mM) 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]:

Cell lines DU145 cells

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

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DU145 cells were cultured in RPMI 1640 medium containing 10% fetal bovine serum (FBS) at 37°C in the presence of 5% CO₂. Cells migrating assay was detected using 8-μm trans-well chambers. DU145 cells were pretreated with STAT3-IN-1 (3μM) and CCL5 (100ng/ml) for 48h. 5×10⁴ cells in 150μl non-Preparation Method serum culture medium were seeded into the upper chambers. A total of 800μl culture medium containing 10% FBS medium was added into the lower chambers. Twenty-four hours later, trans-wells were washed with phosphate-buffered saline (PBS), fixed with formalin, and stained with 0.1% crystal violet. Cells were counted in three random fields per well under a ×100 microscope.

Reaction
Conditions 3μM; 48h

Applications STAT3-IN-1 treatment suppressed the cell migration of DU145 cells induced by CCL5.

Animal experiment [2]:

Animal models Female Kunming mice

Adult female Kunming mice (4-6 weeks of age) were group-housed in polysulfone cages with bedding material, and were housed in a room with controlled humidity (50±10%) and temperature (25±3°C) under a 12/12h light/dark cycle. All mice were adaptively fed for one week. Freshly harvested Preparation Method breast cancer cells 4T1 (2.5×10⁶ cells per mouse, resuspended in 100μl PBS) were injected into the armpit of the mice. The mice were given 20mg/kg STAT3-IN-1, or vehicle p.o. once daily. Body weights were measured every other day. On the 14th day, all mice were killed, and the tumor was segregated and weighed.

Dosage form 20mg/kg/day for 14 days; p.o.

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Applications STAT3-IN-1 treatment inhibited tumor growth in a xenograft model of 4T1 cells without affecting body weight.

References:

- [1] Ma J, Shayiti F, Ma J, et al. Tumor-associated macrophage-derived CCL5 promotes chemotherapy resistance and metastasis in prostatic cancer[J]. Cell biology international, 2021, 45(10): 2054-2062.
- [2] Li S, Zhang W, Yang Y, et al. Discovery of oral-available resveratrol-cafeic acid based hybrids inhibiting acetylated and phosphorylated STAT3 protein[J]. European journal of medicinal chemistry, 2016, 124: 1006-1018.

Background

STAT3-IN-1 is a potent, selective, and orally active inhibitor of STAT3 with IC₅₀ values of 1.82μM and 2.14μM in HT29 and MDA-MB-231 cells, respectively [1]. STAT3-IN-1 inhibits both STAT3 acetylation and phosphorylation, leading to increased NKG2D ligand expression and decreased IL-10 levels[1-2]. STAT3-IN-1 has been widely used to induce growth arrest and tumor cell death in Meta4 organoids[3].

In vitro, STAT3-IN-1 treatment at 1μM for 8h significantly inhibited the decrease in Nos2 mRNA levels and the increase in IL-1β and IL-6 levels caused by 100μg/ml zymosan in mouse primary bone marrow dendritic cells (BMDCs)[4]. STAT3-IN-1 treatment (3μM) for 48h suppressed the cell migration of DU145 cells induced by CCL5 and retarded the upregulation of Snail and TWIST[5].

In vivo, STAT3-IN-1 treatment via oral administration at a dose of 20mg/kg daily for 14 days significantly inhibited tumor growth in a xenograft model of 4T1 cells without affecting body weight[6]. Injecting STAT3-IN-1 (5mg/kg) and temozolomide (5mg/kg) intratumorally every 5 days for 10 consecutive days can significantly inhibit the tumor growth of A375 cell-xenograft mice with overexpressed RPL18 and improve the survival rate[7].

References:

- [1] Hu Y, Dong Z, Liu K. Unraveling the complexity of STAT3 in cancer: molecular understanding and drug discovery[J]. Journal of Experimental & Clinical Cancer Research, 2024, 43(1): 23.

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- [2] Jia Z, Xiao B, Li J, et al. BTK inhibitors enhance NKG2D ligand expression by regulating IL-10/STAT3 pathway in activated non-GCB diffuse large B-cell lymphoma cells[J]. *Anti-Cancer Drugs*, 2025, 36(5): 374-382.
- [3] Kim H, Jang B, Zhang C, et al. Targeting stem cells and dysplastic features with dual MEK/ERK and STAT3 suppression in gastric carcinogenesis[J]. *Gastroenterology*, 2024, 166(1): 117-131.
- [4] Wu H, Yin X, Zhao X, et al. HDAC11 negatively regulates antifungal immunity by inhibiting Nos2 expression via binding with transcriptional repressor STAT3[J]. *Redox Biology*, 2022, 56: 102461.
- [5] Ma J, Shayiti F, Ma J, et al. Tumor-associated macrophage-derived CCL5 promotes chemotherapy resistance and metastasis in prostatic cancer[J]. *Cell biology international*, 2021, 45(10): 2054-2062.
- [6] Li S, Zhang W, Yang Y, et al. Discovery of oral-available resveratrol-caffeic acid based hybrids inhibiting acetylated and phosphorylated STAT3 protein[J]. *European journal of medicinal chemistry*, 2016, 124: 1006-1018.
- [7] Zuo C, Hu C, Jiang X, et al. RPL18 promotes melanoma progression and drug resistance via BTF3/STAT3-dependent mechanisms and immune modulation[J]. *iScience*, 2026, 29(1).

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