

Product Data Sheet

Product Name: TG101348 (SAR302503)

Cat. No.: GC14324

Chemical Properties

Cas. No. 936091-26-8

Chemical Name N-tert-butyl-3-[[[5-methyl-2-[4-(2-pyrrolidin-1-ylethoxy)anilino]pyrimidin-4-yl]amino]benzenesulfonamide

SMILES CC1=CN=C(N=C1NC2=CC(=CC=C2)S(=O)(=O)NC(C)(C)C)NC3=CC=C(C=C3)OCCN4CCCC4

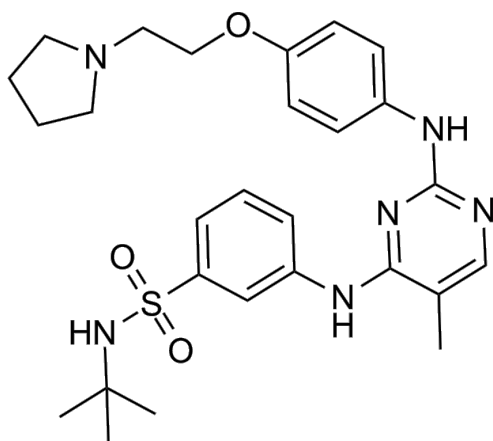
Formula $C_{27}H_{36}N_6O_3S$ M.Wt 524.68

Solubility ≥ 26.2 mg/mL in DMSO, ≥ 6.69 mg/mL in EtOH with ultrasonic and warming 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	Human erythro-megakaryocytic UT7/Epo cells
Preparation Method	The human erythro-megakaryocytic UT7/Epo cells were starved in 0.1% FBS in IMDM for 24hr prior to stimulation. Cells were preincubated with various inhibitors TG101348 (300 and 600nM) 1hr prior to stimulation with 10U/ml EPO for 30min..
Reaction Conditions	300 and 600nM; 30min
Applications	TG101348 strongly suppressed JAK2-mediated STAT5 phosphorylation and AKT activation, and markedly reduced GATA-1 S310 phosphorylation.
Animal experiment [1]:	
Animal models	Immunocompromised RAG2 ^{-/-} γc ^{-/-} mice
Preparation Method	Transplanted RAG2 ^{-/-} γc ^{-/-} mice were also treated with a selective JAK2 inhibitor TG101348 (120mg/kg) or vehicle (DMSO) by oral gavage twice daily for 12 days.
Dosage form	120mg/kg; oral gavage; twice daily for 12 days
Applications	TG101348 significantly reduced erythroid engraftment driven by JAK2V617F ⁺ human progenitors.

References:

[1] Geron I, Abrahamsson A E, Barroga C F, et al. Selective inhibition of JAK2-driven erythroid differentiation of polycythemia vera progenitors. Cancer Cell. 2008 Apr;13(4):321-30.

Background

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TG101348 (SAR302503) is a selective small-molecule inhibitor of JAK2 with an IC₅₀ value of 3nM^[1]. TG101348 is primarily approved for myelofibrosis (MF) and is also under active investigation for myelodysplastic/myeloproliferative neoplasm (MDS/MPN) overlap syndromes and chronic neutrophilic leukemia (CNL)^[2].

In vitro, TG101348 (300 and 600nM) preincubation on human erythro-megakaryocytic UT7/Epo cells for 30min prior to stimulation with 10U/ml erythropoietin (EPO) strongly suppressed JAK2-mediated STAT5 phosphorylation and AKT activation, and markedly reduced GATA-1 S310 phosphorylation^[3]. TG101348 (250nM, 500nM and 2μM) application on pancreatic ductal adenocarcinoma (PDAC) cell lines in combination with the MEK inhibitor trametinib and KRAS inhibitors for 48h led to significant antitumor effects by suppressing ERK and STAT3 phosphorylation and significantly diminishing AKT phosphorylation^[4].

In vivo, TG101348 (60mg/kg) in 200μL volume administrated into mouse models of hemophagocytic lymphohistiocytosis (HLH) via oral gavage twice daily from days 4 to 8 or days 4 through 29 after LCMV infection. TG101348 treatment showed no survival benefit, but improved anemia and thrombocytopenia by inhibiting IFN-γ signaling and modulating G-CSF effects^[5]. TG101348 was given to high-fat high-cholesterol Western diet (WD)-fed Apoe^{-/-} mice orally via gavage at 120mg/kg/day for the first week and then by 240mg/kg/day for the remaining 9 weeks. TG101348 significantly reduced neutrophilia and monocytosis, reduced atherosclerotic lesion area by about 74% in the aorta, and suppressed STAT5 and ERK1/2 phosphorylation in bone marrow cells^[6]. TG101348 (5mg/kg body weight) was injected (i.p.) into ischemia-reperfusion (I/R)-AKI mice model 24h once in advance before surgery. TG101348 ameliorated renal tubular cell injury and protected renal function by inhibiting ferroptosis through TRIM21 expression downregulation^[7].

References:

- [1] Wernig G, Kharas M G, Okabe R, et al. Efficacy of TG101348, a selective JAK2 inhibitor, in treatment of a murine model of JAK2V617F-induced polycythemia vera. *Cancer Cell*. 2008 Apr;13(4):311-20.
- [2] Coltoff A, Mascarenhas J. Fedratinib in 2025 and beyond: indications and future applications. *Blood Adv*. 2025 Apr 22;9(8):1907-1915.
- [3] Geron I, Abrahamsson A E, Barroga C F, et al. Selective inhibition of JAK2-driven erythroid differentiation of polycythemia vera progenitors. *Cancer Cell*. 2008 Apr;13(4):321-30.

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- [4] Miyazaki S, Kitazawa M, Nakamura S, et al. Targeting KRAS-mutant pancreatic cancer through simultaneous inhibition of KRAS, MEK, and JAK2. *Mol Oncol*. 2025 Feb;19(2):377-390.
- [5] Keenan C, Albeituni S, Oak N, et al. Differential effects of itacitinib, fedratinib, and ruxolitinib in mouse models of hemophagocytic lymphohistiocytosis. *Blood*. 2024 Jun 6;143(23):2386-2400.
- [6] Tang Y, Liu W L, Wang W, et al. Inhibition of JAK2 Suppresses Myelopoiesis and Atherosclerosis in Apoe^{-/-} Mice. *Cardiovasc Drugs Ther*. 2020 Apr;34(2):145-152.
- [7] Sun X L, Huang N, Li P, et al. TRIM21 ubiquitylates GPX4 and promotes ferroptosis to aggravate ischemia/reperfusion-induced acute kidney injury. *Life Sci*. 2023 May 15;321:121608.

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