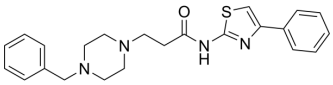
TJ-M2010-5

Cat. No.:	HY-139397
CAS No.:	1357471-57-8
Molecular Formula:	$C_{23}H_{26}N_4OS$
Molecular Weight:	406.54
Target:	MyD88
Pathway:	Immunology/Inflammation
Storage:	4°C, protect from light, stored under nitrogen * In solvent : -80°C, 2 years; -20°C, 1 year (protect from light, stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (245.98 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.4598 mL	12.2989 mL	24.5978 mL
	5 mM	0.4920 mL	2.4598 mL	4.9196 mL
	10 mM	0.2460 mL	1.2299 mL	2.4598 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 (6.15 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (6.15 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

TJ-M2010-5 is a MyD88 inhibitor that binds to the TIR domain of MyD88 to interfere with its homodimerization, and the TLR/MyD88 signal pathway[1][2]. TJ-M2010-5 can be used for the research of myocardial ischemia/reperfusion injury (MIRI)[2].

In Vitro

TJ-M2010-5 (40 μM) inhibits MyD88 homodimerization in transfected HEK293 cells in a concentration-dependent manner and suppresses MyD88 signaling in LPS (100 ng/mL)-responsive RAW 264.7 cells in vitro[1].
?TJ-M2010-5 (5-30 μM) prevents B cell proliferation and induces B cells apoptosis after stimulation with R848 (500 ng/mL)[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[3]

Cell Line:	Purified B cells

Concentration:	0 μM, 5 μM, 10 μM, 20 μM and 30 μM
Incubation Time:	48 hours
Result:	Inhibited the viability of B cells with or without the stimulation of CD40L.

In Vivo

TJ-M2010-5 treatment statistically significantly reduces AOM/DSS-induced colitis and completely prevented CAC development with less related body mass loss, results in 0% mortality of treated mice, decreases cell proliferation, and increased apoptosis in colon tissue in a 10-week CAC mouse model[1].

?TJ-M2010-5 statistically significantly decreases TNF-α, IL-6, G-CSF, MIP-1β, IL-11, IL-17A, IL-22, and IL-23 serum concentrations in mice at both two and seven weeks postinduction, as well as TGF-β1 serum levels at seven weeks postinduction[1].

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

Animal Model:	Female BalB/c mice (6–8 weeks old) [1]
Dosage:	50 mg/kg
Administration:	Treated i.p. daily beginning two days before the first dextran sodium sulfate (DSS) administration throughout a 10-week observation period.
Result:	Significantly prevented inflammation/CAC-related body weight loss and mortality (0% vs 53% in the control group).

CUSTOMER VALIDATION

- Environ Sci Technol. 2023 Apr 5.
- Antiviral Res. 2023 Jul 20;105676.
- Int J Mol Sci. 2023 Sep 7, 24(18), 13878.
- Int Immunopharmacol. 2022 Aug 6;111:109098.
- Toxics. 2023 May 6, 11(5), 437.

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REFERENCES

[1]. Lin Xie, et al. Targeting of MyD88 Homodimerization by Novel Synthetic Inhibitor TJ-M2010-5 in Preventing Colitis-Associated Colorectal Cancer. J Natl Cancer Inst. 2015 Dec 28;108(4):djv364.

[2]. Yan Miao,et al. Inhibition of MyD88 by a novel inhibitor reverses two-thirds of the infarct area in myocardial ischemia and reperfusion injury.Am J Transl Res. 2020 Sep 15;12(9):5151-5169.

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