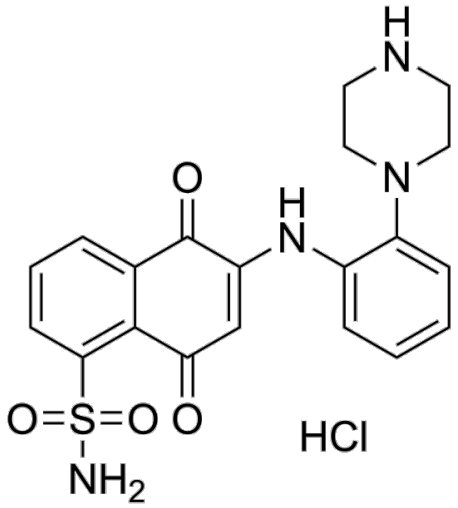
Product Data Sheet

Product Name: STAT3-IN-52 hydrochloride
Cat. No.: GC79465

Chemical Properties

Cas. No.			
Formula	$C_{20}H_{21}ClN_4O_4S$	M.Wt	448.92
Solubility		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



Background

STAT3-IN-52 hydrochloride is an orally active STAT3 inhibitor with a K i value of 2.42 μM. STAT3-IN-52 hydrochloride binds directly and selectively to the pY705 site of STAT3 (K i = 0.44 μM), thereby impairing its function. STAT3-IN-52 hydrochloride induces apoptosis and triggers anti-tumor responses in vivo. STAT3-IN-52 hydrochloride can be used in breast cancer-related research [1].

In Vivo, STAT3-IN-52 (Compound 9) hydrochloride (2.5-10 mg/kg; p.o.; daily; 32 days) hydrochloride orally reduces MDA-MB-231 breast cancer xenograft weight by up to 70% in a dose-dependent manner, with minimal systemic toxicity and potent inhibition of STAT3 phosphorylation[1]. STAT3-IN-

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52 (5 mg/kg; p.o.; daily; 21 days) hydrochloride orally reduces S180 sarcoma xenograft weight by 73% with minimal systemic toxicity[1]. STAT3-IN-52 (200-1000 mg/kg; i.p.; single dose) hydrochloride exhibits very low acute toxicity in ICR mice, with an estimated therapeutic index of over 100[1].

In Vitro, STAT3-IN-52 (Compound 9) hydrochloride selectively binds to the pY705 site of purified STAT3 protein with a Ki of 0.44 μM, and exhibits no binding activity toward purified STAT1 or STAT5 proteins[1]. STAT3-IN-52 binds directly to fluorescently labeled purified STAT3 protein, with a Ki value of 2.42 μM at a ligand-to-protein molar ratio of 1:1[1]. STAT3-IN-52 (0-400 μM; 24 h) hydrochloride inhibits the viability of human normal mammary epithelial MCF-10A cells, with an IC_{50} of 128.9 μM, and its potency against normal cells is 184-fold lower than that against MDA-MB-231 cancer cells[1]. STAT3-IN-52 (0.1-10 μM; 24 h) hydrochloride inhibits the viability of human basal-like breast cancer MDA-MB-231 cells, with an IC_{50} value of 0.7 μM[1]. STAT3-IN-52 (0.1-10 μM; 24 h) hydrochloride inhibits the viability of human medulloblastoma (UW426, UW288-1), human pancreatic cancer (BKPC3), human osteosarcoma (U2OS), and mouse sarcoma (S180) cells, with an IC_{50} of 0.42 μM against S180 cells[1]. STAT3-IN-52 (5 μM; 0-24 h) hydrochloride time-dependently and selectively inhibits the DNA-binding activity of STAT3 homodimers in human basal-like breast cancer MDA-MB-231 cells, exerts weak effects on STAT1 dimer binding, and does not inhibit IFN-γ-induced STAT1 binding or STAT5 dimer binding[1]. STAT3-IN-52 reduces the mRNA expression of the oncogene MMP9 in human basal-like breast cancer MDA-MB-231 cells, but exerts no significant effect on the expression of the CDK2 gene in these cells[1]. STAT3-IN-52 (2.5-10 μM; 12 h) hydrochloride dose-dependently reduces the level of p-STAT3Y705, inhibits IL-6-induced STAT3 phosphorylation, upregulates SHP-1 expression and induces the production of cleaved caspase-3 in human basal-like breast cancer MDA-MB-231 cells, without affecting other modification sites of STAT3, total STAT3, or the phosphorylation of AKT, ERK1/2 or STAT1[1].

References:

[1]. Yu W, et al. Discovery of an Orally Selective Inhibitor of Signal Transducer and Activator of Transcription 3 Using Advanced Multiple Ligand Simultaneous Docking. Journal of medicinal chemistry. 2017 Apr 13;60(7):2718-2731.

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