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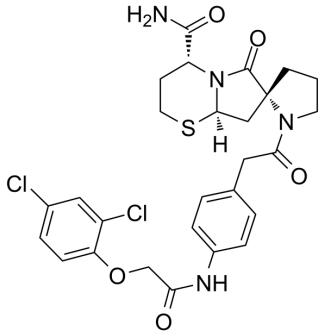
ST 2825



Cat. No.:	HY-50937
CAS No.:	894787-30-5
Molecular Formula:	$C_{27}H_{28}Cl_2N_4O_5S$
Molecular Weight:	591.51
Target:	MyD88
Pathway:	Immunology/Inflammation
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year

SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (169.06 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.6906 mL	8.4529 mL	16.9059 mL
	5 mM	0.3381 mL	1.6906 mL	3.3812 mL
	10 mM	0.1691 mL	0.8453 mL	1.6906 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (4.23 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.23 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

ST 2825 is a specific MyD88 dimerization inhibitor. ST2825 interferes with recruitment of IRAK1 and IRAK4 by MyD88, causing inhibition of IL-1β-mediated activation of NF-κB transcriptional activity[1][2].

IC_{50} & Target

MyD88[1]

In Vitro

ST2825 blocks IL-1R/TLR signaling by interfering with MyD88 homodimerization. ST2825 inhibits this interaction in a concentration-dependent manner with ~40% inhibition of dimerization at 5 μM ST2825 and 80% inhibition at 10 μM ST2825 [1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo	ST2825 dose-dependently inhibits IL-1β-induced production of IL-6 in treated mice after oral administration. The animals are administered orally with the appropriate vehicles or ST2825 at doses ranging from 50 to 200 mg/kg, 5 min prior to i.p. injection with 20 μg/kg IL-1β. ST2825 exertes a significant inhibition of IL-1β-stimulated production of IL-6 at 100 and 200 mg/kg[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	HeLa cells are seeded at 10^5 cells/mL in a 96-well tissue-culture plate. After incubating overnight, the medium is discarded, and the cells are added with tissue culture medium, 10% FBS, containing ST2825 at concentrations ranging from 0.1 to 10 μM and DMSO at 0.1% final concentration. The cells are incubated for 6 and 18 h and then added with the yellow XTT (0.3 mg/mL) for further 2 h of incubation. At the end of the incubation periods, reactions are quantified by using a Sirio S Seac microplate reader[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Mice[1] Mice (female C57Bl/6) are divided into experimental groups of 15 mice. They are injected i.p. with saline (control animals) or recombinant murine IL-1β (20 μg/kg). A time-course analysis of IL-6 production established that the peak of cytokine is reached 2 h after IL-1β injection. ST2825, administered orally as 0.5% suspension in carboxymethylcellulose (CMC) or CMC alone, is supplied to the experimental mice groups. Two hours after IL-1β injection, the animals are killed, and sera are collected to assay IL-6 levels. Mice, which are treated orally with 100 and 200 mg/kg ST2825, shows lower levels of IL-6 versus CMC-treated mice. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Infect. 2019 Sep;79(3):262-276.
- Gut. 2018 Nov;67(11):2035-2044.
- ACS Nano. 2015 Oct 27;9(10):10498-515.
- Nat Commun. 2023 Jan 17;14(1):143.
- Biomaterials. 2020 May;241:119852.

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REFERENCES

[1]. Loiarro M, et al. Pivotal advance: inhibition of MyD88 dimerization and recruitment of IRAK1 and IRAK4 by a novel peptidomimetic compound. J Leukoc Biol. 2007 Oct;82(4):801-10.

[2]. Fantò N, et al. Design, Synthesis, and In Vitro Activity of Peptidomimetic Inhibitors of Myeloid Differentiation Factor 88. J Med Chem. 2008 Mar 13;51(5):1189-202.

[3]. Van Tassell BW, et al. Pharmacologic Inhibition of Myeloid Differentiation Factor 88 (MyD88) Prevents Left Ventricular Dilation and Hypertrophy After Experimental Acute Myocardial Infarction in the Mouse. J Cardiovasc Pharmacol. 2010 Apr;55(4):385-90.

[4]. Zhang HS, et al. Inhibition of myeloid differentiation factor 88(MyD88) by ST2825 provides neuroprotection after experimental traumatic brain injury in mice. Brain Res. 2016 Jul 15;1643:130-9.

[5]. Wang N, et al. Myeloid differentiation factor 88 is up-regulated in epileptic brain and contributes to experimental seizures in rats. Exp Neurol. 2017 Sep;295:23-35.

[6]. Brad Griesenauer, et al. ST2/MYD88 signaling is a therapeutic target alleviating murine acute graft-versus-host disease sparing T regulatory cell function. Indiana University. May 2018.

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

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